

Embracing Complexity in the Characterisation and Tracking of
Neurodevelopment

Karin Madericova

Doctor of Philosophy
Aston University

June 2025

© Karin Madericova, 2025

Karin Madericova asserts her moral right to be identified as the author of this thesis.

This copy of the thesis has been supplied on condition that anyone who consults it is understood to recognise that its copyright belongs to its author and that no quotation from the thesis and no information derived from it may be published without appropriate permission or acknowledgement.

Aston University

Embracing Complexity in the Characterisation and Tracking of Neurodevelopment

Karin Madericova

Doctor of Philosophy

2025

Abstract

Neurodiverse diagnoses (NDs) are currently conceptualised as distinct categories with clear-cut diagnostic boundaries. However, converging research evidence is showing that this conventional approach inadequately captures the individual variation and overlaps that are commonly observed across NDs. This thesis aimed to build upon the emerging quantitative research frameworks, through adopting a transdiagnostic dimensional approach to characterising the complexity of the cognitive and mental health dimensions associated with NDs. First, the findings of the systematic review suggested that the co-occurrence of Attention Deficit Hyperactivity Disorder and Reading Difficulty remains consistently high across the lifespan, and that both children and adults experience comparable levels of difficulty across several cognitive domains. The review indicated that complex profiles of co-occurring NDs persist into adulthood, and that studies of adults are substantially underrepresented in the literature. Second, the results of an empirical study revealed that a transdiagnostic sample of neurodivergent adults was best characterised by three data-driven clusters, none of which mapped directly onto traditional diagnostic categories. These transdiagnostic groups were best characterised by a combination of strengths and weaknesses along two multivariate dimensions. Dimension 1 was associated with cognitive flexibility and processing speed abilities, and Dimension 2 was related to language, memory, emotional difficulties and hyperactive behaviour. Further, cognitive flexibility, inhibitory control and vocabulary skills were highlighted as central processes in facilitating communication between the dimensions characterising the transdiagnostic dataset, thus acting as potential mechanisms underpinning heterogeneity, overlaps and/or the co-occurrence of conditions in NDs. This thesis has provided data-driven evidence to support a dimensional structure of NDs, and highlighted several processes which may contribute to the complexity associated with NDs in adults. Further research yielding data-driven accounts of neurodiverse conditions is required to build on the present findings, and to further extend current transdiagnostic dimensional frameworks beyond strictly categorical approaches.

Keywords: Neurodiverse Diagnoses. Categorical Approach. Transdiagnostic Dimensional Approach. Data-driven. Neurodevelopment. Cognition. Mental Health. Adults. ADHD. Dyslexia.

Acknowledgements

Philippians 1:6

I would like to thank my supervisor Prof Joel B Talcott, who saw my potential and believed in me to take on this project. I did not think I would get to start my PhD journey as soon as I did, but you accelerated my career plans and made it possible. Thank you for your continued support over the past four years. I would also like to thank my associate supervisors Dr Jane Waite, Dr Sarah Carrington, and Dr Katie Chisholm for offering guidance when needed, and for taking the time to read and provide feedback on this (very long) thesis.

A special thank you to mum, who has been my biggest support system in the final months of my PhD. Thank you, mum, for your unconditional love, for always showing interest in my work, and for our endless mental health walks. Thank you for believing in me, supporting my dreams, and always being there to remind me that I am capable of achieving anything that I set my mind to. I would also like to thank my four-legged best friend, Lola, who spent hours lying next to me while writing this thesis, and made me laugh when laughing was the last thing on my mind. Thank you to a special someone, who was always proud of me and made work-life balance possible throughout this journey. Thank you for all our trips abroad, and for always being there to listen to me talk about my PhD.

Thank you to my friends and family who supported me until the very end of this journey, to those who didn't give up on me in my most challenging times, and to all those who showed me kindness over the past three years.

I would also like to thank all the organisations who supported me with participant recruitment. Finally, thank you to all the participants who took the time and effort to be involved in my research. This thesis would not have been possible without you.

Table of Contents

Abstract.....	2
Acknowledgements.....	3
List of Abbreviations.....	10
List of Tables.....	12
List of Figures.....	15
Chapter 1: Introduction.....	18
1.1. Neurodiverse Diagnoses: An Overview.....	18
1.2. Cognitive Difficulties Associated with Neurodiverse Diagnoses: Contributions to Individual Variation, Overlaps and Co-Occurrence of Conditions.....	19
1.2.1. Attention and Executive Function.....	20
1.2.2. Processing Speed.....	22
1.2.3. Memory.....	23
1.2.4. Language.....	24
1.3. Mental Health Difficulties Associated with Neurodiverse Diagnoses: Interactions, Overlaps and Co-Occurrence of Conditions.....	25
1.3.1. Internalising.....	26
1.3.2. Externalising.....	27
1.3.3. Emotion Regulation.....	29
1.4. Approaches to Neurodiverse Diagnoses.....	31
1.4.1. The Categorical Approach.....	31
1.4.2. The Transdiagnostic Dimensional Approach.....	34
1.4.2.1. Transdiagnostic Research.....	35
1.4.2.2. Research Domain Criteria (RDoC).....	37
1.4.2.3. The Hierarchical Taxonomy of Psychopathology (HiTOP).....	38
1.4.3. The Neurodiversity Approach.....	41
1.5. Neurodiversity in Adults.....	43
1.6. Thesis Aims.....	43
1.6.1. Research Questions.....	45
Chapter 2: The Cognitive Manifestations and Prevalence Rates of Co-Occurring ADHD and RD Across Development: A Systematic Review and Meta-Analysis.....	46
2.1. Abstract.....	46

2.2. Introduction.....	48
2.2.1. Aims.....	52
2.3. Methods.....	53
2.3.1. Eligibility Criteria.....	53
2.3.1.1. Study Grouping.....	55
2.3.2. Information Sources and Search Strategy.....	55
2.3.3. Selection Process.....	56
2.3.4. Data Extraction Process.....	56
2.3.5. Data Items.....	56
2.3.5.1. Research Question 1.....	57
2.3.5.2. Research Question 2.....	57
2.3.5.3. Other Variables.....	58
2.3.5.4. Handling of Missing or Unclear Information.....	58
2.3.6. Risk of Bias Assessment.....	58
2.3.7. Effect Measures.....	59
2.3.8. Synthesis Methods.....	60
2.3.9. Meta-Analysis of Proportions.....	61
2.3.9.1. Heterogeneity Analyses.....	62
2.3.9.2. Subgroup Analyses.....	62
2.3.9.3. Publication Bias.....	63
2.3.10. Meta-Analysis of Standardised Mean Differences.....	63
2.3.10.1. Meta-Regression and Independent Samples T-Test.....	64
2.3.10.2. Publication Bias.....	65
2.3.11. Sensitivity Analyses.....	65
2.3.12. Certainty Assessment.....	65
2.4. Results.....	66
2.4.1. Study Selection.....	66
2.4.2. Study Characteristics.....	68
2.4.3. Risk of Bias.....	81
2.4.4. Results of Individual Studies.....	82
2.4.5. Results of Syntheses.....	102
2.4.5.1. Meta-Analysis of Proportions.....	102
2.4.5.1.1. Subgroup Analyses.....	102
2.4.5.1.2. Sensitivity Analyses.....	103

2.4.5.1.3. Risk of Reporting Bias.....	105
2.4.5.2. Meta-Analysis of Standardised Mean Differences.....	105
2.4.5.2.1. Meta-Regression and Independent Samples T-Test.....	106
2.4.5.2.2. Sensitivity Analyses.....	108
2.4.5.2.3. Risk of Reporting Bias.....	108
2.4.6. Certainty of Evidence.....	111
2.5. Discussion.....	112
2.5.1. Prevalence of Co-Occurring ADHD and RD.....	112
2.5.2. Cognitive Manifestations of Co-Occurring ADHD and RD.....	115
2.5.3. Strengths and Limitations.....	119
2.5.3.1. Strengths.....	119
2.5.3.2. Limitations.....	121
2.5.4. Implications for Practice and Future Research.....	124
2.5.5. Conclusion.....	125
2.6. Other Information.....	126
2.6.1. Registration and Protocol.....	126
Chapter 3: Embracing Complexity in the Characterisation and Tracking of Neurodevelopment: A Transdiagnostic Protocol.....	128
3.1. Introduction.....	128
3.1.1. Transdiagnostic Recruitment.....	128
3.1.2. Dimensional Assessment.....	129
3.1.3. Exploratory Data Analysis.....	130
3.2. Methods.....	130
3.2.1. Participants.....	130
3.2.2. Procedure.....	135
3.2.3. Cognitive Assessment: NIH Toolbox (Cognition Battery).....	136
3.2.3.1. Attention and Executive Function.....	136
3.2.3.2. Memory.....	137
3.2.3.3. Processing Speed.....	138
3.2.3.4. Language.....	138
3.2.3.5. Scoring of the NIH Toolbox.....	139
3.2.4. Mental Health Assessment.....	139
3.2.4.1. The Extended Strengths and Weaknesses Assessment of Normal Behaviour (E-SWAN).....	140

3.2.4.2. The Emotion Regulation Skills Questionnaire (ERSQ).....	140
3.2.4.3. The Strengths and Difficulties Questionnaire (SDQ).....	141
3.2.5. Statistical Analysis: An Overview.....	141
3.2.5.1. Cluster Analysis.....	142
3.2.5.2. Network Analysis.....	142
3.2.5.3. Descriptive Statistics and Preliminary Analyses.....	143
3.3. Conclusion.....	144
Chapter 4: Cluster Analysis.....	145
4.1. Introduction.....	145
4.1.1. Cluster Analysis.....	145
4.1.2. Principal Component Analysis.....	146
4.1.3. Linear Discriminant Analysis.....	147
4.1.4. Aims.....	148
4.2. Principal Component Analysis: Methods and Results.....	149
4.3. Cluster Analysis: Methods and Results.....	152
4.4. Linear Discriminant Analysis: Methods and Results.....	159
4.5. Discussion.....	163
4.5.1. Strengths and Limitations.....	169
4.5.2. Conclusion and Future Directions.....	172
Chapter 5: Network Analysis.....	174
5.1. Introduction.....	174
5.1.1. Aims.....	176
5.2. Methods.....	176
5.2.1. Data Preparation.....	176
5.2.2. Statistical Analyses.....	177
5.2.3. Network Estimation and Visualisation.....	177
5.3. Results.....	179
5.3.1. Edge Weights.....	179
5.3.2. Stability of Edge Weights.....	182
5.3.3. Centrality Measures.....	185
5.3.4. Stability of Centrality Measures.....	189
5.3.5. Community Detection.....	191
5.3.6. Exploring Bridging Mechanisms.....	192
5.3.7. Stability of Bridging Mechanisms.....	196

5.4. Discussion.....	197
5.4.1. Key Associations (Edges).....	197
5.4.2. Key Processes (Nodes)	198
5.4.3. Communities and Key Bridging Mechanisms.....	202
5.4.4. Strengths and Limitations.....	205
5.4.5. Conclusion and Future Directions.....	208
Chapter 6: General Discussion.....	209
6.1. Thesis Aims.....	209
6.2. Summary of Key Chapter Findings.....	210
6.2.1. Systematic Review and Meta-Analysis.....	210
6.2.2. Transdiagnostic Study: Cluster Analysis.....	210
6.2.3. Transdiagnostic Study: Network Analysis.....	211
6.3. Discussion of Findings.....	211
6.3.1. Transdiagnostic Patterns and Heterogeneity.....	212
6.3.2. Dimensional vs. Categorical Approaches to Assessment and Support.....	214
6.3.3. Cognition and Mental Health in Neurodivergent Adults.....	215
6.4. Strengths and Limitations.....	217
6.4.1. Strengths.....	217
6.4.2. Limitations.....	220
6.5. Future Directions.....	225
6.6. Conclusion.....	228
References.....	229
Supplementary Materials 1 (Chapter 2: Systematic Review).....	281
Appendix A. Prisma 2020 Checklist.....	281
Appendix B. Full Search Strategy.....	284
Appendix C. JBI Checklist for Studies Reporting Prevalence Data.....	287
Appendix D. JBI Checklist for Analytical Cross-Sectional Studies.....	291
Appendix E. Forest Plots by Sampling Methods (Meta-Analysis of Proportions).....	294
Appendix F. Forest Plots by RD Definition (Meta-Analysis of Proportions).....	298
Appendix G. Risk of Bias Assessment Justifications (RQ1).....	302
Appendix H. Risk of Bias Assessment Justifications (RQ2).....	322
Appendix I. Traffic Light Plot (RQ1).....	325
Appendix J. Traffic Light Plot (RQ2).....	328
Appendix K. Doi Plots Assessing Reporting Bias.....	330

Supplementary Materials 2 (Chapter 3: Transdiagnostic Study Protocol).....	339
Appendix A. Recruitment Poster.....	339
Appendix B. Ethical Approval.....	340
Appendix C. Participant Information Sheet.....	341
Appendix D. Consent Form.....	348
Appendix E. Debrief Form.....	349
Appendix F. Assessment Report.....	350
Appendix G. The Extended Strengths and Weaknesses Assessment of Normal Behaviour (E-SWAN)	352
Appendix H. The Emotion Regulation Skills Questionnaire (ERSQ).....	356
Appendix I. The Strengths and Difficulties Questionnaire (SDQ).....	357
Supplementary Materials 3 (Chapters 4-5: Transdiagnostic Study Results).....	359
Appendix A. Descriptive Statistics and Preliminary Analyses.....	359
Appendix B. Comparison Analyses (Chapter 4).....	386
Appendix C. Additional Figures.....	399

List of Abbreviations

ADHD	Attention Deficit Hyperactivity Disorder
ANOVA	Analysis of Variance
APA	American Psychiatric Association
ASD	Autism Spectrum Disorder
BRIEF	Behaviour Rating Inventory of Executive Function
CANTAB	Cambridge Neuropsychological Test Automated Battery
CD	Conduct Disorder
CI	Confidence Interval
DCCST	Dimensional Change Card Sort Test
DSM	Diagnostic and Statistical Manual of Mental Disorders
E-SWAN	Extended Strengths and Weaknesses Assessment of Normal Behaviour
EC	Emotional Clarity
ED	Emotional Difficulties
EF	Executive Function
EI	Expected Influence
ER	Emotion Regulation
ERSQ	Emotion Regulation Skills Questionnaire
FICAT	Flanker Inhibitory Control and Attention Test
GGM	Gaussian Graphical Model
GRADE	Grading of Recommendations, Assessment, Development and Evaluation
HB	Hyperactive Behaviour
HiTOP	Hierarchical Taxonomy of Psychopathology
ICD	International Classification of Diseases
JBII	Joanna Briggs Institute
LB	Lower Bound
LDA	Linear Discriminant Analysis
LFK	Luis Furuya-Kanamori (Index)
LOO	Leave-One-Out (Sensitivity Analysis)
LRT	Likelihood Ratio Test
LSWMT	List Sorting Working Memory Test

M	Mean
MDD	Major Depressive Disorder
ND	Neurodiverse Diagnosis
NIH	National Institute of Health
NIMH	National Institute of Mental Health
ODD	Oppositional Defiant Disorder
ORRT	Oral Reading Recognition Test
PAQ-S	Perth Alexithymia Questionnaire-Short Form
PC	Principal Component
PCA	Principal Component Analysis
PCPST	Pattern Comparison Processing Speed Test
PRISMA	Preferred Reporting Items for Systematic Reviews and Meta-Analyses
PROSPERO	Prospective Register of Systematic Reviews
PSMT	Picture Sequence Memory Test
PVT	Picture Vocabulary Test
RD	Reading Difficulty
RDoC	Research Domain Criteria
REML	Restricted Maximum Likelihood
RQ	Research Question
SD	Standard Deviation
SDQ	Strengths and Difficulties Questionnaire
SE	Standard Error
SLD	Specific Learning Difficulty
SLI	Specific Language Impairment
SMD	Standardised Mean Differences
SPSS	Statistical Package for Social Sciences
SS	Social Skills
UB	Upper Bound
WM	Working Memory

List of Tables

Chapter 2

Table 1. Key characteristics of included studies addressing RQ1 (prevalence).....	68
Table 2. Key characteristics of included studies addressing RQ2 (cognitive performance)...	75
Table 3. Frequency of participants with ADHD+RD in each study addressing RQ1.....	82
Table 4. Effect sizes with standard errors for each cognitive domain assessed across all studies addressing RQ2.....	87
Table 5. Meta-regression results with cognitive domain as moderator for child studies.....	107
Table 6. Meta-regression results with cognitive domain as moderator for adult studies.....	107
Table 7. Simplified assessment of certainty in the body of evidence in each outcome assessed.....	111

Chapter 3

Table 1. Demographic characteristics of the sample ($n = 175$).....	132
Table 2. Formal diagnostic status of the sample ($n = 175$).....	134

Chapter 4

Table 1. The loadings of mental health variables onto principal components.....	151
Table 2. Percentage of variance explained by each principal component.....	151
Table 3. Frequency of diagnostic groups and types of primary diagnoses within individual clusters derived from cognitive data and mental health principal components ($n = 175$).....	153
Table 4. Mean z -scores and one-way ANOVA results for group differences across cognitive tasks and mental health dimensions for clusters derived from cognitive variables and mental health principal components ($n = 175$).....	156
Table 5. Primary cognitive domains assessed by each cognitive task.....	158
Table 6. Structure matrix displaying pooled within-groups correlations between discriminating variables and standardised canonical discriminant functions.....	161
Table 7. Standardised canonical discriminant function coefficients.....	161

Chapter 5

Table 1. Pearson's correlation matrix for mental health principal components and cognitive variables ($n = 175$).....	178

Table 2. Centrality measures for the $\gamma = 0.5$ model.....	186
Table 3. Centrality measures for the $\gamma = 0$ model.....	188
Table 4. Bridge centrality results for the $\gamma = 0.5$ model.....	195
Table 5. Bridge centrality results for the $\gamma = 0$ model.....	195

Supplementary Materials 2

Appendix G. Risk of Bias Assessment Justifications (RQ1).....	302

Table 1. Detailed justifications for studies with unclear or high risk of bias domains (RQ1)	

Appendix H. Risk of Bias Assessment Justifications (RQ2).....	322

Table 1. Detailed justifications for studies with unclear or high risk of bias domains (RQ2).	

Supplementary Materials 3

Appendix A. Descriptive Statistics and Preliminary Analyses (Chapters 4-5).	

Table 1. Mean standard scores across cognitive tasks for the whole sample ($n = 175$).....	359

Table 2. Mean scores across mental health measures for the whole sample ($n = 175$).....	360

Table 3. Mean scores and one-way ANOVA results for group differences on cognitive tasks across diagnostic groups ($n = 175$).....	361

Table 4. Mean scores and one-way ANOVA results for group differences on mental health measures across diagnostic groups ($n = 175$).....	362

Table 5. Frequency of diagnostic groups and types of primary diagnoses within individual clusters derived from cognitive data only ($n = 175$).....	366

Table 6. Mean standard scores and one-way ANOVA results for group differences on cognitive tasks for clusters derived from cognitive data only ($n = 175$).....	367

Table 7. Frequency of diagnostic groups and types of primary diagnoses within individual clusters derived from cognitive and mental health data ($n = 175$).....	369

Table 8. Mean standard scores and one-way ANOVA results for group differences on cognitive tasks for clusters derived from cognitive and mental health data ($n = 175$).....	370

Table 9. Mean scores and one-way ANOVA results for group differences on mental health measures for clusters derived from cognitive and mental health data ($n = 175$).....	372

Table 10. Cluster silhouette statistics for the three cluster solutions derived from cognitive data for the ND group only ($n = 49$).....	378

Table 11. Cluster membership of individuals with different types of primary NDs based on cognitive data only ($n = 49$).....	379

Table 12. Mean scores and one-way ANOVA results for group differences across cognitive tasks for clusters derived from formal ND groups ($n = 49$).....	380
Table 13. Cluster silhouette statistics for the three cluster solutions derived from cognitive and mental health data for the ND group only ($n = 49$).....	382
Table 14. Cluster membership of individuals with different types of primary NDs based on cognitive data and mental health data ($n = 49$).....	382
Table 15. Mean standard scores and one-way ANOVA results for group differences on cognitive tasks for ND clusters derived from cognitive and mental health data ($n = 49$).....	384
Table 16. Mean scores and one-way ANOVA results for group differences on mental health measures for ND clusters derived from cognitive and mental health data ($n = 49$).....	385

Appendix B. Comparison Analyses (Chapter 4).

Table 1. Spearman's rank-order correlation coefficient matrix for all cognitive and mental health variables ($n = 175$).....	387
Table 2. Component loadings of cognitive variables in principal component analysis.....	388
Table 3. Component loadings of cognitive and mental health variables in PCA.....	390
Table 4. Percentage of variance explained by each principal component in PCA.....	391
Table 5. Factor loadings of cognitive and mental health variables in EFA.....	392
Table 6. Percentage of variance explained by each factor in EFA.....	393
Table 7. Factor loadings of each mental health variable.....	394
Table 8. Percentage of variance explained by each factor.....	395
Table 9. Factor loadings of cognitive variables in exploratory factor analysis.....	396
Table 10. Omnibus Likelihood Ratio Tests for cognitive variables in PCA-based clusters.....	398
Table 11. Omnibus Likelihood Ratio Tests for mental health principal components.....	398

List of Figures

Chapter 2

Figure 1. Prisma 2020 flow diagram (Page et al., 2021).....	67
Figure 2. Forest plot displaying proportions of ADHD+RD and 95% confidence intervals for adult studies addressing RQ1.....	92
Figure 3. Forest plot displaying proportions of ADHD+RD and 95% confidence intervals for the first third of child studies addressing RQ1.....	93
Figure 4. Forest plot displaying proportions of ADHD+RD and 95% confidence intervals for the second third of child studies addressing RQ1.....	94
Figure 5. Forest plot displaying proportions of ADHD+RD and 95% confidence intervals for the final third of child studies addressing RQ1.....	95
Figure 6. Forest plot displaying effect estimates and 95% confidence intervals for adult studies addressing RQ2.....	96
Figure 7. Forest plots displaying effect estimates and 95% confidence intervals for child studies assessing working memory and attention.....	97
Figure 8. Forest plots displaying effect estimates and 95% confidence intervals for child studies assessing memory and processing speed.....	98
Figure 9. Forest plots displaying effect estimates and 95% confidence intervals for child studies assessing vocabulary and inhibition.....	99
Figure 10. Forest plots displaying effect estimates and 95% confidence intervals for child studies assessing cognitive flexibility and planning.....	100
Figure 11. Forest plot displaying effect estimates and 95% confidence intervals for child studies assessing problem-solving.....	101
Figure 12. Funnel plots displaying risk of publication bias for child (left) and adult (right) studies in the main meta-analysis results.....	109
Figure 13. Funnel plots displaying risk of publication bias for child (left) and adult (right) studies in the meta-regression results (after accounting for cognitive domains as moderators).....	110

Chapter 4

Figure 1. A line graph showing average observations across cognitive and mental health variables for the three-cluster solution.....	157
Figure 2. A scatterplot of discriminant function scores by data-driven cluster.....	162

Chapter 5

Figure 1. Estimated network model ($\gamma = 0.5$) showing cognitive and mental health dimensions.....	180
Figure 2. Estimated network model ($\gamma = 0$) showing cognitive and mental health dimensions.....	181
Figure 3. Edge weight stability plot for the $\gamma = 0.5$ network model.....	183
Figure 4. Edge weight stability plot for the $\gamma = 0$ network model.....	184
Figure 5. Strength, closeness and betweenness centrality plots for the $\gamma = 0.5$ model.....	187
Figure 6. Strength, closeness and betweenness centrality plots for the $\gamma = 0$ model.....	189
Figure 7. Stability coefficients for betweenness and strength centrality for the $\gamma = 0.5$ model.....	190
Figure 8. Stability coefficients for betweenness, closeness and strength centrality ($\gamma = 0$).....	191
Figure 9. Communities identified using the Walktrap algorithm in the $\gamma = 0.5$ model.....	192

Supplementary Materials 2

Appendix E. Forest plots displaying proportions of ADHD+RD and 95% confidence intervals, categorised by sampling method

Figure 1. Mixed Sampling.....	294
Figure 2. Purposive Sampling.....	295

Appendix F. Forest plots displaying proportions of ADHD+RD and 95% confidence intervals, categorised by RD definition

Figure 1. Discrepancy Model.....	298
Figure 2. Low Achievement Model.....	299
Figure 3. Discrepancy and Low Achievement Model.....	300
Figure 4. Unclear/Other Model.....	301

Appendix K. Doi Plots Assessing Reporting Bias

Figure 1. Doi plot for the pooled proportions of ADHD+RD across child and adult studies.....	330
Figure 2. Doi plot for the pooled proportions of ADHD+RD for child studies only.....	331
Figure 3. Doi plot for the pooled proportions of ADHD+RD for adult studies only.....	332

Figure 4. Doi plot for the pooled proportions of ADHD+RD for studies employing mixed sampling.....	333
Figure 5. Doi plot for the pooled proportions of ADHD+RD for studies employing purposive sampling.....	334
Figure 6. Doi plot for the pooled proportions of ADHD+RD for studies using an unclear (or not widely used) model to define RD.....	335
Figure 7. Doi plot for the pooled proportions of ADHD+RD for studies using the ‘discrepancy’ model to define RD.....	336
Figure 8. Doi plot for the pooled proportions of ADHD+RD for studies using the ‘low achievement’ model to define RD.....	337
Figure 9. Doi plot for the pooled proportions of ADHD+RD for studies using a combination of ‘discrepancy’ and ‘low achievement’ models to define RD.....	338

Supplementary Materials 3

Appendix A. Descriptive Statistics and Preliminary Analyses (Chapters 4-5).

Figure 1. A line graph showing average observations across cognitive variables for a two-cluster solution.....	375
Figure 2. A line graph showing average observations across cognitive variables for a three-cluster solution.....	376
Figure 3. A line graph showing average observations across cognitive variables for a six-cluster solution.....	377

Appendix C. Additional Figures

Figure 1. Optimal number of clusters (derived from mental health components and cognitive variables).....	399

Chapter 1: Introduction

This literature review aims to provide an overview of the complexity associated with neurodiverse diagnoses (NDs), and a rationale for moving towards transdiagnostic dimensional research frameworks in order to embrace such complexity. The review will begin by providing an introduction to neurodiverse diagnoses (Section 1.1.). This will be followed by highlighting the cognitive difficulties frequently associated with NDs, and how such processes may contribute to within-diagnosis heterogeneity, between-diagnosis overlaps, and the co-occurrence of conditions (Section 1.2.). The review will then outline the importance of considering the interactions, overlaps and co-occurrence of mental health difficulties with neurodiverse conditions (Section 1.3.). This evidence will be discussed in light of the challenges associated with the assessment, diagnosis, and support of NDs. After providing this context, the review will discuss the limitations of the traditional categorical approach in capturing the complexity of NDs, and introduce ways in which the emerging transdiagnostic dimensional approach can account for some of the shortcomings of categorical research (Section 1.4.). The benefits of shifting towards transdiagnostic dimensional accounts of NDs will then be discussed in light of the neurodiversity paradigm (Section 1.4.), followed by an overview of the lack of research into neurodiversity in adults (Section 1.5.). Finally, the aims of this thesis will be presented (Section 1.6.).

1.1. Neurodiverse Diagnoses: An Overview

Neurodiverse diagnoses are complex conditions associated with differences in the development of the brain. Altered developmental trajectories can lead to difficulties in various areas of functioning, including cognitive and affective processing, behaviour and/or communication, with the extent of such difficulties ranging from mild to severe. According to the Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM-5; American Psychiatric Association, 2013), NDs encompass conditions such as Autism Spectrum Disorder (ASD), Attention-Deficit/Hyperactivity Disorder (ADHD), or Specific Learning Difficulties (SLD, e.g., dyslexia). The prevalence rates of NDs vary, with a British coalition of charities concerned with NDs (Embracing Complexity) recently suggesting that up to 10% of the UK population may have a neurodiverse condition (Alexander et al., 2021).

NDs are among the most frequently diagnosed paediatric conditions, with many of these conditions persisting throughout development (Cheung et al., 2015; Farah et al., 2021; Orm et al., 2022). However, research suggests that the characteristics of some NDs may not become apparent until later on in life, whilst the behavioural manifestations of several NDs are likely to change from childhood to adulthood (Caye et al., 2016; Taylor & Seltzer, 2010). Adequate identification and management of NDs are crucial to individual wellbeing across development, as they may affect a number of functional outcomes, such as social interactions or academic attainment (McKay et al., 2023; Duff et al., 2023). The nature, extent, and impact of such outcomes are often dependent on the different ways in which neurodiverse conditions manifest across individuals. For example, research has shown that if an individual with a diagnosis of dyslexia also displays characteristics associated with ADHD (e.g., difficulties with attention), their academic performance is more likely to be affected than that of an individual with dyslexia without co-occurring ADHD, as a result of more complex cognitive difficulties (Willcutt et al., 2001; Germanò et al., 2010).

This is important to consider as neurodiverse conditions are highly complex, with common overlaps between diagnoses and high levels of individual variation being observed in their respective aetiology, characteristics and functional outcomes. For instance, research has shown high co-occurrence rates between many NDs (e.g., 50-70% of autistic individuals will also receive a formal diagnosis of ADHD; Rong et al., 2021), whilst specific cognitive weaknesses may also be shared between various NDs (e.g., processing speed difficulties have been observed in both dyslexia and ADHD; Willcutt et al., 2010). Further, no two individuals with a given diagnosis will present with exactly the same difficulties, with research providing evidence of within-diagnosis heterogeneity in multiple neural functions, such as cognitive or affective processing (Mostert et al., 2018; Yue et al., 2022). Such complexities in the nature, scope, and impact of neurodiverse conditions may have significant implications for the identification, diagnosis, and support of neurodivergent individuals.

1.2. Cognitive Difficulties Associated with Neurodiverse Diagnoses: Contributions to Individual Variation, Overlaps and Co-Occurrence of Conditions

Neurodiverse diagnoses have frequently been associated with difficulties in various areas of functioning, many of which are related to an individual's ability to efficiently and effectively process information in the brain (i.e., cognitive skills). The National Institutes of Health (NIH) panel of experts have recently identified several domains of cognition which have been

proposed to be the most important for daily functioning, independence and academic/professional attainment; these domains include executive function (EF), memory, language, processing speed, and attention (Weintraub et al., 2013).

This section will aim to provide examples of difficulties within each of the above cognitive domains with respect to various NDs, whilst considering the co-occurrence and overlaps of specific difficulties across conditions, as well as within-diagnosis heterogeneity. For the purposes of this literature review, this section will focus on some of the most commonly diagnosed NDs, including ADHD, autism, and dyslexia.

1.2.1. Attention and Executive Function

Executive function refers to a set of higher-order control mechanisms involved in the regulation of thoughts and behaviour (Miyake & Friedman, 2012). There are three cognitive domains that have been identified as the core components of EF: working memory (WM), inhibitory control, and set shifting (Miyake et al., 2000), with each of these abilities exerting their own independent, although sometimes interrelated, influences on thought processing and behavioural responses. The working memory is a cognitive system which enables an individual to temporarily store information in the mind, and consequently use top-down manipulation of this information for reasoning and decision-making behaviour (Miyake & Shah, 1999; Baddeley, 2007; Diamond, 2013). Additionally, WM allows for the updating, simultaneous processing and reordering of information in the brain (Wager & Smith, 2003). Inhibitory control refers to the ability to stop/withhold irrelevant or prepotent responses (Miyake et al., 2000; Alderson et al., 2007), making it an essential component in the regulation of behaviour. Finally, set shifting (often referred to as cognitive flexibility) is the ability to efficiently switch between mental sets and/or task demands in order to adapt one's actions to a changing environment (Miyake et al., 2000). Each of these EF components are directly related to attentional processes. For instance, inhibition can also be referred to as attentional control or selective attention at the level of perception (Diamond, 2013), whilst working memory requires sustained attention during the encoding and manipulation of information (e.g., Fournie, 2008). Similarly, the process of switching between task demands directly requires the shifting of attention (Miyake et al., 2000).

There is a substantial amount of research evidence to suggest that difficulties associated with EF are often implicated in neurodiverse conditions. For instance, recent evidence has shown

that executive function and attention difficulties may be associated with social communication and expressive language problems in autistic children (Howard et al., 2023). Similar patterns of difficulties in inhibition, set shifting and working memory abilities have been observed in both adults and children with dyslexia (Booth et al., 2010; Smith-Spark et al., 2016). Further, the hyperactivity and inattention traits of ADHD have frequently been linked with underdeveloped working memory abilities (Rapport et al., 2009; Kofler et al., 2010), whilst the behavioural disinhibition and high impulsivity often present in ADHD have been associated with differences in central executive processes (Alderson et al., 2010; Raiker et al., 2012). Importantly, Fan and Wang (2023) recently reported a bidirectional interaction between inhibitory control and ADHD traits, with more pronounced ADHD traits at Time 1 predicting inhibitory control difficulties at Time 2, and vice versa. The authors proposed that there may be a longitudinal interactive relationship between inhibition and ADHD, such that children with impaired inhibitory control may present with worse cognitive outcomes (which may consequently result in behavioural manifestations characteristic of ADHD), whilst individuals who have already developed ADHD traits may struggle to develop inhibitory abilities through the inability to focus on target tasks (Fan & Wang, 2023). Together, these findings demonstrate that changes in the developmental trajectories of attention and executive functions may underpin a variety of difficulties associated with neurodiverse conditions.

However, the findings regarding EF difficulties in neurodiverse conditions have not always been consistent. For example, some studies have failed to identify any difficulties in shifting abilities in individuals with ADHD (e.g., Biederman et al., 2007), whilst others have only identified lower accuracy on shifting tasks (e.g., Holmes et al., 2010). Similar discrepancies have been observed in studies with dyslexic children, with a large body of evidence suggesting that difficulties in executive function are not uniform across all individuals with reading difficulties (Booth et al., 2010). Whilst the authors suggested that some of the observed heterogeneity may result from methodological differences between studies, these findings demonstrate that differences in EF may contribute to the high levels of within-diagnosis variability often observed across NDs. Additionally, research has shown that differences in executive function may also contribute to the co-occurrence of NDs. For example, a meta-analysis of EF skills in dyslexia, which considered the impact of co-occurring ADHD, revealed that executive function problems may be a shared difficulty between the two conditions (Lonergan et al., 2019). This suggests that differences in executive function may also play a significant role in the overlap or co-occurrence of NDs.

Further, research has shown that attention is a particular cognitive domain in which some NDs may show substantial overlaps. For instance, Van der Meer and colleagues (2012) explored the presence of co-occurring ASD and ADHD, using latent class analysis to compare groups for overlaps in EF and attention abilities. The authors found significant overlaps in both verbal and visual attention, making it difficult to distinguish between clinical cases of ADHD and ASD on tasks assessing this domain, suggesting that the classes identified were independent of diagnosis. This is interesting given that both ADHD and ASD have been associated with high individual variability in cognition (Verté et al., 2006), yet they still demonstrate substantial overlap in specific domains. Such findings demonstrate the complexity in characterising the cognitive profiles associated with NDs, as it is possible for individuals with distinct diagnoses to display more similar cognitive functioning than those within the same diagnostic category.

1.2.3. Processing Speed

Processing speed is defined as the rate at which the brain receives, processes and responds to information, and is closely related to executive function skills (Shanahan et al., 2006; Gordon et al., 2018). Processing speed difficulties have been observed in several NDs. For instance, a large body of literature has provided evidence for slowed processing speed in autistic individuals compared to neurotypical controls (Zapparrata et al., 2023), whilst similar difficulties have also been observed in adults with ADHD (Mostert et al., 2015; Theiling & Patermann, 2016), as well as children and adults with dyslexia (Dubois et al., 2010; Stenneken et al., 2011). Interestingly, slow processing speed has been found to be significantly associated with attention and working memory difficulties in ADHD (Kibby et al., 2019; Jacobson et al., 2011), demonstrating the complex interactions that may contribute to the high variability in neurodiverse conditions. To this end, many NDs have been identified with ‘subtypes’, or more homogenous subgroups of distinct cognitive profiles within a given diagnostic category. For instance, Leib and colleagues (2021) identified four different subtypes of ADHD based on patterns of variability among cognitive profiles, with each subgroup displaying a distinct pattern of working memory and processing speed difficulties. These findings demonstrate that exploring the interactions between various cognitive processes may aid in the characterisation of within-condition heterogeneity and its underlying mechanisms.

Further, slowed processing speed has been proposed to be a shared cognitive weakness in children with co-occurring ADHD and dyslexia, whilst slowed processing speed in conjunction with working memory difficulties have been identified as significant predictors of reading

fluency in children with ADHD (Willcutt et al., 2010; Jacobson et al., 2011). This evidence suggests that processing speed may not only contribute to within-condition heterogeneity, but may also be a process that increases the susceptibility to developing co-occurring conditions in neurodivergent individuals, or a mechanism through which NDs may demonstrate overlaps. This is important since research has repeatedly shown that dyslexia often co-occurs with ADHD, with well-established bidirectional co-occurrence rates ranging from 25% to 40% (Willcutt & Pennington, 2000; DuPaul et al., 2013). Therefore, identifying cognitive mechanisms through which NDs co-occur is critical for gaining a deeper understanding of the complex nature of such conditions.

1.2.4. Memory

Whilst the implications of working memory differences in neurodiverse conditions are well-known (briefly discussed in the EF section above), there are other types of memory difficulties which have been shown to be associated with various NDs. Specifically, episodic memory (a type of declarative memory, which allows for the storage of personal events and experiences) has been proposed to be especially important across the lifespan, and has been associated with both strengths and weaknesses in neurodiverse conditions (Dikmen et al., 2015; Boucher et al., 2012; Skowronek et al., 2008; Eide & Eide, 2011). For example, autistic individuals have been reported to demonstrate difficulties with retrieving specific contextual details of previous events (Bowler et al., 2011), with research suggesting that this difficulty may be related to EF and cognitive control difficulties in autism (e.g., Maister et al., 2013). Conversely, substantial research evidence has found episodic memory to be a particular strength in some neurodivergent individuals. For example, Eide and Eide (2011) suggested that individuals with dyslexia may display enhanced episodic memory abilities due to their reliance on such strategies for long-term memory, with declarative memory also being proposed as a compensatory mechanism for a wide variety of difficulties across several NDs (Ullman & Pullman, 2015). These findings highlight the role of episodic memory in neurodiverse conditions, and suggest that relative strengths and weaknesses within this domain may be dependent on its interactions with other cognitive processes implicated in NDs, resulting in variable presentations across conditions. Such evidence emphasises the importance of exploring relationships between multiple cognitive domains in order to gain a more comprehensive understanding of the variable cognitive profiles associated with NDs.

Moreover, variability in memory abilities has also been observed within conditions; specifically in ASD, where high functioning individuals have been shown to display intact declarative memory, whilst low functioning autistic individuals have demonstrated specific difficulties within this domain (e.g., Boucher & Mayes, 2011). Additionally, research has shown children with ADHD to demonstrate specific difficulties in semantic memory (i.e., general knowledge), whilst simultaneously displaying good episodic memory (Skowronek et al., 2008). This is interesting given that semantic and episodic memory are closely related since they are both a type of declarative memory (e.g., Greenberg & Verfaellie, 2010), yet they appear to show disparate trajectories in neurodivergent individuals. These findings further emphasise the underlying heterogeneity of neurodiverse conditions, and highlight memory as a potential mechanism contributing to the complex presentations of NDs.

1.2.5. Language

Language difficulties are common in neurodiverse conditions, although the pattern of these difficulties may vary across diagnoses and individuals due to differing developmental trajectories (Luyster et al., 2011). Such difficulties may present as challenges in the processing, comprehension or expression of language. For instance, whilst language problems are not included in the current diagnostic criteria for ADHD, research has shown that children with ADHD may find it difficult to comprehend and interpret complex information (McInnes et al., 2003). Further, dyslexia is often referred to as a language-based learning difficulty, with research providing substantial evidence for the presence of phonological, vocabulary and syntax difficulties in this condition (e.g., Lyon et al., 2003; Snowling et al., 2003). Interestingly, both ADHD and dyslexia have demonstrated substantial overlap with Specific Language Impairment (SLI), with a population-based study showing that around 42-46% of children with ADHD or dyslexia were identified with SLI, whilst this percentage increased to around 80% in those with co-occurring ADHD and dyslexia (Helland et al., 2016). This is substantially higher compared to the prevalence of SLI in the general population, which is approximately 7% in children (Tomblin et al., 1997). In the same study by Helland and colleagues (2016), children in the co-occurring group exhibited significantly more difficulties on measures of receptive language, pragmatics, phonology and expressive language, compared to the ADHD and dyslexia groups alone. Similarly, research has shown that autistic children may also experience language difficulties, such as challenges in grammar, syntax, or pragmatics, with the co-occurrence of communication difficulties (e.g., apraxia of speech) also being common

in autism (Vogindroukas et al., 2022). These findings highlight the presence of language difficulties across NDs, and suggest that the complex cognitive profiles associated with the co-occurrence of neurodiverse conditions may lead to an increased susceptibility to developing further (or more severe) difficulties that may not typically be associated with a given condition (e.g., language difficulties in ADHD).

This is especially important given the frequent overlaps and high co-occurrence rates of NDs (Bonti et al., 2024), and even more important as the associations between language and cognitive development have been well established (Zauche et al., 2016). For example, research conducted with autistic children has suggested that receptive language may be correlated with executive function abilities, although the direction of this relationship may be unclear (Ellis Weismer et al., 2018; Friedman & Sterling, 2019), whilst executive function difficulties have been suggested to underpin pragmatic language problems in ADHD (Hawkins et al., 2016). These findings imply that the development of language and other cognitive abilities may be interdependent, and that altered developmental trajectories in one domain may have a cascading effect on the other. This evidence highlights the need for a deeper understanding of the interactions between different cognitive processes in order to characterise the complexity of NDs, especially in individuals with co-occurring conditions, or cognitive profiles that do not align with traditional diagnostic criteria.

Overall, it is evident from the literature that cognitive differences across multiple domains play a key role in understanding the complex profiles associated with a range of NDs. These differences frequently overlap across conditions, and show considerable variability within diagnoses, underscoring both shared underlying mechanisms and individual heterogeneity. The following section will explore the role of mental health difficulties in neurodiverse conditions.

1.3. Mental Health Difficulties Associated with Neurodiverse Diagnoses: Interactions, Overlaps and Co-Occurrence of Conditions

Neurodiverse diagnoses not only co-occur with conditions within the same diagnostic grouping (i.e., the co-occurrence of two NDs, such as ADHD and dyslexia), but also with conditions that may not be within the same diagnostic group (England-Mason, 2020). Research suggests that NDs most often co-occur with affective and/or mental health difficulties, many of which can be categorised into ‘internalising’ and ‘externalising’ conditions. For instance, a large body of literature has demonstrated that youth with learning difficulties (e.g., dyslexia) experience

elevated levels of depressive and/or anxious symptomatology (i.e., internalising problems) relative to their typically developing peers (Nelson & Harwood, 2011). Similarly, externalising difficulties (e.g., conduct problems) have been shown to be the most frequent co-occurring conditions in children with ADHD (Jensen & Steinhausen, 2015), whilst both internalising and externalising problems have been reported to be the most prevalent co-occurring conditions in autism (van Steensel et al., 2013; Gjevik et al., 2011; Underwood et al., 2019). Further, emotion regulation problems have frequently been recognised as a common difficulty across various NDs, including ADHD (Graziano & Garcia, 2016), ASD (Mazefsky et al., 2015), or SLD (Kouvava et al., 2022). This is important since emotion regulation has been shown to be associated with both internalising and externalising problems in neurodivergent individuals, and proposed as a potential transdiagnostic mechanism for the development of co-occurring mental health conditions in NDs (e.g., Boyes et al., 2020; England-Mason, 2020).

This section will briefly review the importance of exploring the co-occurrence of internalising and externalising difficulties alongside neurodiverse conditions, and how such co-occurring problems may further contribute to the complexity associated with NDs – potentially impacting assessment and support. The transdiagnostic role of emotion regulation will also be explored, with particular attention to how difficulties in this area may underpin the overlap between different NDs, as well as their co-occurrence with internalising and/or externalising problems.

1.3.1. Internalising

Internalising difficulties can be characterised as disturbances in mood or emotional states and are often related to mental health conditions, such as anxiety or depression (Zahn-Waxler et al., 2000). Research has shown that the difficulties associated with neurodiverse conditions may make individuals more susceptible to experiencing internalising problems (Baraskewich & McMorris, 2019). For instance, intolerance of unexpected changes to routine is a key characteristic of ASD (APA, 2013), with research showing that such intolerance of uncertainty may be a crucial mechanism underpinning the co-occurrence of anxiety conditions in autistic individuals (South & Rodgers, 2017; Hwang et al., 2020). Similarly, reading difficulties characteristic of dyslexia have been shown to be associated with increased levels of anxiety (Zuppardo et al., 2021), further highlighting how neurodiverse difficulties may contribute to the development internalising problems. In contrast, internalising may also exacerbate the difficulties associated with neurodiverse conditions. For example, research has shown that the presence of anxiety may increase working memory problems in adolescents (D'Agati et al.,

2019). This evidence suggests that it is important to monitor the co-development and/or interactions between internalising problems and neurodiverse conditions, as their co-occurrence may lead to more complex profiles of difficulties. This may have important implications for the support of neurodivergent individuals, with research suggesting that there is currently a lack of adequate assessment and support techniques addressing more complex needs resulting from the co-occurrence of NDs and internalising problems (e.g., Rodgers & Ofield, 2018; Koyuncu et al., 2022).

The challenges associated with the assessment and support of co-occurring internalising conditions in NDs may be further exacerbated by their phenotypic overlap. For instance, Grogan and colleagues (2018) emphasised that differentiating between anxiety and ADHD is extremely difficult due to an overlap in their behavioural presentations. For example, restlessness is a characteristic that may be shared by both anxiety and ADHD (Baraskewich & McMorris, 2019); although it may arise from hyperactivity in ADHD, the same behavioural presentation may result from the body's fight or flight response to perceived threatening stimuli in anxious individuals (e.g., Martella et al., 2020; Adwas et al., 2019). Thus, the co-occurrence of internalising and neurodiverse conditions may not only lead to more complex profiles of functioning, but may also be more difficult to identify in clinical contexts. This emphasises the need for a deeper understanding of the interplay between internalising and NDs in order to inform more adequate assessment and support. This is especially important given the significant impact of co-occurring internalising problems on the quality of life and/or daily functioning of neurodivergent individuals (Rogers & Ofield, 2018).

1.3.2. Externalising

Externalising refers to a set of behaviours that consist of aggressive, hyperactive and/or disruptive tendencies (Hinshaw, 1987), and is often associated with specific diagnoses, such as oppositional defiant disorder (ODD) or conduct disorder (CD; Liu, 2004), as well as difficulties with emotion regulation, self-control or inhibition of impulsive responses (Cai et al., 2021; Berger & Buttelmann, 2021; Martel et al., 2017). Externalising problems frequently co-occur with neurodiverse conditions. For example, research suggests that 30-50% of individuals with ADHD also meet diagnostic criteria for CD and ODD (Gnanavel et al., 2019), with population-based studies suggesting that ADHD and externalising traits often display significant covariation in the general population (Costello et al., 2003). Similarly, externalising problems

have been noted in autism, with estimated prevalence rates suggesting that 28-68% of autistic children display aggression towards the self (i.e., self-injurious behaviour), or others (Soke et al., 2016; Kanne & Mazurek, 2011). The high co-occurrence of externalising problems with NDs highlights the importance of considering such difficulties in the assessment and support of neurodiverse conditions.

Research suggests that the pattern of co-development of NDs with externalising problems may change over time. For instance, in a longitudinal study, Kuja-Halkola and colleagues (2015) found externalising difficulties in middle childhood to influence ADHD traits in early adolescence, whilst ADHD traits in late adolescence had an impact on externalising behaviours in early adulthood. These findings suggest that it is unclear whether externalising problems increase the risk for developing neurodiverse conditions, or vice versa, indicating that the dynamic relationship between NDs and externalising problems may change throughout development. Additionally, the relationship between ADHD and externalising traits was found to increase over time (Kuja-Halkola et al., 2015), suggesting that co-occurring externalising problems may be more prominent in adulthood. Together, these findings highlight the importance of considering developmental timing in the assessment and support of co-occurring externalising difficulties in NDs, suggesting that tailored approaches may be needed at different life stages, from childhood through to adulthood.

Further, research suggests that individuals with more complex profiles of co-occurring NDs may be more susceptible to developing externalising problems. For example, children with co-occurring ADHD and dyslexia have been found to display substantially higher levels of self-reported impulsivity compared to those with dyslexia alone (Duranović et al., 2023). Similarly, some studies have reported higher levels of externalising behaviour in co-occurring ASD and ADHD groups, compared to ASD or ADHD alone (e.g., Berenguer et al., 2018; Tureck et al., 2013). This is especially important to consider given that neurodivergent individuals are more likely to present with a combination of NDs, as opposed to having a single ‘pure’ diagnosis (Bonti et al., 2024). This evidence further highlights the importance of considering the role of externalising difficulties in neurodivergent individuals, especially in those with more complex profiles of co-occurring conditions.

1.3.3. Emotion Regulation

This section will briefly discuss the importance of exploring emotion regulation (ER) in neurodivergent populations, and its potential role as a transdiagnostic mechanism underpinning the co-occurrence of conditions.

Emotion regulation is a multidimensional construct that refers to the ability to effectively monitor, evaluate and modify emotional responses and experiences (Thompson, 2008). Adaptive ER strategies, such as cognitive reappraisal (i.e., altering the impact of an emotion-eliciting situation by re-interpreting negative events), enable individuals to manage their emotional responses effectively (Gross & John, 2003). Conversely, maladaptive ER strategies, such as avoidance of negative emotional experiences or persistent focus on negative content (i.e., worry/rumination), may contribute to emotion dysregulation and result in further difficulties (Kashdan et al., 2006; Aldao et al., 2010).

Research suggests that neurodivergent individuals often fail to employ adaptive ER strategies. For instance, Graziano and Garcia (2016) conducted a meta-analysis of ER difficulties in ADHD and demonstrated that youth with ADHD show difficulties in understanding and recognising emotions, which may contribute to problems with social interactions. Similarly, a large body of evidence has shown that adults with ADHD display a more frequent use of maladaptive ER strategies compared to adults without ADHD (Soler-Gutiérrez et al., 2023). Interestingly, some authors have suggested that the emotional traits (e.g., impulsivity/irritability) resulting from poor ER in ADHD should in fact be considered as ‘core symptoms’ of the diagnosis (Faraone et al., 2019). Likewise, ER difficulties are common among autistic individuals, who have been shown to consistently self-report and/or demonstrate maladaptive patterns of ER strategy use (Cai et al., 2018). Additionally, research suggests that poor ER may be an important feature of autism, and underly some of the core socioemotional and/or behavioural difficulties characteristic of this condition (Mazefsky et al., 2013). This evidence demonstrates the importance of exploring the role of ER in neurodivergent populations.

Many studies have conceptualised ER as a transdiagnostic construct due to its association with a broad range of diagnoses. It has been suggested that ER may be a mechanism underlying the co-occurrence of conditions not only *within* diagnostic groupings (i.e., co-occurring NDs), but

also *across* conditions from different diagnostic groupings, such as co-occurring anxiety and ASD (England-Mason, 2020). For instance, Jaisle and colleagues (2023) recently provided evidence for the role of emotion dysregulation in both ASD and ADHD. They demonstrated that the hyperactivity and impulsivity traits of ADHD, and the restricted and repetitive behaviours characteristic of ASD, were related to social competence indirectly through emotion dysregulation. These findings suggest that ER may be a shared mechanism underlying some of the functional difficulties associated with NDs. Since social difficulties are common in ASD and ADHD respectively, but also in co-occurring ASD and ADHD (APA, 2013; Hoza, 2007; Dellapiazza et al., 2021), and ER difficulties seem to mediate the relationship between both conditions and social difficulties, the abovementioned evidence provides a strong case for the role of ER as a transdiagnostic process underlying the co-occurrence of conditions within diagnostic groupings.

There is also substantial evidence supporting the role of emotion regulation in the co-occurrence of conditions across diagnostic groupings (i.e., NDs co-occurring with mental health difficulties). For example, Cai and colleagues (2021) investigated the relationship between ER and internalising and externalising problems in a community sample of adolescents with or without neurodiverse conditions, spanning the normative-clinical continuum. The authors reported significant associations between emotion dysregulation and internalising/externalising problems within both typically and atypically developing groups. Interestingly, emotion dysregulation was a unique contributor to individual differences in the severity of both internalising and externalising problems, above and beyond diagnostic status (Cai et al., 2021). These findings demonstrate the transdiagnostic role of ER in contributing to the co-occurrence of NDs with mental health difficulties, and highlight ER as a dimensional construct that may help to better characterise the complexity of NDs.

Moreover, research suggests that ER difficulties may be more pronounced in individuals with NDs, relative to typically developing populations. Cai and colleagues (2021) postulated that this may be due to additional executive function difficulties present in individuals with NDs, as EF has been suggested to predict the ability to regulate emotions (Schmeichel & Tang, 2015). Interestingly, this relationship has recently been explored by Tajik-Parvinchi and colleagues (2021), who investigated whether ER strategies mediate the relationship between cognitive control and internalising/externalising behaviour in children with NDs. They found that children with greater cognitive difficulties were more likely to experience maladaptive ER,

which was in turn associated with both internalising and externalising problems (aggression, anxiety or depression). A similar inverse relationship was revealed by Battistutta and colleagues (2021), who found the severity of internalising problems to be associated with more difficulties in executive function, through increased emotion dysregulation and ADHD traits. This research demonstrates that ER may also be implicated in cognition. Specifically, there may be a bidirectional interaction between cognitive difficulties and emotion dysregulation in individuals with NDs. It is possible that the interplay between these processes may increase the risk of developing and/or maintaining co-occurring mental health conditions in neurodivergent individuals.

Overall, existing literature highlights a high degree of co-occurrence between NDs and mental health difficulties, with growing evidence pointing to emotion regulation as a key transdiagnostic mechanism. A more nuanced understanding of these interrelated difficulties is essential to informing accurate assessment and tailored support for neurodivergent individuals.

1.4. Approaches to Neurodiverse Diagnoses

The following section will explore different approaches to studying NDs. First, it will focus on the limitations of the traditional categorical approach in capturing the complexities associated with neurodiverse conditions, as outlined above. This section will then discuss the ways in which transdiagnostic dimensional research can help to account for some of the shortcomings of the categorical approach. Finally, the benefits of shifting towards transdiagnostic dimensional accounts of NDs will be discussed in light of the novel neurodiversity paradigm.

1.4.1. The Categorical Approach

Neurodiverse diagnoses are currently assessed through a variety of educational, health and/or medical pathways, all of which typically utilise conventional assessment measures, derived from the Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM-5; APA, 2013) or the International Classification of Diseases (11th ed.; ICD-11; World Health Organisation, 2019). The DSM has been the most influential framework for diagnosing a range of NDs, and has proven useful in both clinical and research settings. For instance, the standardisation of diagnoses across different clinicians and treatment providers has enabled individuals to receive appropriate treatment world-wide (e.g., Raskin et al., 2022). Similarly, the unified approach to

identifying conditions has enabled researchers from different backgrounds to investigate specific conditions using standardised methodology, enabling the cross-comparison of findings, which has in turn aided the development of clinical practice and interventions.

In recent years, there has been considerable debate around the extent to which the DSM-5 reflects the true complex nature of NDs (e.g., Insel et al., 2010; Kotov et al., 2017). For the majority of conditions, the DSM-5 posits that individuals must meet specific thresholds in order to receive a formal diagnosis, following strict inclusionary and exclusionary criteria. To this end, each diagnosis has been conceptualised as a distinct category with its own set of criteria to aid in diagnosis and treatment (APA, 2013). However, this approach fails to account for the variability and overlaps that are commonly observed across neurodiverse conditions (as described in Section 1.2.). The DSM-5 has attempted to account for this drawback, for example, through reconceptualising autism as a spectrum along which individuals may vary in their traits (APA, 2013). Similarly, in the latest text-revised version, the DSM-5-TR has included an additional section for individuals meeting criteria for more than one diagnosis, such as autism with language delay (APA, 2022). Despite such revisions, the majority of neurodiverse conditions are still predominantly conceptualised as distinct categories.

The rigidity of current diagnostic criteria inherently excludes many individuals with milder difficulties that do not reach diagnostic thresholds, regardless of the significant impact that such difficulties may have on their lived experience (Astle et al., 2022). As a result, the needs of many undiagnosed individuals often go unmet as they lack access to adequate support. Importantly, current diagnostic classification systems also predominantly focus on childhood presentations of NDs, with existing diagnostic criteria not being well aligned with the variable presentations of NDs observed in adulthood (e.g., Antolini & Colizzi, 2023). Therefore, it may be even more difficult for individuals to receive an adequate diagnosis at later stages in development, which is a rising issue given the recently increasing number of adults being referred for assessments and/or seeking help for NDs. For instance, the United Kingdom has recently observed a 20-fold increase in adult ADHD diagnoses (McKechnie et al., 2023). This emphasises the need for more comprehensive diagnostic procedures, which becomes even more critical for the diagnosis of NDs in adulthood.

Further, the shortcomings of the categorical approach have posed a specific issue in neurodiversity research, as for many years, diagnostic categories have been adopted by

researchers as reflecting an underlying ‘reality’ of neurodiverse diagnoses (Astle et al., 2022). This has led to the development of research protocols that inevitably reinforce the categorical structure of NDs (Astle et al., 2022). For instance, case-control study designs (i.e., recruiting participants based on the presence of a particular diagnosis) have dominated developmental science for many decades. This is problematic as, in reality, only a small proportion of individuals have a ‘pure’ diagnosis of a single ND, with research suggesting that approximately 86% of autistic individuals will have at least one co-occurring condition, with comparable rates being observed in ADHD and SLD (Mohammadi et al., 2019; Kadesjö & Gillberg, 2001; Margari et al., 2013). As a result, study samples may not be an accurate representation of neurodivergent populations, as many individuals may be better characterised by more complex profiles. Additionally, individuals with subclinical presentations of NDs have the capacity to enrich research with valuable information on the wide range of abilities displayed along the spectrum, yet they have often been excluded from studies, which has often limited the advances that could be made in neurodiversity research (Astle & Fletcher-Watson, 2020). Such sampling limitations may have a direct impact on clinical practice; for instance, intervention strategies grounded in conceptually categorical research may not be equally as effective for all individuals (i.e., those with complex profiles or co-occurring conditions), and may be limited to only addressing ‘clinically relevant’ difficulties.

Another methodological limitation of categorical research are the measures used to assess NDs. Many ‘gold standard’ assessment tools have been designed to produce dichotomous representations of individual abilities (i.e., typical versus atypical). For instance, the Conners Comprehensive Behaviour Rating Scale (CBRS; Conners, 2011) is widely used by clinical practitioners to assist in the diagnosis of ADHD and for research purposes. The items in this questionnaire, like many other assessment tools, have been directly derived from the DSM-5 diagnostic criteria for ADHD, and although the measure covers various domains of functioning, it focuses solely on maladaptive behaviours, without taking into consideration individual strengths. In order to move away from the traditional categorical view of NDs, it is important to begin to assess the full range of behaviours displayed by neurodivergent individuals, rather than simply focusing on the atypical end of the spectrum. This approach may be particularly useful for assessing cognitive and mental health processes, as within-condition heterogeneity and between-condition overlaps have frequently been associated with individual strengths and weaknesses within these domains and their respective dimensions (e.g., Bathelt, Holmes & Astle, 2018; England-Mason, 2020).

Moreover, the categorical approach also sits comfortably with the ‘core deficit’ hypothesis of neurodiverse conditions. This hypothesis proposes that the social, emotional, behavioural and/or cognitive difficulties associated with a given diagnosis can be accounted for by a single underlying deficit in neurocognitive function (see Astle & Fletcher-Watson, 2020 for a review). For instance, several accounts of autism have attempted to reduce the broad range of difficulties associated with the condition to a simple impairment in, for example, theory of mind (Baron-Cohen, 1985) or executive function (Pennington & Ozonoff, 1996). Similarly, dyslexia has frequently been thought to be underpinned by a ‘core deficit’ in phonological processing (e.g., Melby-Lervåg et al., 2012). However, research has repeatedly shown that a single difficulty cannot exhaustively explain the broad range of profiles observed within a specific diagnostic category (Astle & Fletcher-Watson, 2020). For example, Mammarella and colleagues (2021) recently demonstrated that the differences between cognitive profiles of children with mathematical learning disabilities and a control group were better characterised by general characteristics along the developmental continuum, than by a core deficit in basic number processing skills. Thus, it is evident that reductionist approaches cannot comprehensively characterise the complex presentations of neurodiverse conditions, emphasising the need to shift away from categorical accounts of NDs.

1.4.2. The Transdiagnostic Dimensional Approach

With the aim to study neurodiversity from a broader perspective, the transdiagnostic dimensional approach reconceptualises NDs as a multidimensional space, where neurodevelopment can be studied in terms of several dimensions that fall along a continuum (Astle et al., 2022). Adopting this approach enables researchers to study difficulties that cut across diagnostic boundaries, as opposed to solely focusing on core characteristics that have traditionally been thought to underpin specific diagnostic categories (Dalglish et al., 2020). The transdiagnostic dimensional approach therefore promotes a shift away from conventional study designs to more inclusive sampling strategies, novel data collection methods, and exploratory data analysis techniques that enable more accurate, data-driven theoretical accounts of NDs.

1.4.2.1. Transdiagnostic Research

The transdiagnostic approach aims to identify common maladaptive mechanisms that may underpin a broad range of clinically relevant presentations and functional outcomes in NDs (Harvey et al., 2004; Finlay-Jones et al., 2019). The main objective is to move beyond attempting to pinpoint a core neurocognitive ‘deficit’ with the power to fully explain the broad array of profiles observed within a specific diagnostic category (Astle & Fletcher-Watson, 2020). Transdiagnostic research begins with a shift away from traditional case-control study designs which tend to sustain the cycle of adhering to ill-fitting diagnostic criteria and do not allow for the exploration of characteristics that cut across diagnostic boundaries. To this end, transdiagnostic recruitment involves the relaxation of strict inclusionary criteria, with the aim to capture heterogeneity through sampling a broader population of individuals that vary in the extent and nature of their difficulties (Astle et al., 2022). For instance, participants who do not conform to standard criteria for any specific diagnostic labels, as well as individuals with formal diagnoses, and/or those with more complex profiles of co-occurring conditions may be included in transdiagnostic studies. Such sampling enables the collection of dimensional data across the developmental continuum, and creates the opportunity for studies to capture between-condition overlaps and within-condition variability.

Another pivotal aspect of research that is central to adopting a transdiagnostic dimensional approach are data collection methods. As mentioned in the previous section, ‘gold standard’ tasks have often been used to classify ‘typical’ and ‘atypical’ performance in order to differentiate between cases and controls. For instance, the ‘Go/No-Go’ task has frequently been used in case-control studies to provide evidence for a primary ‘deficit’ in response inhibition that has been thought to underpin ADHD (e.g., Wodka et al., 2007). However, the use of tasks based on previous ‘core deficit’ accounts of NDs reduces the opportunity to investigate other possible underlying mechanisms that may help to better characterise a given difficulty (Astle & Fletcher-Watson, 2020). Therefore, dimensional research encourages the administration of broader assessments that measure a wide variety of behavioural, cognitive and/or mental health dimensions along which individuals may differ. Such measures also allow for the assessment of relative strengths *and* weaknesses associated with a particular area of functioning, thus spanning the whole spectrum of abilities, as opposed to focusing solely on the clinically relevant trait range (Alexander et al., 2020). Thus, dimensional assessments not only provide the opportunity to gain a more comprehensive understanding of individual functioning, but also

have the capacity to reliably and meaningfully distinguish individuals from one another even in subclinical parts of the population, making them well suited for use with transdiagnostic samples (Alexander et al., 2020).

Moreover, the employment of dimensional measures in transdiagnostic samples offers the potential to identify broader areas of need that are common across diagnoses. For instance, Boulton and colleagues (2021) recently developed a transdiagnostic assessment protocol for studying neurodiverse conditions in children. The authors highlighted a set of critical domains that would benefit from transdiagnostic assessment across NDs (e.g., quality of life and mental health), and suggested that the identification of common areas of need can facilitate the development of novel interventions, which may be accessible to individuals based on symptom presentation, as opposed to primary diagnosis (Boulton et al., 2021). This is important as many neurodivergent individuals present with complex profiles of strengths and difficulties that span across several diagnostic categories, and often lack access to adequate support (Astle et al., 2022). Thus, identifying transdiagnostic dimensions may aid in the development of more effective interventions that are based on individual needs, rather than designed to target ‘core’ difficulties associated with specific diagnostic labels.

The essence of transdiagnostic research also lies in the use of novel exploratory data analysis techniques, which aim to provide more accurate, data-driven accounts of NDs. The use of more heterogeneous samples and broader assessment measures naturally produces multivariate data, where data reduction methods can be used to generate simplified models of such large, varied datasets. For example, once a broad multidimensional space has been established using dimensional measures, cluster analysis may be conducted to identify subgroups of individuals based on their similarities and differences in specific characteristics within a given sample (Rivard et al., 2023; Astle et al., 2022). Importantly, any subgroups identified from dimensional measures are not based on a hypothesis or any predefined grouping criteria (e.g., diagnostic labels), but are fully data-driven, enabling the exploration of profiles that may transcend diagnostic categories (Astle et al., 2022). Such flexible data-driven accounts of NDs have the capacity to capture shifts in group membership (and thus developmental change) over time, enabling the characterisation, tracking and predicting of individual wellbeing across development (Rivard et al., 2023). Using data-driven techniques, recent transdiagnostic studies have shown that individual profiles of difficulties fail to directly map onto traditional diagnostic categories (e.g., Mareva et al., 2023; Mareva et al., 2024). This evidence suggests that

dimensional approaches may aid in the development of more informative accounts of individual wellbeing in neurodivergent populations, above and beyond diagnostic labels. Two such approaches will be discussed in the following sections.

1.4.2.2. Research Domain Criteria (RDoC)

With the aim to promote the transition from categorical to transdiagnostic dimensional research, the National Institute of Mental Health (NIMH) have developed the Research Domain Criteria (RDoC; Insel et al., 2010) to provide a framework for investigating mental health conditions without adhering to conventional diagnostic categories. The objective of the RDoC framework is to explore the full range of variation (from genetic to behavioural functioning) in order to gain a more comprehensive understanding of what is typical, and what is clinically relevant (Cuthbert, 2014). This framework is one of the first alternative approaches established to systematically account for the shortcomings of symptom-based diagnostic categories in diagnosing psychological conditions (Cuthbert, 2022).

The framework conceptualises mental health diagnoses (including NDs) as conditions of brain circuits, where multiple ‘units of analysis’ help to elucidate the mechanisms that underpin difficulties in functioning (Cuthbert, 2014). These levels of analysis progress either upwards from measures of neural function to clinically relevant variations in behavioural presentations, or downwards to genetic factors that in turn influence the functioning of the brain (Insel et al., 2010). The RDoC framework includes a set of constructs nested within their respective domains – for example, the ‘Cognitive Systems’ domain is made up of constructs such as attention, memory, perception, language and cognitive control (Cuthbert & Kozak, 2013). These psychological constructs are linked to behavioural dimensions, which are grounded in convergent empirical evidence for the existence of neural circuits that implement psychological functions, with the ultimate aim of RDoC-based research being to relate extremes of functioning along identified dimensions to specific difficulties (Kozak & Cuthbert, 2016). In this way, the framework enables researchers to move away from attempting to validate heterogeneous diagnostic categories, but rather to yield biopsychological explanations of clinically relevant behaviours, through focusing on individual difficulties or more homogenous sets of symptoms (Kozak & Cuthbert, 2016). This approach not only yields more detailed aetiological accounts of complex behavioural manifestations, but also allows researchers to collect data that transcend beyond diagnostic categories, thus enabling dimensional research to

better characterise the mechanisms that may underpin within-diagnosis variability and/or overlaps across conditions.

A recent review by Pacheco and colleagues (2022) has outlined the benefits of applying the RDoC framework to neurodiversity research. For instance, through identifying early manifestations of NDs, RDoC-based research can aid in providing targets for support to improve atypical developmental trajectories at early stages (Pacheco et al., 2022). This is because the framework focuses on exploring the effects of atypical developmental trajectories and environmental factors on individual functioning, which allows for the RDoC constructs to be represented across development, in line with the evolving trajectories of neural circuits and psychological functions (Cuthbert, 2022). This is especially important in neurodiverse conditions for several reasons. First, early identification of difficulties is crucial for improving individual outcomes (e.g., Okoye et al., 2023). Second, NDs often present with diverse developmental trajectories across the lifespan (e.g., Hens & Van Goidsenhoven, 2023). Thus, being able to identify and accurately characterise such differences at different stages across development is crucial for providing adequate support for both children and adults.

1.4.2.3. The Hierarchical Taxonomy of Psychopathology (HiTOP)

The Hierarchical Taxonomy of Psychopathology (HiTOP) is an alternative dimensional classification system that conceptualises psychopathology as a set of hierarchically organised dimensions and focuses on symptom manifestations of mental health conditions that motivate individuals to seek clinical treatment (Ruggero et al., 2019; Conway et al., 2022). Its objective is to address the limitations of conventional diagnostic systems (DSM-5/ICD-10) through characterising and classifying conditions using quantitative nosology (i.e., data-driven empirical findings), as opposed to relying on *a priori* theoretical assumptions grounded in the categorical approach to psychopathology (Kotov, 2016). Although initially developed for the reconceptualisation of mental health conditions, it has recently been suggested that the neurodevelopmental spectrum should also be incorporated into the model, as this has potential for advancing the understanding, assessment, and support of NDs and their co-occurrence with mental health difficulties (Michelini et al., 2024).

HiTOP constructs *syndromes* (i.e., composites of closely related traits or behaviours) from the data-driven covariation of symptoms, and groups these empirically formed groups into

transdiagnostic *spectra* (i.e., larger constellations of syndromes, such as internalising) based on covariation between such variables (Kotov et al., 2017). In this way, the paradigm addresses the issue of arbitrary diagnostic cut-off points and attempts to include the important information that is often lost when excluding individuals who do not meet such criteria despite experiencing significant difficulty (Kotov et al., 2017). Through constructing more homogenous, empirically defined groups of behavioural phenotypes, the paradigm addresses the issue of within-diagnosis heterogeneity that often accompanies traditional diagnostic categories (Ruggero et al., 2019). Importantly, the assignment of syndromes to spectra within the HiTOP paradigm naturally addresses the issue of co-occurrence, as higher-level dimensions cut across diagnostic categories (Ruggero et al., 2019). For instance, if a particular characteristic of interest is common to multiple syndromes, clinicians/researchers may focus on a higher-order HiTOP dimension (Kotov et al., 2017). Internalising, for example, may span traits relevant to both anxiety and depression, allowing clinicians/researchers to simultaneously explore both conditions. In this way, the hierarchical structure of HiTOP allows for the paradigm to be used as a flexible framework for investigating the co-existence of conditions, enabling a shift away from categorical models of psychopathology/NDs with rigid sets of between-diagnosis relationships (Forbes et al., 2016; Michelini et al., 2024).

Such transdiagnostic insights may be used to examine the role of shared genetic and/or environmental risk factors in the development of specific conditions, while also providing information on how individuals may differ on given dimensions across the lifespan (Forbes et al., 2016; Caspi et al., 2020). This is important as the biological and behavioural mechanisms underlying psychopathology (i.e., HiTOP dimensions) may change throughout development (e.g., Michelini et al., 2021). For instance, individual symptom trajectories, gene-environment interactions, as well as the co-occurrence of conditions may vary with age, which may have implications for the way in which individuals are described in clinical settings, or the treatment they receive. To this end, the multilevel approach of the HiTOP and its focus on data-driven dimensions enables individuals to be described more flexibly, depending on the level of specificity required (Kotov et al., 2017). Consequently, the paradigm has the capacity to more accurately and reliably capture the complexity of the clinical manifestations of neurodiverse conditions.

The HiTOP classification system is equally as valuable for research as it is for clinical practice. In their recent review of HiTOP as a research framework, Conway and colleagues (2022)

highlighted that not only do HiTOP dimensions enable researchers to better characterise the behavioural phenotypes of mental health conditions, but the model also has implications for the formulation and testing of theoretical assumptions, as well as the ways in which studies are designed and interpreted. It has previously been shown that continuous (i.e., dimensional) measures of psychopathology show increased reliability and validity over discrete (i.e., categorical) assessments (Markon et al., 2011). In line with such evidence, hierarchical dimensional models (e.g., HiTOP) and their accompanying statistical approaches enable researchers to study clinical phenomena in a way that more reliably reflects the empirical structure of psychopathology (Conway et al., 2022). For instance, factor analysis groups variables based on their pattern of interrelations, and allows for researchers to explore the optimal organisation of variables (e.g., the dimensional structure of conditions), or to confirm whether such hypothesised dimensions fit empirical data (Kotov et al., 2017). In this way, researchers may establish the dimensions relevant to a given domain, allowing for the characterisation of dimensional phenotypes that may serve as potential targets for research or clinical assessment and/or treatment (Conway et al., 2022).

The combination of HiTOP and RDoC in research may enable a more comprehensive understanding and treatment of NDs. For example, a clinical presentation of a particular neurodiverse diagnosis that is captured by the HiTOP framework may be underpinned by several distinct biological processes; understanding the neurobiological mechanisms contributing to such behavioural manifestations may facilitate the development and provision of more adequate, individualised support (Perkins et al., 2020). Conversely, some NDs may show high variability in their behavioural manifestations and/or functional outcomes in spite of sharing biomarkers, leading them to go unidentified despite a comprehensive analysis of their signs using HiTOP dimensions; taking the RDoC approach to studying the neurobiological underpinnings of such heterogeneous conditions may enable the identification of new constructs that would benefit from phenotypic operationalisation, further elucidating novel quantitative accounts of neurodiverse diagnoses (Kotov et al., 2017).

Overall, research demonstrates that the traditional categorical approach does not adequately capture the complexity associated with NDs. Moving towards transdiagnostic dimensional frameworks, as outlined above, may offer a more precise characterisation of NDs, with the potential to improve the assessment, diagnosis, and support of neurodivergent individuals.

1.4.3. The Neurodiversity Approach

Transdiagnostic dimensional research is highly aligned with the neurodiversity approach. The following section will describe the neurodiversity paradigm, and explore how transdiagnostic research can support a shift towards reframing neurodiverse conditions as part of the natural variation in cognition and behaviour, rather than as ‘deficits’ or ‘disorders’.

Neurodiverse diagnoses have traditionally been conceptualised as ‘disorders’ of brain development that undesirably deviate from the accepted ‘norm’ (Pellicano & den Houting, 2022). This perception of NDs has predominantly been influenced by the medical model of disability, where conditions such as autism have been pathologised and perceived as disabilities resulting from a ‘core deficit’ (e.g., biological/psychological) that require appropriate treatment (Chapman, 2021; Llewellyn & Hogan, 2000). Such interventions have focused on altering individual behaviours to be more in line with socially accepted norms, with the aim to reduce disability and enhance daily functioning (Pellicano & den Houting, 2022). Hence, the medicalisation of NDs has placed extreme focus on individual ‘deficits’ through striving to transform ‘disabled’ persons into typically developing individuals, without considering the role that external factors (e.g., social/environmental barriers) may play in disabling such individuals (Dwyer, 2022).

With the aim to move away from the pathologisation of NDs, the neurodiversity approach reconceptualises atypical development as falling along a range of natural diversity that exists in neurodevelopment, where ‘neurotypical’ and ‘neurodivergent’ individuals are regarded as equal, despite differences in the ways in which individuals may think, learn or behave (Pellicano & den Houting, 2022; Fletcher-Watson, 2022). Importantly, the neurodiversity approach posits that disability results from an interaction between the characteristics of a neurodivergent person and their immediate environment (Dwyer, 2022), moving away from the perception that disability is the consequence of an impairment existing *within* an individual. Thus, disability may be addressed by reshaping the environment and/or society (e.g., by changing attitudes), or by teaching disabled individuals adaptive *skills* to aid daily functioning, rather than attempting to make neurodivergent individuals appear more neurotypical (Dwyer, 2022).

Transdiagnostic research is highly aligned with the neurodiversity paradigm, as it does not rely on case-control designs where neurotypical performance is regarded as ‘correct’, with any differences in performance reflecting ‘deficits’; instead, it is concerned with characterising individual profiles of ability in a multidimensional space, which allows for clusters of abilities to be understood as ‘value-neutral’ (Fletcher-Watson, 2022). As previously mentioned, recent transdiagnostic studies have shown that the behavioural profiles of neurodivergent individuals fail to directly map onto diagnostic labels (Mareva et al., 2023). This suggests that the dichotomy between ‘normal’ and ‘abnormal’ is arbitrary and does not reflect the heterogeneous nature of neurodiverse conditions, further supporting the neurodiversity model. Through the identification of dimensions that directly map onto common areas of need, neurodiversity-informed transdiagnostic research has the capacity to capture profiles of needs that are directly related to individual functional outcomes (Fletcher-Watson, 2022). This may enable neurodivergent individuals to gain access to more adequate support and thus improve their lived experience.

A further, and important, benefit of the transdiagnostic research is that it opens the door for the increased involvement of neurodivergent individuals in the guidance of study design and interpretation of research (Fletcher-Watson, 2022). A recent review of participatory approaches (i.e., working with neurodivergent individuals to draw on ‘practical wisdom’ to inform research methods) has recently observed a 5-fold increase in the proportion of studies reporting the involvement of autistic community members (Tan et al., 2025). This is important as the involvement of neurodivergent individuals in different stages of the research process increases its applicability to improving quality of life in the communities studied (Fletcher-Watson et al., 2021). For instance, research has shown that community involvement ensures that interventions are more accessible and directly tailored to fit the community’s needs (e.g., Callard et al., 2012; Pellicano et al., 2014). Through the inclusive nature of transdiagnostic approaches (e.g., community-based recruitment), transdiagnostic research has the potential to increase collaboration between neurodivergent individuals and researchers.

Overall, the transdiagnostic dimensional approach aligns well with the neurodiversity paradigm by characterising individual profiles in a value-neutral, dimensional space, thus supporting a more inclusive understanding of NDs. Importantly, this approach also promotes research that is more responsive to the needs and experiences of neurodivergent individuals.

1.5. Neurodiversity in Adults

There is currently an evident lack of research into how neurodiverse conditions present in adulthood, with the majority of the abovementioned research being conducted with child or adolescent populations. The lack of research into the functioning of neurodivergent adults has been observed across a wide range of NDs (including autism, dyslexia, and ADHD), and has had a substantial adverse impact on the identification of such conditions in adulthood (Lipinski et al., 2022; Sadusky et al., 2021; Goodman et al., 2024). The diagnostic assessment of many NDs is not only grounded in categorical research, but the vast majority of diagnostic criteria are also based on childhood presentations of such conditions (APA, 2013). This is an issue, since the developmental trajectories of many cognitive and mental health processes associated with NDs are dynamic, and inclined to change over time (e.g., Hens & Goidsenhoven, 2023). Thus, the diagnosis of neurodiverse conditions appears to be even more challenging in adulthood, with this difficulty being further exacerbated as a result of the complex and heterogeneous nature of such conditions (Astle et al., 2022). Consequently, whilst research has frequently provided evidence for a lack of access to adequate support in neurodivergent children (Astle et al., 2022), the current state of literature and diagnostic procedures indicates that this may be even more difficult for neurodivergent adults. To this end, research suggests that such a lack of adequate support may be even more critical for neurodivergent adults with co-occurring mental health conditions (e.g., Camm-Crosbie et al., 2018), emphasising the need for further research into both the cognitive and mental health processes of neurodivergent adults.

1.6. Thesis Aims

As discussed in this chapter, there is an evident need for more comprehensive data-driven accounts of neurodiverse conditions that promote a shift away from categorical research (Astle et al., 2022). This is especially important for gaining a deeper understanding of the complex cognitive profiles associated with neurodiverse diagnoses, and the variable interactions between cognitive and mental health processes that often have a significant impact on individual functioning and lived experience (Astle et al., 2022; Rodgers & Ofield, 2018). Given the developmental nature of neurodiverse conditions and the lack of information on how they present in adulthood (e.g., Nigg et al., 2020; Hens & Goidsenhoven, 2023; Lipinski et al., 2022), it is particularly important to yield empirical data that enable a more precise

characterisation of cognitive and mental health functioning in neurodivergent adults. The aim of this thesis is therefore to build on the emerging quantitative research frameworks discussed in this chapter, with a specific focus on cognition and mental health in neurodivergent adults.

This thesis comprises three empirical chapters, consisting of a) a systematic review and meta-analysis of co-occurring ADHD and reading difficulties (RD) across the lifespan, and b) a transdiagnostic study of the cognitive and mental health dimensions associated with broadly sampled NDs in adults (divided into two chapters based on analytical approaches). The specific aims of each empirical chapter are detailed below:

- Chapter 2 aimed to synthesise existing evidence on the co-occurrence rates and cognitive manifestations of ADHD and RD across the lifespan. The prevalence of ADHD+RD and performance across a broad set of cognitive domains were compared between children and adults. This systematic review provided additional rationale for the development of a transdiagnostic protocol for investigating cognitive functioning in neurodivergent adults (outlined in Chapter 3).
- Chapter 4 applied a cluster analysis approach to identify naturally occurring groups within the dataset, based on individual scores along cognitive and mental health dimensions. Data-driven clusters were compared against formal diagnostic labels, and a linear discriminant analysis was performed to identify the processes (i.e., dimensions) that best characterise the underlying structure of the dataset. Thus, the main aim of this chapter was to explore the ways in which individual profiles may be best represented in a multidimensional space of strengths and difficulties in cognition and mental health, and whether data-driven clusters may be more effective at characterising individual functioning compared to traditional diagnostic categories. Ultimately, this chapter aimed to explore the cognitive and/or mental health mechanisms that may underpin the complexity of NDs (in terms of their co-occurrence, individual variation, and/or between-diagnosis overlaps).
- Chapter 5 aimed to build on the initial cluster analysis, through adopting a network analysis approach in order to gain a deeper understanding of the complex (and more subtle) interactions between the cognitive and mental health processes that characterised the present transdiagnostic dataset. This was explored through estimating the network structure of the dataset, identifying the most influential processes, and detecting communities of

functions and their potential ‘bridging’ mechanisms. This chapter helped to further elucidate the mechanisms that may contribute to the complexity of NDs.

1.6.1. Research Questions

Ultimately, the aims of the transdiagnostic study were to address the following research questions:

- 1) In what ways do neurodivergent adults differ on key cognitive and mental health dimensions?
- 2) How can individual profiles be best represented in a multidimensional space of strengths and difficulties?
- 3) Can data-driven transdiagnostic clusters be used to better characterise individual functioning compared to traditional diagnostic categories? (Do data-driven clusters map directly onto traditional diagnostic categories?)
- 4) How can transdiagnostic dimensional research frameworks help to elucidate the mechanisms underlying the complexity of NDs (in terms of between-diagnosis overlaps, individual variation, and/or co-occurring conditions)?

Chapter 2: The cognitive manifestations and prevalence rates of co-occurring ADHD and RD across development: A systematic review and meta-analysis

2.1. Abstract

Background

Attention deficit hyperactivity disorder (ADHD) and reading difficulty (RD) are two of the most frequently co-occurring neurodiverse conditions, but it is unclear whether the rate at which they co-occur remains constant across the lifespan. The co-occurrence of ADHD and RD has also been associated with complex cognitive profiles, but it is unclear whether and/or how the cognitive manifestations of ADHD+RD change throughout development. Thus, the aim of this systematic review was to investigate whether the co-occurrence rates and cognitive manifestations of ADHD+RD change from childhood to adulthood.

Methods

The following databases were searched to identify potential studies for inclusion: Embase, MEDLINE, PsycINFO, Web of Science, CINAHL and PubMed. All sources were accessed on 15th August 2023. Any studies with information on the proportion of children or adults with ADHD+RD were included, as well as studies which assessed the cognitive ability of individuals with ADHD+RD in at least one of the following domains: attention, cognitive flexibility, working memory, inhibition, memory, planning, problem-solving, processing speed and vocabulary. A random-effects proportional meta-analysis was used to synthesise the co-occurrence rates of ADHD+RD across child and adult studies, which were subsequently compared using a *z*-test. Additionally, a random-effects meta-analysis of standardised mean differences was performed to synthesise pooled effect estimates across cognitive domains for children and adults, which were compared using an independent samples *t*-test. The JBI Checklist for Studies Reporting Prevalence Data and JBI Checklist for Analytical Cross-Sectional studies were used to assess risk of bias. This review was pre-registered on PROSPERO (number: CRD42023463062).

Results

Of the 3,981 records identified, 153 studies were eligible. Of these, 98 were included to address the prevalence of co-occurring ADHD+RD (90 child, 8 adult), and 69 were included to address the cognitive manifestations of ADHD+RD (63 child, 6 adult). The majority of studies with

prevalence information demonstrated moderate to high risk of bias, while the majority of studies with cognitive performance data had low to moderate risk of bias. The pooled proportion of ADHD+RD was 0.23 (95% CI [0.20, 0.27]) for child studies, and 0.23 (95% CI [0.12, 0.40]) for adult studies. The pooled co-occurrence estimates differed substantially based on sampling method, with studies employing purposive sampling reporting a significantly higher proportion of individuals with ADHD+RD, compared to studies employing mixed sampling methods ($z = -29.30$, $p < .001$). The pooled effect estimate for overall cognitive performance was -1.11 (SE = 0.09, 95% CI [-1.28, -0.95]) for children, and -0.70 (SE = 0.11, 95% CI [-0.91, -0.49]) for adults. There were no significant differences between children and adults in any of the cognitive domains assessed. Heterogeneity levels remained substantially high across all syntheses (all I^2 values above the 75% threshold).

Conclusions

The results of this systematic review suggest that the co-occurrence of ADHD+RD and the level of cognitive difficulty experienced by individuals with ADHD+RD remain constant across development. The findings of this review emphasise the need for further research into the co-occurrence of ADHD+RD in adults, and are discussed in light of practical implications for the assessment, diagnosis, and support of co-occurring ADHD+RD across development, particularly in adults. The findings add to the emerging pool of transdiagnostic literature on neurodiverse conditions, and highlight several methodological considerations for future research.

2.2. Introduction

Attention deficit hyperactivity disorder (ADHD) is a neurodiverse condition characterised by patterns of inattention, hyperactivity and impulsivity, which can significantly affect daily functioning (APA, 2013). Reading difficulty (RD) is a specific learning disorder, marked by weaknesses in word decoding, reading fluency, and comprehension (APA, 2013). The term ‘dyslexia’ (difficulty in word decoding) is often used interchangeably with RD (Lyon et al., 2003). Given that dyslexia is the most common type of learning difficulty (Yang et al., 2022), this systematic review will predominantly focus on dyslexia literature.

ADHD and RD are two of the most common neurodiverse diagnoses (NDs), with prevalence rates suggesting that approximately 7% of the population may have either of these conditions (Thomas et al., 2015; Yang et al., 2022). The two NDs often co-occur, with estimated bidirectional co-occurrence rates ranging from 25-40% in both children and adults (Willcutt & Pennington, 2000; DuPaul et al., 2013). Research has shown that both conditions have a significant impact on individual lived experience. For example, ADHD is often associated with an increased susceptibility to mental health problems and risk-taking behaviour (Langley et al., 2023), whilst RD has frequently been shown to have an adverse impact on academic and professional attainment (e.g., Elgendi et al., 2021, Morris & Turnbull, 2007). Importantly, a large body of evidence has shown that children with a combination of ADHD+RD are at a greater risk for poor long-term outcomes that are likely to persist into adulthood (Sexton et al., 2012).

Despite the high rates of co-occurrence, research has often focused on ADHD and RD in isolation, characterising each condition by a distinct set of ‘core’ difficulties in cognitive function. For example, ADHD has primarily been associated with weaknesses in attentional control and working memory (e.g., Barkley, 1997; Claesdotter et al., 2018), whilst RD has predominantly been attributed to differences in phonological processing (e.g., Lyon et al., 2003). However, substantial research evidence has shown that there is considerable cognitive overlap between the two NDs, such as shared processing speed difficulties (e.g., McGrath et al., 2011), or working memory problems (Tiffin-Richards et al., 2008). There has been notable uncertainty regarding the cognitive manifestations of ADHD+RD. For instance, earlier studies have suggested that individuals with co-occurring ADHD+RD may simply display a combination of the cognitive weaknesses associated with the two conditions (i.e., a ‘double

dissociation'; e.g., Willcutt et al., 2001), whilst others have suggested that ADHD+RD is characterised by a distinct cognitive profile with unique difficulties that are not present in either condition alone (e.g., Bental & Tirosh, 2007). Importantly, research has also shown that co-occurring ADHD+RD may manifest in the form of more severe difficulties in cognitive functioning (e.g., Mayes et al., 2000), and more complicated cognitive profiles (Wang & Chung, 2018).

The key cognitive domains that have frequently been associated with ADHD and RD include working memory, general memory, attention, inhibition, cognitive flexibility, planning, problem-solving, processing speed and vocabulary. A large body of evidence has shown that executive function (including working memory, inhibition, and cognitive flexibility) may be a shared difficulty underpinning the co-occurrence of ADHD with dyslexia (Lonergan et al., 2019). Difficulties in broader executive functioning (such as planning and problem-solving) have also been associated with both ADHD and RD (Patros et al., 2019; Capri et al., 2019; Akyurek & Bumin, 2019). Similarly, processing speed and attention difficulties have been observed in both dyslexic individuals and those with ADHD (Lewandowska et al., 2014; de Oliveira et al., 2014; Kibby et al., 2019; McGrath et al., 2011). Further, broader memory difficulties have been shown to result from encoding (i.e., working memory) problems in ADHD (Skodzik et al., 2017). In contrast, individuals with RD have been shown to display relative strengths in memory (especially episodic memory) due to relying on such strategies to support long-term memory abilities (Eide & Eide, 2011). Finally, weak vocabulary skills have been associated with broader reading problems in individuals with RD (Lefèvre et al., 2025), while vocabulary problems have been associated with poor reading comprehension in individuals with ADHD (Martinussen & Mackenzie, 2015). Despite such evidence, there is considerable uncertainty around how each of these cognitive functions manifest in co-occurring ADHD+RD, and how they may differ between children and adults with complex co-occurring profiles. This underscores the need for a broader evidence synthesis across a range of cognitive domains, in order to gain a more comprehensive understanding of cognitive functioning in ADHD+RD, and how this may differ across developmental stages.

The uncertainty concerning the cognitive manifestations of co-occurring ADHD+RD has direct implications for diagnostic processes. Current research suggests that many individuals with complex profiles (e.g., those with co-occurring conditions) often fail to meet diagnostic criteria, which impacts their ability to access relevant support (Astle et al., 2022). Many individuals

will also often receive an incorrect or incomplete diagnosis (e.g., either ADHD or RD), typically due to the misidentification of one or the other because of overlapping characteristics, which frequently results in the provision of inadequate support (Baraskewich & McMorris, 2019; Astle et al., 2022). These practical issues highlight the crucial need for a deeper understanding of the cognitive profiles associated with co-occurring ADHD+RD, as such information could provide a basis for the enhancement of diagnostic tools, and ultimately increase individual access to adequate support.

The challenges associated with the identification of co-occurring ADHD+RD raise questions around the true co-occurrence rates of these conditions. As previously mentioned, it has been estimated that around 25-40% of individuals with ADHD or RD will also meet criteria for the other condition (DuPaul et al., 2013), yet it is unclear how many individuals display signs of these conditions that simply fail to align with traditional diagnostic criteria. Further, it is important to note that whilst the onset of ADHD and RD predominantly happens in childhood (Faraone & Biederman, 2016; Pennington & Lefly, 2001), both conditions have been shown to persist into adulthood (Asherson & Agnew-Blais, 2019; Reis et al., 2020). Some studies have suggested that the characteristics of ADHD and RD alone decline with age; for instance, due to compensatory strategies developed to ‘mask’ difficulties associated with the two conditions (Faraone et al., 2006; MacCullagh et al., 2017; van der Putten et al., 2024). This suggests that the identification of ADHD and RD may be even more difficult in adulthood, and creates additional uncertainty around current prevalence estimates of ADHD and/or RD across the lifespan.

This uncertainty is further exacerbated by the fact that current diagnostic criteria for both ADHD and RD are based on childhood presentations of the two conditions (APA, 2022), with a lack of research into how ADHD and/or RD may manifest in adulthood, as the majority of studies have been conducted with child populations. It has recently been suggested that most diagnostic assessments of ADHD in adults rely on retrospective reporting of childhood experiences of ADHD (Vater et al., 2024), whilst diagnostic assessments of RD in adulthood have been shown to predominantly rely on standardised practices used with children (Sadusky et al., 2021). Therefore, there is currently an evident lack of diagnostic measures appropriate for different developmental stages across the lifespan, which emphasises the need for a deeper understanding of how the cognitive manifestations of ADHD and/or RD may change from childhood to adulthood. This is especially important for understanding the co-occurrence of

these two conditions in adulthood, as research has shown that ADHD in adults is often accompanied by an increase in the co-occurrence of learning difficulties, such as RD (Taylor et al., 2019).

The uncertainty regarding the co-occurrence rates and cognitive manifestations of ADHD+RD has also been rooted in methodological inconsistencies. First, several approaches have been used to define RD (specifically dyslexia) in research, inherently resulting in substantial variability across studies. The most common approaches to defining RD are the ‘discrepancy’ and ‘low achievement’ models, where the presence of RD is defined by either a significant discrepancy between expected and actual reading attainment (i.e., a discrepancy between literacy skills and IQ), or consistently low academic achievement for age (Snowling et al., 2020). The number of participants identified as presenting with RD is likely to vary based on the criteria used to define the condition, which has the capacity to directly affect prevalence estimates, as well as the cognitive strengths and difficulties that may be identified in a given sample.

Further, the sampling methods used to recruit participants can also introduce relative inconsistencies. Some studies may adopt a ‘mixed sampling’ approach, where participants may be broadly sampled from schools, research centres or communities, with a given difficulty being confirmed by pre-ascertained assessments within the study. This type of sampling may include individuals with subthreshold or ‘subclinical’ presentations of ADHD and/or RD. On the other hand, other studies may employ a more strict ‘purposive sampling’ approach, where participants may be specifically selected based on prior diagnoses, or clinical referrals. This method of sampling may be more specific, but may lack sensitivity in identifying individuals with ADHD and/or RD whose profiles do not directly align with traditional diagnostic criteria (Astle et al., 2022). Finally, there are a multitude of standardised measures used to assess performance across different cognitive domains, with each study employing a different combination of assessments. Each of these assessments may measure different outcomes on different scales, such as reaction time or accuracy, or even assess cognition via self/informant-report. This creates complexity around comparing performance across different measures and cognitive domains. Ultimately, there are several methodological sources of variability that may contribute to the overall uncertainty regarding the prevalence and cognitive manifestations of co-occurring ADHD+RD in both children and adults.

2.2.1. Aims

Given the uncertainty around how the cognitive manifestations and co-occurrence rates of ADHD+RD may differ across development, this systematic review aims to:

- a) Quantify the prevalence of co-occurring ADHD+RD across development, and investigate whether the prevalence of ADHD+RD changes from childhood to adulthood.
- b) Explore whether variability in prevalence estimates of co-occurring ADHD+RD is due to methodological differences (i.e., sampling methods, or criteria used to define RD).
- c) Characterise the cognitive profiles of children and adults with ADHD+RD, and explore potential age-related differences across cognitive domains.

The review has been divided into two syntheses, addressing the following research questions:

Research Question 1 (RQ1)

What is the prevalence of co-occurring ADHD+RD in children and adults?

Research Question 2 (RQ2)

What are the cognitive manifestations of ADHD+RD in childhood and adulthood?

This review is important given the practical challenges in assessing co-occurring ADHD+RD across the lifespan, especially in adults. There is currently a lack of research directly comparing the co-occurrence rates of ADHD+RD in children and adults, as well as a lack of investigations exploring how the cognitive manifestations of co-occurring ADHD+RD may differ from childhood to adulthood. To our knowledge, this is the first systematic review that has attempted to synthesise and assess existing evidence regarding the prevalence and cognitive manifestations of co-occurring ADHD+RD from a developmental perspective.

This review has the capacity to highlight potential differences in the co-occurrence rates of ADHD+RD across the lifespan. The findings of this review may also help to characterise potential cognitive mechanisms through which ADHD+RD may present differently in children and adults, providing the opportunity to inform the assessment and identification of the combined condition in adulthood. This review may also reveal targets for future support concerned with improving the cognitive functioning of individuals with ADHD+RD.

Importantly, through adopting a dimensional approach (i.e., synthesising evidence across a broader range of cognitive domains), the findings of this review will contribute to the emerging transdiagnostic dimensional literature concerning neurodiverse conditions, and may highlight potential methodological considerations or objectives for future research in this area.

2.3. Methods

This review was reported in line with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Page et al., 2021). See Appendix A (Supplementary Materials 1).

2.3.1. Eligibility Criteria

Studies were included to address RQ1 (prevalence) if they met the following criteria:

- Any study design (including cohort, case-control, cross-sectional, longitudinal).
- Any studies that quantified the prevalence of the co-occurrence of ADHD (or attention difficulties) and dyslexia (or reading difficulties); i.e., formal diagnoses or subclinical conditions (could be in academic/clinical settings or general population).
- Population-based, community-based, or epidemiological studies.
- Studies conducted with children, adolescents or adults in any country.
- Studies assessing how ADHD predicts reading difficulty or vice versa, provided that the data were not extracted from baseline assessments, but at Time 2 where both difficulties were present.

Studies were excluded to address RQ1 (prevalence) based on the following criteria:

- Studies that did not quantify the co-occurrence of ADHD+RD (or where the frequency of co-occurrence could not be calculated).
- Studies where learning difficulty was not defined as reading difficulties, or prevalence was not reported for ADHD+RD specifically.
- Studies where the ADHD+RD group included individuals with other conditions (e.g., specific language impairments, perceptual-motor functioning difficulties, dyscalculia, or autism).

- Studies that reported on the prevalence of ADHD or RD separately, rather than as co-occurring conditions.
- Studies that were not published in journal articles and/or not peer reviewed (e.g., theses, dissertations, conference abstracts, grey literature), and any previous systematic reviews or case studies.
- Studies not published in English.

Studies were included to address RQ2 (manifestation) if they met the following criteria:

- Any study design (including cohort, case-control, cross-sectional, longitudinal).
- Studies assessing the cognitive profiles of children, adolescents or adults with formal ADHD and co-occurring dyslexia diagnoses (in any country).
- Prospective studies where no formal diagnosis is yet present (e.g., studies within academic settings where groups were defined by attention and reading difficulties).
- Quantitative studies that provided quantifiable cognitive data from standardised neurocognitive or psychological assessments (i.e., means and standard deviations).
- Studies focusing on the core cognitive domains of executive function (e.g., inhibition, working memory), attention (e.g., processing speed, sustained attention), learning and memory (e.g., implicit learning, cued/free recall), and language (e.g., vocabulary).

Studies were excluded to address RQ2 (manifestation) based on the following criteria:

- Qualitative studies.
- Correlational studies (e.g., examining how cognition predicts reading ability in individuals with ADHD), where a co-occurring ADHD+RD group was not present.
- Studies that did not report mean scores and standard deviations (e.g., data were solely reported in graphs/charts).
- Neuroimaging or genetic studies without any cognitive performance measures.
- Studies where learning difficulty was not defined as reading difficulties.
- Studies that solely assessed reading, attention or academic performance, without considering cognitive processes that underpin ADHD+RD.
- Studies assessing perceptual-motor functioning (e.g., visual perception, motor co-ordination), social cognition (e.g., theory of mind, recognition of emotions),

specific language impairments, or intelligence (without any cognitive subscales), unless relevant cognitive domains were also assessed (see inclusion criteria).

- Studies that employed unstandardised cognitive measures.
- Studies that were not published in journal articles (e.g., theses, dissertations, conference abstracts, grey literature), and any previous systematic reviews or case studies.
- Studies not published in English.

2.3.1.1. Study Grouping

Studies were grouped for syntheses based on the research question addressed. Some studies addressed both RQ1 and RQ2, in which case they were included twice, with different data being extracted each time (i.e., prevalence rates vs. cognitive performance scores). Studies were also grouped based on age, with studies assessing participants aged < 18 forming the ‘Child’ group, and studies assessing individuals aged over 18 forming the ‘Adult’ group. Moreover, in order to address RQ1, studies were split into further subgroups based on sampling methods (i.e., mixed vs. purposive sampling) and models used to define reading difficulty (i.e., discrepancy model, low achievement model, both discrepancy and low achievement, or unclear/other). Similarly, in order to address RQ2, the studies were also sub-grouped based on cognitive domain assessed (specifically attention, cognitive flexibility, working memory, inhibition, memory, planning, problem-solving, processing speed and vocabulary).

2.3.2. Information Sources and Search Strategy

Candidate search terms were identified by reviewing key words in titles, abstracts and subject indexing of relevant literature. A draft search strategy was developed using these terms, with additional search terms being identified from the results of the initial strategy. The search strategies for each database were developed by an information specialist (GZ) and reviewed by the first author (KM) for relevance. The following databases were searched to identify potential studies for inclusion: Embase, MEDLINE, PsycINFO, Web of Science, CINAHL and PubMed. All sources were accessed on 15/08/2023, at which point all identified records were retrieved, with no further searches being conducted. PubMed results were not included in the review as too many records were retrieved due to the unavailability of a proximity operator during the

search process. However, PubMed covers MEDLINE, which was searched through Web of Science, thus no potentially relevant records were excluded. The full search strategies are presented in Appendix B (Supplementary Materials 1).

2.3.3. Selection Process

Title and abstract screening of all retrieved records ($n = 3,981$) was conducted by the first author (KM). Full-text screening ($n = 382$) was conducted independently by the same reviewer, with 50% of full-text records also assessed by a second reviewer (JW). Any discrepancies regarding inclusion decisions were resolved through discussion. The initial inter-rater agreement for full-text screening was 75.2%, which increased to 85.6% after discussion. The second reviewer was uncertain on the remaining subset of papers, thus final decisions on study inclusion were made by the first author (KM). EndNote (Version X9.3.3; The EndNote Team, 2013) was used to identify and remove any duplicates. No other automation tools were used in the screening process. Study investigators were not contacted for further information. Please refer to Figure 1 in the Results section for a detailed flow diagram of the selection process.

2.3.4. Data Extraction Process

Key study characteristics were extracted by the first author (KM), and imported into an Excel spreadsheet with predefined columns for relevant domains. To ensure reliability, a second reviewer (JW) independently extracted key study details from 50% of included studies during full text screening. Any discrepancies were resolved through discussion (see above for inter-rater agreement). A separate Excel spreadsheet was designed for the extraction of quantitative data from each included study. These data were extracted by the first author (KM) only. Study investigators were not contacted for additional information; if relevant data were not available, papers were excluded from the review. No automation tools were used in the data extraction process.

2.3.5. Data Items

The following data were extracted from each included study to address RQ1 and RQ2:

2.3.5.1. Research Question 1

Data were sought for any reported proportions of children or adults with co-occurring ADHD+RD. This could be reported for the purposes of sample characterisation (e.g., number of participants with ADHD+RD from the total study sample), or as prevalence rates in epidemiological studies (e.g., rate of ADHD+RD in a given population). The co-occurrence rate could be determined from the proportion of individuals with ADHD+RD from a given sample or population, or the proportion of RD in ADHD (or vice versa). If a study reported different rates of co-occurring RD in different ADHD subtypes, the rate of RD was calculated for the ADHD group as a whole. In cases where multiple co-occurrence rates were reported for different types of reading difficulty (e.g., accuracy vs. fluency) in ADHD, an average was calculated. In studies where different rates of co-occurring ADHD+RD were reported at baseline and follow-up, baseline data were extracted.

2.3.5.2. Research Question 2

Data were sought for performance on cognitive measures of working memory, general memory, attention, inhibition, cognitive flexibility, planning, problem-solving, processing speed and vocabulary. Any standardised measure of cognitive function relating to these domains was eligible for inclusion. In cases where multiple measures were used to assess the same outcome domain, tasks which directly assessed cognitive performance were prioritised over self-report or informant-report measures. If multiple data were reported for the same cognitive domain across different tasks, an average standardised effect size was calculated for the given domain. If different data were reported for multiple cognitive abilities relating to the same cognitive construct (e.g., interference vs. inhibition, shifting/switching vs. cognitive flexibility), an average standardised effect size was calculated. If a study reported baseline and follow-up data, baseline data were extracted. In cases where multiple studies by the same author were conducted using the same sample and similar data were reported (e.g., several working memory measures), only one of those results were extracted to avoid bias and redundancy. To this end, the more widely used measure or larger effect size was prioritised to increase robustness of results. Further, if studies reported results for different ADHD subtypes, data were extracted for the combined type to ensure consistency, since the majority of studies reported on the combined type. In cases where results were reported for different types of reading difficulty, an average standardised effect size was calculated.

2.3.5.3. Other Variables

Data were collected on participant age range (i.e., under or over 18) in order to group studies for comparisons between children/adolescents and adults (for both RQ1 and RQ2). Further information was extracted for the sampling methods used (i.e., mixed or purposive sampling), and the model used to define RD (i.e., discrepancy, low achievement model, discrepancy and low achievement, or unclear/other), with the aim to investigate possible causes of heterogeneity among co-occurrence rates.

2.3.5.4. Handling of Missing or Unclear Information

In studies where the exact participant age was unclear, this was estimated from the current education level reported (e.g., in studies conducted with students). If the definition of RD was unclear, these studies were classified as ‘Unclear or Other’. Studies with unclear or missing quantitative data (e.g., means and standard deviations) were not included in the extraction spreadsheet and excluded from the review ($n = 57$). If key study characteristics (e.g., diagnostic criteria) were missing or unclear, this was addressed in the risk of bias assessment (see below).

2.3.6. Risk of Bias Assessment

The risk of bias in the included studies was assessed using critical appraisal tools developed by the Joanna Briggs Institute (JBI). The following tools were applied depending on the research question addressed (note that studies addressing both research questions were included in both risk of bias assessments):

1. The **JBI Checklist for Studies Reporting Prevalence Data** (Munn et al., 2015; Appendix C, Supplementary Materials 1) was used to evaluate studies examining the prevalence of co-occurring ADHD+RD.
2. The **JBI Checklist for Analytical Cross-Sectional Studies** (Moola et al., 2020; Appendix D, Supplementary Materials 1) was used to assess studies investigating cognitive performance in ADHD+RD.

The risk of bias assessments focused on the following domains: a) bias due to participant sampling (including sampling frame, sampling methods, inclusion criteria, and sample size), b) bias arising from the lack of demographic information, c) bias due to inappropriate statistical analyses, d) bias in methods used to identify or confirm the conditions, e) bias due to validity and reliability of cognitive assessments, f) bias arising from potential confounding variables, g) bias due to inadequate response or participation rates. An overall risk of bias judgment was not made across domains as the JBI critical appraisal tools do not provide an overall risk of bias score or classification. Note that Item 3 (“Was the exposure measured in a valid and reliable way?”) in the JBI Checklist for Analytical Cross-Sectional Studies was adapted such that ‘exposure’ was understood as ‘condition’. Therefore, this item was used to evaluate the validity and reliability of the measures used to confirm ADHD and RD diagnoses and/or difficulties. To this end, Item 4 (“Were objective, standard criteria used for the measurement of the condition?”) was used to assess the validity of the criteria used to define the conditions (e.g., DSM criteria).

The first author (KM) conducted the risk of bias assessment for all included studies, while a second reviewer (JW) independently assessed 1/3 of the studies (in alphabetical order). Any disagreements were resolved through discussion until consensus was reached. The overall inter-rater agreement across both critical appraisal tools was 80.6%. Study investigators were not contacted to confirm or obtain any additional information. No automation tools were used in the risk of bias assessment.

2.3.7. Effect Measures

For studies addressing RQ1, raw proportions (i.e., number of ADHD+RD cases divided by the total sample size) were used to conduct a proportional meta-analysis. This method was chosen because many studies did not present effect measures such as odds or risk ratios, but instead reported the number or percentage of participants with ADHD+RD. A meta-analysis of proportions allows for such dichotomous data to be included in the analysis to generate a single summary proportion estimate and its variance (Barker et al., 2021).

For studies addressing RQ2 (i.e., continuous data), standardised mean difference (SMD) effect size (Hedges’ *g* or Cohen’s *d*) was used to allow for direct comparisons across studies with varying cognitive measures yielding different types of data (e.g., accuracy, reaction time) on

varying scales. Hedges' g was calculated for studies which reported all relevant data (i.e., means, standard deviations and sample sizes) for both the control and the ADHD+RD group. In cases where multiple tasks measured the same cognitive domain, and sample sizes differed across tasks for the clinical and/or control group, a weighted average Hedges' g was computed to account for sample size variations. In studies with missing data (i.e., unavailable control group), published normative data were used as a substitute. To this end, Cohen's d was calculated instead of Hedges' g because it did not require sample size information for the calculation of standard errors. This approach ensured that standardised effect sizes could be computed even when the exact sample size was uncertain for normative data. Finally, the direction of effect sizes was standardised by reversing the sign where necessary, such that all higher scores reflected higher performance in order to ensure consistency across studies.

2.3.8. Synthesis Methods

For studies addressing RQ1, the following characteristics were tabulated to decide which studies were eligible for each synthesis:

1. Age Group (Child or Adult)
2. Sampling Method (Mixed Sampling or Purposive Sampling)
3. Definition of RD (Discrepancy Model, Low Achievement Model, Discrepancy and Low Achievement Model, or Unclear/Other Model)

If a given study presented multiple characteristics (e.g., a child study using purposive sampling and a discrepancy model to define RD), it was included in each subgroup analysis.

For studies addressing RQ2, the following characteristics were tabulated to decide which studies were eligible for each synthesis:

1. Age Group (Child or Adult)
2. Cognitive Domain Assessed (Working Memory, General Memory, Attention, Inhibition, Cognitive Flexibility, Planning, Problem-Solving, Processing Speed, Vocabulary)

If a given study assessed several cognitive domains (e.g., a child study assessing inhibition, planning and vocabulary), it was included in each subgroup analysis for comparison with adults.

Raw data from individual studies were displayed in summary tables, including total sample sizes with proportions of co-occurring ADHD+RD (RQ1), and effect sizes with standard errors (RQ2), with corresponding details about eligibility for each synthesis (Tables 3 and 4, Results section). Further, proportions (RQ1) and effect estimates (RQ2) with 95% confidence intervals were presented for each study in separate summary tables for children and adults (see Results section). Results from individual studies were also visually displayed in separate forest plots for children and adults (see Figures 2-11). For studies addressing RQ1, these plots were further categorised by sampling method and RD definition (see Appendices E-F, Supplementary Materials 1). For studies addressing RQ2, the plots were categorised by cognitive domain.

2.3.9. Meta-Analysis of Proportions (RQ1)

To synthesise the prevalence of co-occurring ADHD+RD across studies, a meta-analysis of proportions was performed in R Studio (Version 4.4.3; R Core Team, 2025), using the *meta* package (Balduzzi et al., 2019). Raw proportional data were logit-transformed to ensure homogeneity of variances required for parametric statistical methods employed in the meta-analysis (Seiffert et al., 2024). A random-effects model was favoured over a fixed-effects approach, since the assumption that there is one true estimate is unlikely to hold for proportional data synthesised from several independent studies (Barker et al., 2021). This is because factors measured by proportions typically vary due to differences in sample characteristics (Saha et al., 2005), or other factors such as diagnostic criteria and assessment tools in this case. Individual study proportions were weighted using inverse-variance weighting, ensuring that studies with higher precision (i.e., larger studies with lower variance) received a greater weighting and contributed more to the overall prevalence estimate (Deeks et al., 2019). This approach helps to mitigate potential bias of weighting from smaller-scale studies with higher variance (Marín-Martínez & Sánchez-Meca, 2010).

The Restricted Maximum Likelihood (REML) estimator (Viechtbauer, 2005) was used to estimate between-study variance. REML is the default method in the *meta* package (Balduzzi et al., 2019), and has been shown to yield more accurate estimates of variance through correcting for degrees of freedom lost by estimating fixed effect coefficients (Maestrini et al., 2024). Further, the Likelihood Ratio Test (LRT) was used to calculate the confidence interval for the summary effect as it is more robust to violations of assumptions (such as non-normality),

and therefore more reliable than the Wald test (Banerjee & Wellner, 2005). Additionally, exact confidence intervals for individual study results were calculated using the Clopper-Pearson method (Clopper & Pearson, 1934).

2.3.9.1. Heterogeneity Analyses

The presence and extent of statistical heterogeneity among studies were assessed by calculating Tau-squared (τ^2) and I-squared (I^2). The τ^2 statistic estimates the variance of true effects across studies, with values closer to 1 (than 0) indicating greater variability (Borenstein, 2023). The I^2 statistic quantifies the percentage of variability across studies that is attributable to true differences, rather than chance (Higgins & Thompson, 2002), with values of 75% or above suggesting substantial heterogeneity (Deeks et al., 2008). It has recently been emphasised that high I^2 values in the context of proportional meta-analyses do not automatically suggest inconsistency in findings, given the heterogeneous nature of prevalence estimates resulting from expected variance due to study characteristics (Barker et al., 2021). I^2 should therefore be interpreted with caution.

2.3.9.2. Subgroup Analyses

To explore potential sources of heterogeneity, subgroup analyses were performed (using the *meta* package; Balduzzi et al., 2019) based on key study characteristics, including age group (child vs. adult), sampling method (mixed vs. purposive sampling), and model used to define RD (discrepancy, low achievement, discrepancy and low achievement, or unclear/other). Each study was included in multiple subgroup analyses based on the above factors. For child and adult studies, separate meta-analyses of proportions were conducted and subsequently compared using a z -test, for statistically comparing proportions between two independent groups (Fleiss et al., 2003). The same approach was used to compare proportions between studies using mixed or purposive sampling. The z -statistic, p -value, 95% confidence intervals and effect sizes (Cohen's h) were used to determine the significance of differences in proportions between these subgroups. Further, separate meta-analyses of proportions were conducted for different RD definitions and subsequently compared using a Chi-squared test (χ^2). This test is appropriate for evaluating whether proportions differ significantly across multiple groups (Cochran, 1956). Here, the χ^2 statistic and p -value were used to determine the significance of between-group differences. The effect size for Chi-squared (Cramér's V) was

not calculated as it is not included in the default *meta* package. Within-subgroup heterogeneity was assessed using the same method as above (via calculating τ^2 and I^2).

2.3.9.3. Publication Bias

It has recently been suggested that the standard procedures for evaluating publication bias (i.e., funnel plots and Egger's test) are not recommended for meta-analyses of proportions, given that these tests have been developed in the context of comparative data, and under the assumption that significant results are published more frequently than null results (Barker et al., 2021). Instead, risk of bias due to missing evidence was assessed using Doi plots (Furuya-Kanamori et al., 2018), which have been proposed to be a more accurate assessment of publication bias for studies addressing prevalence and proportions (Cheema et al., 2022). The LFK index was used to statistically detect and quantify asymmetry of study effects in Doi plots; this method has been shown to display higher sensitivity than the widely used Egger's test in proportional meta-analyses (Furuya-Kanamori et al., 2018). An LFK index outside the range of -1 and +1 was interpreted as indicating asymmetry and therefore publication bias (Furuya-Kanamori et al., 2018). This analysis was performed using the *metasens* package (Schwarzer et al., 2023).

2.3.10. Meta-Analysis of Standardised Mean Differences (RQ2)

To synthesise the cognitive performance of children and adults with co-occurring ADHD+RD, a meta-analysis of standardised mean differences (SMD) was performed in R Studio (Version 4.4.3; R Core Team, 2025), using the *metafor* package (Viechtbauer, 2010). This method is appropriate for calculating pooled estimates from studies assessing continuous outcomes on a variety of different scales (Higgins et al., 2024). Due to the lack of adult studies and unavailability of adult data for certain cognitive domains, performing a meta-analysis to calculate pooled estimates across child and adult data was not appropriate (Lensen, 2023). Instead, separate meta-analyses were conducted for children and adults to determine the pooled effect size within each age group.

A random-effects model was selected as it assumes that the observed effect estimate can vary due to real differences across studies, as well as other factors such as sampling variability or cognitive measures used (Riley et al., 2011), making this model well suited for the

heterogeneous nature of the included studies. Individual study effect sizes were weighted using inverse-variance weighting to ensure that studies with lower variance contributed more to the overall estimate (Deeks et al., 2019). Similarly to the previous section, the REML method was used to estimate between-study variance, the LRT was used to calculate the confidence interval for the summary effect, and the Clopper-Pearson approach was used to determine exact confidence intervals for individual study results. Additionally, I-squared (I^2) and Tau-squared (τ^2) were used to evaluate the presence and extent of statistical heterogeneity among study results (see previous section for a more detailed description).

2.3.10.1. Meta-Regression and Independent Samples T-Test

To explore potential causes of heterogeneity, separate meta-regressions were conducted for child and adult studies, including cognitive domain as a moderator with 9 levels (working memory, general memory, attention, inhibition, cognitive flexibility, processing speed, planning, problem-solving and vocabulary). This analysis aimed to identify differences in effect sizes across cognitive domains within each age group, and to clarify how each domain contributed to the overall variability in pooled effect sizes. A mixed-effects model was used to account for both within-study and between-study variability, considering both fixed and random effects (da Silveira et al., 2023). A z -test was used to determine whether a given cognitive domain significantly influenced the overall estimate, whilst R^2 was calculated to estimate how much of the total heterogeneity was accounted for by differences in cognitive domain (Raudenbush, 2009). I-squared (I^2) and Tau-squared (τ^2) were used to assess residual heterogeneity after accounting for the moderator. All meta-regression analyses were performed using the *metafor* package (Viechtbauer, 2010).

Notably, it has been recommended that there should be at least 10 studies available for every factor modelled in a meta-regression to prevent unreliable results due to insufficient data (Higgins et al., 2024). Therefore, given the small number of adult studies included (precisely 6), the results of the meta-regression should be interpreted with caution. To this end, planned meta-regressions using age group as a moderator to investigate age differences in pooled estimates were not feasible, even for cognitive domains for which both child and adult data were available. Instead, independent samples t -tests were used to statistically compare age group differences in effect sizes across cognitive domains for which both child and adult data were available. All t -tests were performed in jamovi (Version 2.6; The jamovi project, 2025).

2.3.10.2. Publication Bias

To assess risk of bias due to missing results in studies addressing RQ2, funnel plots were generated for meta-analyses of child and adult data and visually inspected for asymmetry. Egger's regression test was conducted to statistically evaluate asymmetry, with $p < .05$ indicating publication bias, or small study effects (Egger et al., 1997). In order to account for potential publication bias, the trim-and-fill method was applied to estimate the number of missing studies and adjust the summary effect estimate accordingly (Duval & Tweedie, 2000). This method has been suggested to be effective for statistically imputing missing studies in meta-analyses where extracting unpublished results is not feasible (Shi & Lin, 2019). It is important to note that the trim-and-fill method is not directly applicable to analyses with multiple outcomes or moderators, such as meta-regression, and was therefore only applied to the main meta-analysis if bias was present. All publication bias analyses were conducted using the *metafor* package (Viechtbauer, 2010).

2.3.11. Sensitivity Analyses

To assess the robustness of the synthesised results, a leave-one-out (LOO) sensitivity analysis was conducted using the *loo* R package (Vehtari et al., 2017). This method involves the sequential exclusion of one study at a time to assess how much the overall estimate is influenced by each individual study (Vehtari et al., 2017). Consistency in recalculated logit-transformed effect sizes, heterogeneity statistics, and 95% confidence intervals can be used to infer robustness and stability of the overall estimate (Borenstein et al., 2009; Viechtbauer, 2010). The LOO sensitivity analysis was conducted for the proportional meta-analysis (including subgroup analyses), as well as for the meta-analyses of standardised mean differences (including meta-regressions).

2.3.12. Certainty Assessment

To assess certainty in the body of evidence, a simplified version of the Grading of Recommendations, Assessment, Development and Evaluation (GRADE) framework (Balshem et al., 2011) was employed. The first author (KM) independently assessed the certainty of all included studies across the following domains: study limitations, consistency of effect,

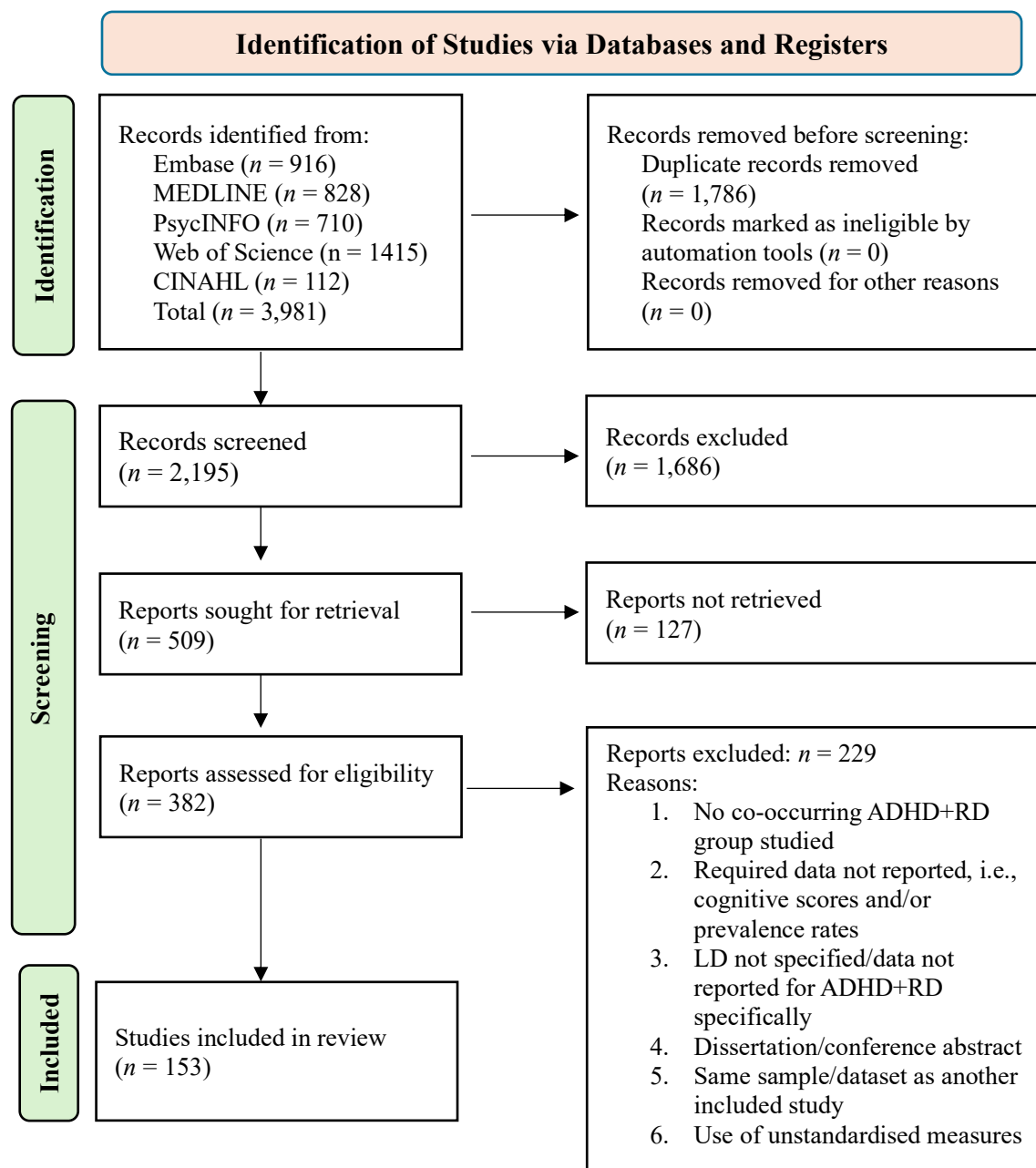
imprecision, and publication bias. Indirectness was not assessed because studies were only included in the review if they directly addressed the research questions in terms of the population and outcomes measured. Study limitations were evaluated based on sampling methods, tools used to assess or confirm ADHD and RD, and appropriateness of statistical analyses (evaluated in the risk of bias assessment using JBI critical appraisal tools). Consistency of effect was assessed using statistical tests of heterogeneity (I^2) calculated for primary child and adult meta-analyses of proportions and standardised mean differences. Imprecision was evaluated based on the width of confidence intervals of overall effect estimates for child and adult meta-analyses. Finally, publication bias was evaluated using Doi plots and the LFK index for studies addressing prevalence, whilst funnel plots and Egger's test results were evaluated for studies addressing cognitive performance. The certainty of evidence was assessed as high, moderate, or low. Study investigators were not contacted to confirm or obtain any further information. No automation tools were used in the assessment of certainty. The results were presented in a summary table with columns corresponding to each domain assessed (see Results section).

2.4. Results

2.4.1. Study Selection

After searching each database, a total of 3,981 records were identified for screening. After the removal of 1,786 duplicates, the titles and abstracts of 2,195 records were screened. After the exclusion of 1,686 records, 509 were sought for retrieval. Of these, 127 could not be retrieved, thus 382 reports were assessed for eligibility through full-text screening. A final 153 reports were included in the review, of which 98 addressed RQ1 (prevalence), with the remaining 69 addressing RQ2 (cognitive performance). This information is presented in Figure 1. Four studies that appeared to meet the inclusion criteria were excluded due to having used the same sample or dataset as another study included in the review (which provided more information, or the same but more robust data). These studies included Schuerholz et al. (1995) and Seidman et al. (2001) for RQ1, and Kallitsoglou (2018) and Purvis and Tannock (1997) for RQ2.

Figure 1. Prisma 2020 flow diagram (Page et al., 2021).



Note. The total number of reports excluded and reasons for exclusion were recorded. However, counts per reason were not systematically tracked and are therefore not included in the diagram.

2.4.2. Study Characteristics

Table 1. Key characteristics of included studies addressing RQ1 (prevalence).

Author (Year)	Country	Sample Size	Age	Gender	Diagnosis
Aro et al. (2022)	Finland	579	Ages 8-15	368 Male 211 Female	Clinician-confirmed within study
Aaron et al. (2002)	USA	50	Aged around 7-11	30 Male 20 Female	Broad sample
Adams et al. (1999)	UK	364	Aged around 7-11	181 Male 183 Female	Clinician-confirmed within study
Aitken et al. (2020)	USA	501	Ages 6-9	247 Male 254 Female	Broad sample; later clinician-confirmed
Altay et al. (2018)	Turkey	80	Ages 6-15	49 Male 31 Female	Clinician-confirmed within study
Asberg et al. (2010)	Sweden	110	Ages 8-17	All female	Clinician-confirmed within study
Becker et al. (2021)	Germany	159	Aged around 10	108 Male 51 Female	Clinician-confirmed within study
Biederman et al. (2002)	USA	522	Ages 6-17	260 Male 262 Female	Clinician-confirmed within study
Bonafina et al. (2000)	USA	54	Ages 7-11	51 Male 3 Female	Clinician-confirmed within study
Brandenburg et al. (2021)	Germany	302	Aged around 8	168 Male 134 Female	Clinician-confirmed within study
Brimo et al. (2021)	Sweden	1,688	Ages 9+	49% Male	Clinician-confirmed within study
Capano et al. (2008)	Canada	476	Ages 7-12	389 Male 87 Female	Clinician-confirmed within study
Carlsson et al. (2022)	Sweden	32	Ages 65+	17 Male 15 Female	Broad sample
Carmine Pastura et al. (2009)	Brazil	396	Ages 9-14	Mostly male	Clinician-confirmed within study
Carroll et al. (2005)	UK	5,752	Ages 9-15	Majority male	Broad sample

Author (Year)	Country	Sample Size	Age	Gender	Diagnosis
Casey et al. (2000)	USA	107	Ages 6-14	82 Male 25 female	Clinician-confirmed within study
Cederlof et al. (2017)	Sweden	8,719	Ages 16-61	58.2% Female	Formal diagnosis prior to study
Condo et al. (2022)	USA	162	Ages 8-13	112 Male 50 Female	Formal diagnosis prior to study
Crawford et al. (2006)	Canada	102	Ages 8-16	84 Male 18 Female	Clinician-confirmed within study
Czamara et al. (2013)	Germany	4,861	Aged around 10	2,475 Male 2,386 Female	Broad sample
Darweesh et al. (2020)	Egypt	100	Ages 7-15	53 Male 47 Female	Clinician-confirmed within study
Del'Homme et al. (2007)	USA	507	Ages 6-17	369 Male 138 Female	Clinician-confirmed within study
Doyle et al. (2005)	USA	538	Aged around 28-34	243 Male 295 Female	Clinician-confirmed within study
Dykman et al. (1991)	USA	234	Aged around 10	186 Male 48 Female	Clinician-confirmed within study
Efron et al. (2014)	Australia	391	Ages 6-8	259 Male 132 Female	Clinician-confirmed within study
El Sheikh et al. (2016)	Egypt	567	Aged around 9-11	305 Male 262 Female	Broad sample
Espanol-Martin et al. (2023)	Spain	1,530	Ages 5-17	51.8% Male 48.2% Female	Broad sample
Faraone et al. (1993)	USA	260	Ages 6-17	All male	Clinician-confirmed within study
Fortes et al. (2016)	Brazil	1,618	Aged around 7-12	Unclear	Broad sample
Frances et al. (2023)	Spain	289	Aged around 6	155 Male 134 Female	Formal diagnosis prior to study
Goker et al. (2014)	Turkey	25,013	Aged around 8	65.4% Male	Formal diagnosis

Author (Year)	Country	Sample Size	Age	Gender	Diagnosis
Goldston et al. (2007)	USA	188	Aged around 15	55% Male	prior to study Broad sample
Halleland et al. (2019)	Norway	156	Aged 19-59	Around 43% male	Formal diagnosis prior to study
Hechtman et al. (2005)	Canada	579	Ages 7-9	Unclear	Clinician-confirmed within study
Heikkila et al. (2016)	Finland	205	Aged around 7-12	62% Male	Formal diagnosis prior to study
Helland et al. (2012)	Norway	5,672	Ages 7-9	2,841 Male	Broad sample
Herrera-Gutierrez et al. (2021)	Spain	111	Ages 6-17	81.1% Male	Formal diagnosis prior to study
Hinshaw (2002)	USA	228	Ages 6-12	All female	Clinician-confirmed within study
Ho et al. (2005)	China	106	Aged around 9	85 Male 21 Female	Clinician-confirmed within study
Huang et al. (2007)	Taiwan	27	Aged around 9	23 Male 4 Female	Formal diagnosis prior to study
Johnson et al. (2021)	Sweden	87	Ages 6-18	61 Male 26 Female	Formal diagnosis prior to study
Kadesjo et al. (2001)	Sweden	409	Aged around 7	224 Male 185 Female	Clinician-confirmed within study
Kaplan et al. (2001)	Canada	179	Ages 8-16	136 Male 43 Female	Clinician-confirmed within study
Kita et al. (2020)	Japan	3,852	Ages 6-9	Unclear	Clinician-confirmed within study
Klorman et al. (1999)	USA	359	Ages 7-13	Majority male	Clinician-confirmed within study

Author (Year)	Country	Sample Size	Age	Gender	Diagnosis
Koerner et al. (2021)	Germany	3,020	Aged around 9	1,575 Male 1,445 Female	Clinician-confirmed within study
Lamminmaki et al. (1995)	Chile	110	Aged around 9	37 Male 73 Female	Clinician-confirmed within study
Langberg et al. (2010)	USA	128	Ages 7-11	72% Male	Clinician-confirmed within study
Li et al. (2020)	USA	1,432	Ages 8-15	Unclear	Clinician-confirmed within study
Little et al. (2010)	Canada	353	Aged around 4-16	79.2% Male	Clinician-confirmed within study
Livingston et al. (1990)	USA	182	Ages 7-11	153 Male 29 Female	Clinician-confirmed within study
Loo et al. (2004)	USA	407	Ages 5-18	296 Male 111 Female	Clinician-confirmed within study
Luoni et al. (2015)	Italy	369	Ages 6-7	202 Male 167 Female	Broad sample
Marshall et al. (1997)	USA	44	Ages 6-12	35 Male 9 Female	Clinician-confirmed within study
Martinussen et al. (2015)	Canada	87	Ages 13-18	Not reported	Formal diagnosis prior to study
Mayes et al. (2006)	USA	949	Ages 6-16	73% Male	Formal diagnosis prior to study
Mayes et al. (2020)	USA	499	Ages 6-16	71.9% Male	Formal diagnosis prior to study
McGee et al. (2000)	Canada	100	Ages 6-11	79% Male	Clinician-confirmed within study
Miranda et al. (2017)	Spain	60	Ages 18-24	All male	Formal diagnosis prior to study
Molitor et al. (2016)	USA	326	Aged around 11-14	71% Male	Clinician-confirmed within study

Author (Year)	Country	Sample Size	Age	Gender	Diagnosis
Nakashima et al. (2012)	Japan	20	Ages 8-13	17 Male 3 Female	Formal diagnosis prior to study
Narhi et al. (1995)	Finland	73	Ages 8-12	All male	Clinician-confirmed within study
Naz et al. (2019)	Pakistan	48	Aged around 12	19 Male 29 Female	Clinician-confirmed within study
Nigg (1999)	USA	50	Aged around 6-12	31 Male 19 Female	Clinician-confirmed within study
Nigg et al. (1998)	USA	171	Ages 6-12	All male	Clinician-confirmed within study
Pagirsky et al. (2017)	USA	91	Ages 5-18	52 Male 39 Female	Clinician-confirmed within study
Papaioannou et al. (2016)	Greece	923	Ages 6-11	465 Male 458 Female	Clinician-confirmed within study
Pauc (2005)	UK	100	Ages 4-15	77% Male	Clinician-confirmed within study
Pennington et al. (1993)	USA	70	Ages 7-10	All male	Clinician-confirmed within study
Pereira et al. (2020)	Brazil	104	Ages 8-11	50 Male 54 Female	Clinician-confirmed within study
Pisecco et al. (2001)	New Zealand	1,039	Aged around 11	All male	Broad sample
Pritchard et al. (2016)	USA	96	Aged around 8-14	68% Male	Formal diagnosis prior to study
Punt et al. (2010)	Netherlands	104	Ages 8-13	Majority male	Clinician-confirmed within study
Russell et al. (2015)	UK	12,631	Ages 7-11	Majority male	Clinician-confirmed within study
Samuelsson et al. (2004)	Norway	120	Aged around 29-37	All male	Clinician-confirmed within study

Author (Year)	Country	Sample Size	Age	Gender	Diagnosis
Sanchez-Moran et al. (2018)	Spain	3,357	Ages 6-16	1,693 Male 1,664 Female	Clinician-confirmed within study
Schachar et al. (2004)	Canada	151	Ages 7-16	76.2% Male	Formal diagnosis prior to study
Semrud-Clikeman et al. (1992)	USA	126	Aged around 9-11	Unclear	Clinician-confirmed within study
Sesma et al. (2009)	USA	60	Ages 9-15	50% Male	Clinician-confirmed within study
Shanahan et al. (2006)	USA	395	Ages 8-18	203 Male 192 Female	Clinician-confirmed within study
Sheikhi et al. (2013)	Australia	7,209	Ages 4-18	3,681 Male 3,528 Female	Clinician-confirmed within study
Stack-Cutler et al. (2016)	Canada	120	Aged around 25	71% Female	Clinician-confirmed within study
Stark et al. (2022)	South Africa	847	Ages 8-18	65.9% male	Clinician-confirmed within study
Stevenson et al. (2005)	UK	152	Ages 6-13	140 Male 12 Female	Formal diagnosis prior to study
Swanson et al. (1999)	USA	90	Aged around 6-11	55 Male	Clinician-confirmed within study
Thomson et al. (2005)	USA	209	Ages 6-17	115 Male 94 Female	Clinician-confirmed within study
Toffalini et al. (2022)	Italy	948	Ages 6-16	81% Male	Formal diagnosis prior to study
Toplak et al. (2003)	Canada	198	Ages 6-16	Majority male	Formal diagnosis prior to study
Tosto et al. (2021)	Ireland	117	Ages 8-12	80% Male	Formal diagnosis prior to study

Author (Year)	Country	Sample Size	Age	Gender	Diagnosis
Vakil et al. (2012)	Israel	112	Ages 11-17	Majority male	Clinician-confirmed within study
Visser et al. (2020)	Germany	3,014	Ages 8-11	52.1% Male	Clinician-confirmed within study
Voigt et al. (2017)	USA	567	Aged around 27-28	377 Male 190 Female	Formal diagnosis prior to study
Wadsworth et al. (2007)	USA	278	Ages 8-23	Unclear	Clinician-confirmed within study
Wassenberg et al. (2010)	Netherlands	60	Ages 7-16	Majority male	Formal diagnosis prior to study
Westervelt et al. (1998)	USA	42	Ages 9-14	64% Male	Formal diagnosis prior to study
Willcutt et al. (2000)	USA	867	Unclear; child study	455 Male 412 Female	Clinician-confirmed within study
Wisniewska et al. (2007)	Poland	28	Ages 7-13	23 Male 5 Female	Formal diagnosis prior to study
Yoshimasu et al. (2010)	USA	5,718	Aged around 5	2,956 Male 2,762 Female	Clinician-confirmed within study

Note. A total of 98 studies were included to address RQ1 (90 child studies, 8 adult studies). Studies addressing both research questions are presented in both Table 1 and Table 2. Note that the total sample size in this table reflects the full sample in each study, including typically developing controls (if available), and those with ADHD and/or RD. Therefore, the age and gender are reflective of all participants included in a given study, not just those with ADHD and/or RD. The ‘Diagnosis’ column reflects whether ADHD and RD were formally diagnosed prior to the study, confirmed within the study based on pre-ascertained criteria, or whether the study included a broader sample with ADHD/RD-related difficulties without a formal diagnostic label.

Table 2. Key characteristics of included studies addressing RQ2 (cognitive performance).

Author (Year)	Country	Sample Size	Age/Grade	Gender	Diagnosis	Cognitive Domain Assessed
Adams et al. (2001)	UK	21	Ages 8-11	17 Male 4 Female	Clinician-confirmed within study	Working Memory Attention
Al Dahhan et al. (2022)	USA	88	Aged around 8-12	56 Male 32 Female	Clinician-confirmed within study	Memory Attention Processing Speed
Areces et al. (2018)	Spain	101	Ages 5-16	64 Male 37 Female	Formal diagnosis prior to study	Processing Speed
Becker et al. (2021)	Germany	159	Aged around 10	108 Male 51 Female	Clinician-confirmed within study	Working Memory Processing Speed Vocabulary
Bental et al. (2007)	Israel	86	Aged around 7-11	All male	Clinician-confirmed within study	Working Memory Attention Inhibition Cognitive Flexibility Planning Processing Speed Vocabulary
Caldani et al. (2022)	France	96	Aged around 10	Unclear	Clinician-confirmed within study	Working Memory Processing Speed Vocabulary
Capano et al. (2008)	Canada	476	Aged around 7-12	389 Male 87 Female	Clinician-confirmed within study	Vocabulary
Chadwick et al. (1999)	UK	3,176	Ages 6-7	All male	Clinician-confirmed within study	Vocabulary
Chan et al. (2008)	China	92	Aged around 8	78 Male 14 Female	Formal diagnosis prior to study	Working Memory Processing Speed Vocabulary
Crippa et al. (2015)	Italy	172	Aged around 8-12	Majority male	Formal diagnosis prior to study	Memory Attention Inhibition

Author (Year)	Country	Sample Size	Age/Grade	Gender	Diagnosis	Cognitive Domain Assessed
De Groot et al. (2017)	Netherlands	1,262	Ages 8-13	619 Male 643 Female	Clinician-confirmed within study	Cognitive Flexibility Planning Problem-Solving Processing Speed Processing Speed
De Jong et al. (2012)	Netherlands	94	Aged around 10-11	54 Male 40 Female	Formal diagnosis prior to study	Processing Speed
De Jong et al. (2009)	Netherlands	120	Ages 8-12	82 Male 38 Female	Clinician-confirmed within study	Working Memory Inhibition Vocabulary
Dewey et al. (2001)	Canada	291	Aged around 11-12	218 Male 73 Female	Formal diagnosis prior to study	Working Memory Memory
Fernandez-Andres et al. (2021)	Ecuador	140	Aged around 9-10	51.43% Male	Clinician-confirmed within study	Working Memory Attention Inhibition Cognitive Flexibility Planning Problem-Solving Processing Speed Vocabulary
Ghelani et al. (2004)	Canada	96	Ages 14-17	66 Male 30 Female	Clinician-confirmed within study	Working Memory Attention Inhibition Vocabulary Attention Inhibition Vocabulary
Gooch et al. (2011)	UK	101	Ages 5-14	62 Male 39 Female	Formal diagnosis prior to study	Working Memory Attention Inhibition Vocabulary
Hall et al. (1997)	USA	70	Ages 6-13	47 Male 23 Female	Clinician-confirmed within study	Attention Inhibition Vocabulary
Ho et al. (2005)	China	106	Aged around 9	85 Male 21 Female	Clinician-confirmed within study	Working Memory Processing Speed
Horowitz-Kraus et al. (2016)	Israel	28	Aged around 12-13	26 Male 2 Female	Formal diagnosis prior to study	Working Memory Cognitive Flexibility Planning

Author (Year)	Country	Sample Size	Age/Grade	Gender	Diagnosis	Cognitive Domain Assessed
Horowitz-Kraus et al. (2019)	USA	54	Aged around 9	50% Male	Clinician-confirmed within study	Processing Speed Attention Inhibition Cognitive Flexibility
Jakobson et al. (2007)	Estonia	152	Ages 7-10	Majority male	Clinician-confirmed within study	Processing Speed Working Memory Vocabulary
Johnson et al. (1999)	USA	80	Ages 7-13	Majority male	Clinician-confirmed within study	Working Memory Vocabulary
Kaplan et al. (2000)	Canada	200	Aged around 12	154 Male 46 Female	Clinician-confirmed within study	Memory Problem-Solving Vocabulary
Kibby et al. (2008)	USA	113	Ages 6-15	Majority male	Clinician-confirmed within study	Working Memory Memory Processing Speed Vocabulary
Kibby et al. (2021)	USA	263	Ages 8-12	Majority male	Clinician-confirmed within study	Working Memory Inhibition Cognitive Flexibility Planning Problem-Solving
Langer et al. (2019)	USA	60	Aged around 9-10	35 Male 25 Female	Formal diagnosis prior to study	Working Memory Inhibition Cognitive Flexibility Processing Speed Vocabulary
Lee et al. (2023)	China	104	Aged around 9	Unclear	Clinician-confirmed within study	Working Memory Memory Inhibition Cognitive Flexibility
Li et al. (2022)	China	151	Grades 1-3	Unclear	Clinician-confirmed within study	Vocabulary
Maehler et al. (2016)	Germany	122	Aged around 9-10	93 Male 29 Female	Clinician-confirmed within study	Working Memory

Author (Year)	Country	Sample Size	Age/Grade	Gender	Diagnosis	Cognitive Domain Assessed
Mano et al. (2017)	USA	187	Grades 2-5	115 Male 72 Female	Clinician-confirmed within study	Processing Speed Vocabulary
Martinussen et al. (2006)	Canada	143	Aged around 7-13	Majority male	Clinician-confirmed within study	Working Memory Vocabulary
McGee et al. (2000)	Canada	100	Ages 6-11	79% Male	Clinician-confirmed within study	Attention Processing Speed
Mohl et al. (2018)	USA	40	Ages 9-16	All male	Clinician-confirmed within study	Working Memory Cognitive Flexibility Vocabulary
Moura et al. (2017)	Portugal	116	Ages 8-10	69% Male	Clinician-confirmed within study	Working Memory Memory Cognitive Flexibility Processing Speed Vocabulary
Narhi et al. (1995)	Finland	73	Ages 8-12	All male	Clinician-confirmed within study	Cognitive Flexibility Processing Speed Vocabulary
Nigg et al. (1998)	USA	171	Ages 6-12	All male	Clinician-confirmed within study	Cognitive Flexibility Planning Processing Speed Vocabulary
Pagirsky et al. (2017)	USA	91	Ages 5-18	52 Male 39 Female	Clinician-confirmed within study	Processing Speed Vocabulary
Parke et al. (2020)	USA	296	Ages 6-16	69% Male	Formal diagnosis prior to study	Working Memory Processing Speed Vocabulary
Pennington et al. (1993)	USA	70	Ages 7-10	All male	Clinician-confirmed within study	Attention Inhibition Cognitive Flexibility Planning Vocabulary
Pereira et al. (2020)	Brazil	104	Ages 8-11	50 Male 54 Female	Clinician-confirmed	Working Memory Cognitive Flexibility

Author (Year)	Country	Sample Size	Age/Grade	Gender	Diagnosis	Cognitive Domain Assessed
Pisecco et al. (2001)	New Zealand	1,039	Aged around 11	All male	within study Broad sample	Processing Speed Vocabulary Vocabulary
Poon et al. (2014)	China	117	Ages 12-18	All male	Clinician-confirmed within study	Memory Inhibition Planning Problem-Solving Processing Speed Working Memory
Poon et al. (2021)	China	202	Ages 6-11	122 Male 80 Female	Formal diagnosis prior to study	
Purvis et al. (2000)	Canada	68	Ages 7-11	Majority male	Clinician-confirmed within study	Inhibition Problem-Solving Processing Speed Vocabulary Working Memory
Rucklidge et al. (2002)	New Zealand	108	Ages 13-16	59 Male 49 Female	Clinician-confirmed within study	Inhibition Problem-Solving Processing Speed Vocabulary
Savitz et al. (2003)	South Africa	36	Aged around 8-12	All male	Formal diagnosis prior to study	Inhibition Vocabulary
Seidman et al. (2001)	USA	275	Ages 6-17	All male	Formal diagnosis prior to study	Working Memory Memory Attention Inhibition Cognitive Flexibility Planning Processing Speed
Shanahan et al. (2006)	USA	395	Ages 8-18	203 Male 192 Female	Clinician-confirmed within study	Inhibition Cognitive Flexibility Processing Speed Vocabulary
Shaywitz et al. (1995)	USA	186	Aged around 7-9	133 Male 53 Female	Clinician-confirmed within study	Working Memory Attention Processing Speed Vocabulary
Shin et al. (2003)	Korea	57	Ages 6-13	53 Male 4 Female	Clinician-confirmed within study	Memory Vocabulary

Author (Year)	Country	Sample Size	Age/Grade	Gender	Diagnosis	Cognitive Domain Assessed
Stubenrauch et al. (2014)	Germany	152	Ages 8-12	110 Male 42 Female	Clinician-confirmed within study	Inhibition
Swanson et al. (1999)	USA	90	Grades 1-5	55 Male 35 Female	Clinician-confirmed within study	Working Memory Vocabulary
Tannock et al. (2000)	Canada	67	Ages 7-12	80% Male	Clinician-confirmed within study	Problem-Solving Processing Speed Vocabulary
Tiffin-Richards et al. (2008)	Germany	79	Ages 10-14	Majority male	Clinician-confirmed within study	Working Memory Cognitive Flexibility Vocabulary
Toplak et al. (2003)	Canada	198	Aged around 6-16	Majority male	Formal diagnosis prior to study	Working Memory Problem-Solving Processing Speed Vocabulary
Turker et al. (2019)	Germany	166	Ages 8-18	103 Male 63 Female	Formal diagnosis prior to study	Working Memory Vocabulary
Van de Voorde et al. (2010)	Belgium	76	Ages 8-12		Clinician-confirmed within study	Working Memory Inhibition Processing Speed
Wang et al. (2018)	Taiwan	121	Grades 3-5	71 Male 50 Female	Clinician-confirmed within study	Working Memory Attention Inhibition Processing Speed
Willcutt et al. (2001)	USA	314	Ages 8-16	Unclear	Clinician-confirmed within study	Working Memory Inhibition Cognitive Flexibility Vocabulary
Willcutt et al. (2005)	USA	437	Ages 8-18	Unclear	Clinician-confirmed within study	Processing Speed
Wu et al. (2002)	Australia	112	Ages 7-13	92 Male 20 Female	Formal diagnosis prior to study	Working Memory Attention Cognitive Flexibility Planning Processing Speed

Author (Year)	Country	Sample Size	Age/Grade	Gender	Diagnosis	Cognitive Domain Assessed
Zhang et al. (2022)	China	88	Ages 6-12	69 Male 19 Female	Clinician-confirmed within study	Working Memory Attention Planning Problem-Solving Processing Speed Vocabulary
Gregg et al. (2008)	USA	411	Aged around 20	203 Male 208 Female	Clinician-confirmed within study	Processing Speed Vocabulary
Katz et al. (2011)	USA	109	Ages 16-55	62 Male 47 Female	Clinician-confirmed within study	Working Memory Attention Inhibition Processing Speed
Laasonen et al. (2012)	Finland	101	Ages 18-55	50 Male 51 Female	Formal diagnosis prior to study	Attention Inhibition Cognitive Flexibility Planning
Laasonen et al. (2009)	Finland	119	Ages 18-55	60 Male 59 Female	Formal diagnosis prior to study	Working Memory Problem-Solving Processing Speed Vocabulary
Miranda et al. (2017)	Spain	60	Ages 18-24	All male	Formal diagnosis prior to study	Working Memory Inhibition Cognitive Flexibility Planning Problem-Solving
Whipple et al. (2016)	USA	203	Aged around 21	52% Male	Clinician-confirmed within study	Processing Speed

Note. 69 studies were included to address RQ2 (63 child studies, 6 adult studies). Studies addressing both research questions are presented in both Table 1 and 2. Child studies are presented in alphabetical order first, followed by adult studies. Note that the sample size in this table reflects the full sample in each study, including typically developing controls (if available), and those with ADHD and/or RD. Therefore, the age/grade and gender are reflective of all participants included in a given study, not just those with ADHD and/or RD. The ‘Diagnosis’ column reflects whether ADHD and RD were formally diagnosed prior to the study, confirmed within the study based on pre-ascertained criteria, or whether the study included a broader sample with ADHD/RD-related difficulties without a formal diagnostic label.

2.4.3. Risk of Bias

The majority of studies addressing RQ1 demonstrated high risk of bias in sampling procedures (i.e., sample frames, sampling methods, and power calculations). Some studies also showed high risk of bias in methods used to identify ADHD and RD (i.e., validity and reliability of diagnostic assessments). The majority of studies displayed some concerns in statistical analysis used (i.e., appropriateness for estimating prevalence rates), as well as in methods used to manage low participation rates. Low risk of bias was observed for most studies in providing detailed descriptions of participants and study settings. Further, studies addressing RQ2 generally demonstrated low risk of bias across all domains. The main source of concern was that inclusion and exclusion criteria were not always clearly defined, and that it was unclear whether valid assessments were used for the identification of the conditions. Some studies also displayed concerns in the identification and control of confounding factors. Generally, studies with earlier publication dates tended to demonstrate higher risk of bias or uncertainty across both RQ1 and RQ2. A full summary of the risk of bias assessment is presented in Appendices G-J (Supplementary Materials 1), with justifications for each unclear or high risk of bias judgment, and a *robvis* traffic light plot (McGuinness & Higgins, 2020) to demonstrate risk of bias judgments across all domains for each study.

2.4.4. Results of Individual Studies

Table 3. Frequency of participants with ADHD+RD in each study addressing RQ1.

Study	Age Group	Sampling Method	RD Definition	Sample Size	ADHD+RD Frequency
Aaron et al. (2002)	Child	Purposive	Unclear/Other	39	6
Adams et al. (1999)	Child	Purposive	Discrepancy	32	5
Aitken et al. (2020)	Child	Mixed	Low Achievement	501	42
Altay et al. (2018)	Child	Mixed	Low Achievement	80	19
Aro et al. (2022)	Child	Purposive	Unclear/Other	197	81
Asberg et al. (2010)	Child	Purposive	Low Achievement	36	20
Becker et al. (2021)	Child	Purposive	Low Achievement	62	19

Study	Age Group	Sampling Method	RD Definition	Sample Size	ADHD+RD Frequency
Biederman et al. (2002)	Child	Purposive	Discrepancy	280	31
Bonafina et al. (2000)	Child	Purposive	Discrepancy	54	24
Brandenburg et al. (2021)	Child	Mixed	Discrepancy and Low Achievement	302	24
Brimo et al. (2021)	Child	Purposive	Unclear/Other	128	27
Capano et al. (2008)	Child	Purposive	Low Achievement	476	77
Carmine Pastura et al. (2009)	Child	Purposive	Unclear/Other	26	3
Carroll et al. (2005)	Child	Purposive	Discrepancy	289	25
Casey et al. (2000)	Child	Purposive	Discrepancy	107	41
Condo et al. (2022)	Child	Purposive	Unclear/Other	104	15
Crawford et al. (2006)	Child	Purposive	Low Achievement	102	27
Czamara et al. (2013)	Child	Purposive	Unclear/Other	394	99
Darweesh et al. (2020)	Child	Purposive	Unclear/Other	50	9
Del'Homme et al. (2007)	Child	Purposive	Discrepancy and Low Achievement	507	109
Dykman et al. (1991)	Child	Purposive	Discrepancy	182	94
Efron et al. (2014)	Child	Purposive	Low Achievement	179	59
El Sheikh et al. (2016)	Child	Purposive	Unclear/Other	64	9
Espanol-Martin et al. (2023)	Child	Purposive	Unclear/Other	282	52
Faraone et al. (1993)	Child	Purposive	Discrepancy	140	25
Fortes et al. (2016)	Child	Purposive	Discrepancy and Low Achievement	121	8
Frances et al. (2023)	Child	Mixed	Low Achievement	289	13
Goker et al. (2014)	Child	Purposive	Unclear/Other	103	85
Goldston et al. (2007)	Child	Purposive	Low Achievement	94	17

Study	Age Group	Sampling Method	RD Definition	Sample Size	ADHD+RD Frequency
Hechtman et al. (2005)	Child	Purposive	Discrepancy	579	92
Heikkila et al. (2016)	Child	Mixed	Low Achievement	205	30
Helland et al. (2012)	Child	Mixed	Unclear/Other	5672	121
Herrera-Gutierrez et al. (2021)	Child	Purposive	Unclear/Other	111	22
Hinshaw (2002)	Child	Purposive	Low Achievement	140	17
Ho et al. (2005)	Child	Purposive	Low Achievement	54	16
Huang et al. (2007)	Child	Purposive	Unclear/Other	17	14
Johnson et al. (2021)	Child	Purposive	Unclear/Other	87	14
Kadesjo et al. (2001)	Child	Purposive	Low Achievement	57	18
Kaplan et al. (2001)	Child	Purposive	Low Achievement	116	63
Kita et al. (2020)	Child	Mixed	Unclear/Other	2205	16
Klorman et al. (1999)	Child	Purposive	Discrepancy	207	47
Koerner et al. (2021)	Child	Purposive	Low Achievement	415	67
Lamminmäki et al. (1995)	Child	Purposive	Low Achievement	45	11
Langberg et al. (2010)	Child	Purposive	Low Achievement	128	14
Li et al. (2020)	Child	Purposive	Low Achievement	329	142
Little et al. (2010)	Child	Purposive	Unclear/Other	272	86
Livingston et al. (1990)	Child	Purposive	Discrepancy	174	71
Loo et al. (2004)	Child	Purposive	Discrepancy and Low Achievement	407	83
Luoni et al. (2015)	Child	Purposive	Low Achievement	44	13
Marshall et al. (1997)	Child	Purposive	Unclear/Other	44	11
Martinussen et al. (2015)	Child	Purposive	Low Achievement	42	12
Mayes et al. (2006)	Child	Purposive	Discrepancy	617	103

Study	Age Group	Sampling Method	RD Definition	Sample Size	ADHD+RD Frequency
Mayes et al. (2020)	Child	Purposive	Discrepancy	499	123
McGee et al. (2000)	Child	Purposive	Discrepancy	68	14
Molitor et al. (2016)	Child	Purposive	Discrepancy and Low Achievement	326	67
Nakashima et al. (2012)	Child	Purposive	Low Achievement	20	7
Narhi et al. (1995)	Child	Purposive	Low Achievement	63	25
Naz et al. (2019)	Child	Purposive	Low Achievement	24	12
Nigg (1999)	Child	Purposive	Discrepancy	25	3
Nigg et al. (1998)	Child	Purposive	Discrepancy and Low Achievement	42	6
Pagirsky et al. (2017)	Child	Purposive	Low Achievement	91	45
Papaioannou et al. (2016)	Child	Purposive	Low Achievement	88	20
Pauc (2005)	Child	Purposive	Unclear/Other	73	29
Pennington et al. (1993)	Child	Purposive	Discrepancy	47	16
Pereira et al. (2020)	Child	Purposive	Low Achievement	71	15
Pisecco et al. (2001)	Child	Purposive	Low Achievement	36	16
Pritchard et al. (2016)	Child	Purposive	Low Achievement	96	50
Punt et al. (2010)	Child	Purposive	Unclear/Other	104	9
Russell et al. (2015)	Child	Purposive	Discrepancy and Low Achievement	628	87
Sanchez-Moran et al. (2018)	Child	Purposive	Unclear/Other	563	45
Schachar et al. (2004)	Child	Purposive	Low Achievement	151	36
Semrud-Clikeman et al. (1992)	Child	Purposive	Discrepancy and Low Achievement	60	15
Sesma et al. (2009)	Child	Purposive	Low Achievement	29	7
Shanahan et al. (2006)	Child	Purposive	Low Achievement	251	51
Sheikhi et al. (2013)	Child	Mixed	Unclear/Other	6535	974

Study	Age Group	Sampling Method	RD Definition	Sample Size	ADHD+RD Frequency
Stark et al. (2022)	Child	Purposive	Unclear/Other	801	327
Stevenson et al. (2005)	Child	Purposive	Discrepancy	152	82
Swanson et al. (1999)	Child	Purposive	Discrepancy	90	37
Thomson et al. (2005)	Child	Purposive	Discrepancy	209	22
Toffalini et al. (2022)	Child	Purposive	Low Achievement	755	110
Toplak et al. (2003)	Child	Purposive	Low Achievement	109	43
Tosto et al. (2021)	Child	Purposive	Unclear/Other	117	78
Vakil et al. (2012)	Child	Purposive	Low Achievement	64	14
Visser et al. (2020)	Child	Purposive	Low Achievement	80	14
Wadsworth et al. (2007)	Child	Purposive	Unclear/Other	124	27
Wassenberg et al. (2010)	Child	Purposive	Unclear/Other	30	10
Westervelt et al. (1998)	Child	Purposive	Unclear/Other	42	16
Willcutt et al. (2000)	Child	Purposive	Low Achievement	494	96
Wisniewska et al. (2007)	Child	Purposive	Unclear/Other	28	3
Yoshimasu et al. (2010)	Child	Purposive	Discrepancy and Low Achievement	379	165
Carlsson et al. (2022)	Adult	Purposive	Unclear/Other	32	13
Cederlof et al. (2017)	Adult	Purposive	Unclear/Other	8699	334
Doyle et al. (2005)	Adult	Purposive	Unclear/Other	106	9
Halleland et al. (2019)	Adult	Purposive	Unclear/Other	74	31
Miranda et al. (2017)	Adult	Purposive	Low Achievement	30	13
Samuelsson et al. (2004)	Adult	Mixed	Discrepancy	120	12
Stack-Cutler et al. (2016)	Adult	Purposive	Unclear/Other	120	42
Voigt et al. (2017)	Adult	Purposive	Discrepancy	232	111

Note. This table contains information about the age group, sampling method, and RD definition employed in each study. This information was used for subgroup analyses. Note that the studies are presented in alphabetical order for child studies first, followed by adult studies. Also note that the sample sizes in this table may not correspond to the total sample sizes listed in Tables 1-2. This is because the figures in this table reflect the number of participants from which the proportion of ADHD+RD was calculated. This differed across studies depending on information available. For instance, some studies included a general population sample and reported the proportion of participants with ADHD+RD from this sample (in which case the reported sample sizes across Tables 1-3 would not differ). Other studies may have reported the number of participants with ADHD out of the total sample, then the proportion of those who also met criteria for RD (and vice versa). In this case, the sample size in this table would represent the number of participants with ADHD, and the ‘ADHD+RD Frequency’ section would reflect the proportion of those participants who also met criteria for RD.

Table 4. Effect sizes with standard errors for each cognitive domain assessed across all studies addressing RQ2.

Study	Age Group	Cognitive Domain	Effect Size	Standard Error
Adams et al. (2001)	Child	Working Memory	-0.16	0.38
Adams et al. (2001)	Child	Attention	-0.50	0.38
Al Dahhan et al. (2022)	Child	Memory	-1.22	0.27
Al Dahhan et al. (2022)	Child	Attention	-0.75	0.26
Al Dahhan et al. (2022)	Child	Processing Speed	-1.27	0.28
Areces et al. (2018)	Child	Processing Speed	-1.37	0.31
Becker et al. (2021)	Child	Working Memory	-0.51	0.22
Becker et al. (2021)	Child	Processing Speed	-0.76	0.22
Becker et al. (2021)	Child	Vocabulary	-0.01	0.21
Bental et al. (2007)	Child	Working Memory	-0.80	0.30
Bental et al. (2007)	Child	Attention	-1.80	0.34
Bental et al. (2007)	Child	Inhibition	-0.30	0.29
Bental et al. (2007)	Child	Cognitive Flexibility	-0.36	0.29
Bental et al. (2007)	Child	Planning	-0.44	0.29
Bental et al. (2007)	Child	Processing Speed	-1.55	0.32
Bental et al. (2007)	Child	Vocabulary	-0.71	0.29
Caldani et al. (2022)	Child	Working Memory	-1.44	0.32
Caldani et al. (2022)	Child	Processing Speed	-0.73	0.30
Caldani et al. (2022)	Child	Vocabulary	0.00	0.30
Capano et al. (2008)	Child	Vocabulary	-0.42	0.12
Chadwick et al. (1999)	Child	Vocabulary	-0.26	0.31
Chan et al. (2008)	Child	Working Memory	-0.24	0.16
Chan et al. (2008)	Child	Processing Speed	-1.61	0.16
Chan et al. (2008)	Child	Vocabulary	-1.82	0.16
Crippa et al. (2015)	Child	Memory	-1.08	0.31
Crippa et al. (2015)	Child	Attention	-1.97	0.34
Crippa et al. (2015)	Child	Inhibition	-0.87	0.31
Crippa et al. (2015)	Child	Cognitive Flexibility	-1.10	0.32
Crippa et al. (2015)	Child	Planning	-1.16	0.32
Crippa et al. (2015)	Child	Problem-Solving	-1.28	0.32
Crippa et al. (2015)	Child	Processing Speed	-1.15	0.32
De Groot et al. (2017)	Child	Processing Speed	-1.61	0.25
De Jong et al. (2012)	Child	Processing Speed	-1.24	0.32

Study	Age Group	Cognitive Domain	Effect Size	Standard Error
De Jong et al. (2009)	Child	Working Memory	-2.12	0.34
De Jong et al. (2009)	Child	Inhibition	-4.73	0.53
De Jong et al. (2009)	Child	Vocabulary	-1.32	0.30
Dewey et al. (2001)	Child	Working Memory	-0.72	0.16
Dewey et al. (2001)	Child	Memory	-0.42	0.16
Fernandez-Andres et al. (2021)	Child	Working Memory	-1.30	0.26
Fernandez-Andres et al. (2021)	Child	Attention	-1.54	0.27
Fernandez-Andres et al. (2021)	Child	Inhibition	-1.13	0.26
Fernandez-Andres et al. (2021)	Child	Cognitive Flexibility	-0.96	0.25
Fernandez-Andres et al. (2021)	Child	Planning	-1.14	0.26
Fernandez-Andres et al. (2021)	Child	Problem-Solving	-1.27	0.26
Ghelani et al. (2004)	Child	Processing Speed	-1.81	0.36
Ghelani et al. (2004)	Child	Vocabulary	-1.21	0.33
Gooch et al. (2011)	Child	Working Memory	-0.55	0.27
Gooch et al. (2011)	Child	Attention	-0.71	0.27
Gooch et al. (2011)	Child	Inhibition	-0.18	0.27
Gooch et al. (2011)	Child	Vocabulary	-0.87	0.26
Hall et al. (1997)	Child	Attention	-0.59	0.36
Hall et al. (1997)	Child	Inhibition	-1.31	0.39
Hall et al. (1997)	Child	Vocabulary	-0.85	0.37
Ho et al. (2005)	Child	Working Memory	-0.79	0.25
Ho et al. (2005)	Child	Processing Speed	-1.93	0.26
Horowitz-Kraus (2016)	Child	Working Memory	-1.88	0.28
Horowitz-Kraus (2016)	Child	Cognitive Flexibility	-4.57	0.31
Horowitz-Kraus (2016)	Child	Planning	1.19	0.27
Horowitz-Kraus (2016)	Child	Processing Speed	-1.99	0.28
Horowitz-Kraus et al. (2019)	Child	Attention	-0.66	0.34
Horowitz-Kraus et al. (2019)	Child	Inhibition	-0.69	0.34
Horowitz-Kraus et al. (2019)	Child	Cognitive Flexibility	-0.21	0.33
Horowitz-Kraus et al. (2019)	Child	Processing Speed	-1.12	0.36
Jakobson et al. (2007)	Child	Working Memory	-0.84	0.23
Jakobson et al. (2007)	Child	Vocabulary	-0.66	0.23
Johnson et al. (1999)	Child	Working Memory	-1.71	0.18
Johnson et al. (1999)	Child	Vocabulary	0.17	0.17
Kaplan et al. (2000)	Child	Memory	-0.32	0.13
Kaplan et al. (2000)	Child	Problem-Solving	0.06	0.13
Kaplan et al. (2000)	Child	Vocabulary	-0.34	0.13
Kibby et al. (2008)	Child	Working Memory	-1.12	0.28
Kibby et al. (2008)	Child	Memory	-0.42	0.26
Kibby et al. (2008)	Child	Processing Speed	-0.87	0.27
Kibby et al. (2008)	Child	Vocabulary	-0.23	0.26
Kibby et al. (2021)	Child	Working Memory	-9.05	0.61
Kibby et al. (2021)	Child	Inhibition	-8.77	0.59
Kibby et al. (2021)	Child	Cognitive Flexibility	-6.30	0.45
Kibby et al. (2021)	Child	Planning	-5.56	0.40
Kibby et al. (2021)	Child	Problem-Solving	-10.52	0.70
Langer et al. (2019)	Child	Working Memory	-2.27	0.47
Langer et al. (2019)	Child	Inhibition	-1.23	0.40

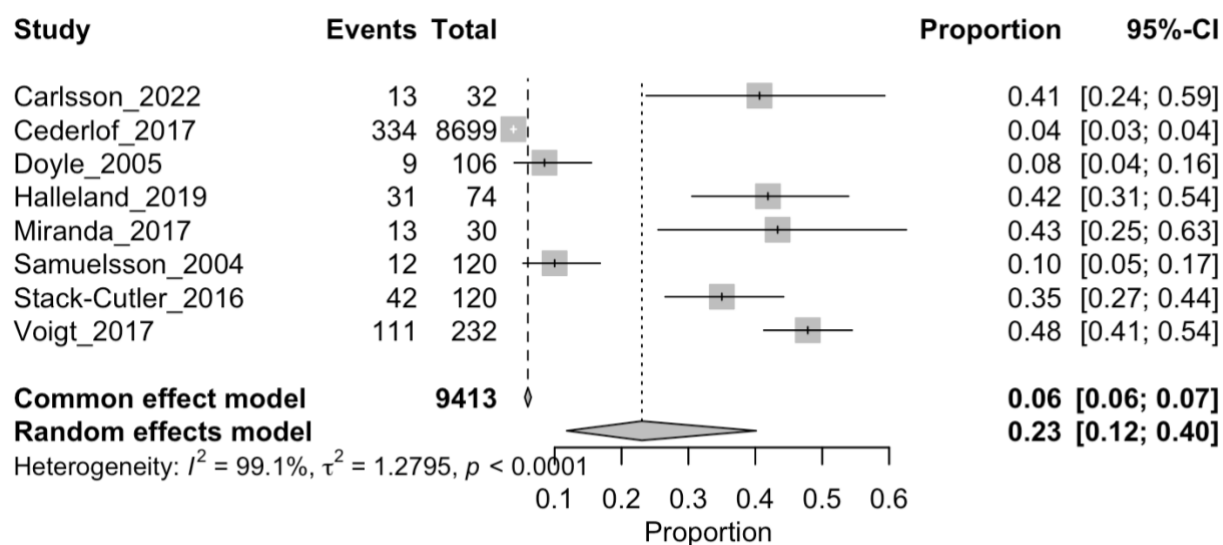
Study	Age Group	Cognitive Domain	Effect Size	Standard Error
Langer et al. (2019)	Child	Cognitive Flexibility	-1.01	0.39
Langer et al. (2019)	Child	Processing Speed	-1.28	0.41
Langer et al. (2019)	Child	Vocabulary	-1.96	0.45
Lee (2023)	Child	Working Memory	-2.23	0.37
Lee (2023)	Child	Memory	-1.76	0.34
Lee (2023)	Child	Inhibition	-2.03	0.36
Lee (2023)	Child	Cognitive Flexibility	-1.68	0.33
Li et al. (2022)	Child	Vocabulary	-2.31	0.31
Maehler et al. (2016)	Child	Working Memory	-0.55	0.25
Mano et al. (2017)	Child	Processing Speed	-0.82	0.08
Mano et al. (2017)	Child	Vocabulary	-1.34	0.09
Martinussen et al. (2006)	Child	Working Memory	-1.34	0.31
Martinussen et al. (2006)	Child	Vocabulary	-1.98	0.30
McGee et al. (2000)	Child	Attention	-0.81	0.33
McGee et al. (2000)	Child	Processing Speed	-0.56	0.33
Mohl et al. (2018)	Child	Working Memory	-0.39	0.40
Mohl et al. (2018)	Child	Cognitive Flexibility	-0.07	0.40
Mohl et al. (2018)	Child	Vocabulary	-0.69	0.41
Moura et al. (2017)	Child	Working Memory	-1.35	0.32
Moura et al. (2017)	Child	Memory	-0.32	0.29
Moura et al. (2017)	Child	Cognitive Flexibility	-1.31	0.32
Moura et al. (2017)	Child	Processing Speed	-1.95	0.35
Moura et al. (2017)	Child	Vocabulary	-0.96	0.31
Narhi et al. (1995)	Child	Cognitive Flexibility	-0.27	0.37
Narhi et al. (1995)	Child	Processing Speed	-1.11	0.40
Narhi et al. (1995)	Child	Vocabulary	-0.53	0.38
Nigg et al. (1998)	Child	Cognitive Flexibility	-1.69	0.31
Nigg et al. (1998)	Child	Planning	-1.19	0.30
Nigg et al. (1998)	Child	Processing Speed	-1.26	0.30
Nigg et al. (1998)	Child	Vocabulary	-1.31	0.30
Pagirsky et al. (2017)	Child	Processing Speed	-0.92	0.21
Pagirsky et al. (2017)	Child	Vocabulary	-1.11	0.21
Parke et al. (2020)	Child	Working Memory	-0.30	0.13
Parke et al. (2020)	Child	Processing Speed	-0.44	0.13
Parke et al. (2020)	Child	Vocabulary	0.07	0.13
Pennington et al. (1993)	Child	Attention	-0.14	0.33
Pennington et al. (1993)	Child	Inhibition	-0.19	0.33
Pennington et al. (1993)	Child	Cognitive Flexibility	-0.07	0.33
Pennington et al. (1993)	Child	Planning	0.05	0.32
Pennington et al. (1993)	Child	Vocabulary	-0.59	0.33
Pereira et al. (2020)	Child	Working Memory	-0.98	0.33
Pereira et al. (2020)	Child	Cognitive Flexibility	-1.18	0.33
Pereira et al. (2020)	Child	Processing Speed	-1.53	0.35
Pereira et al. (2020)	Child	Vocabulary	-0.71	0.32
Pisecco et al. (2001)	Child	Vocabulary	-0.30	0.26
Poon et al. (2014)	Child	Memory	-1.03	0.28
Poon et al. (2014)	Child	Inhibition	-0.39	0.27
Poon et al. (2014)	Child	Planning	-0.58	0.27

Study	Age Group	Cognitive Domain	Effect Size	Standard Error
Poon et al. (2014)	Child	Problem-Solving	-0.17	0.26
Poon et al. (2014)	Child	Processing Speed	-0.77	0.27
Poon et al. (2021)	Child	Working Memory	-0.90	0.23
Purvis et al. (2000)	Child	Inhibition	-0.72	0.35
Purvis et al. (2000)	Child	Problem-Solving	-0.69	0.35
Purvis et al. (2000)	Child	Processing Speed	-0.71	0.35
Purvis et al. (2000)	Child	Vocabulary	-0.81	0.36
Rucklidge et al. (2002)	Child	Working Memory	-1.27	0.29
Rucklidge et al. (2002)	Child	Inhibition	-0.70	0.27
Rucklidge et al. (2002)	Child	Problem-Solving	-0.14	0.26
Rucklidge et al. (2002)	Child	Processing Speed	-1.06	0.28
Rucklidge et al. (2002)	Child	Vocabulary	-1.29	0.29
Savitz et al. (2003)	Child	Inhibition	-2.72	0.36
Savitz et al. (2003)	Child	Vocabulary	-0.37	0.27
Seidman et al. (2001)	Child	Working Memory	-0.94	0.27
Seidman et al. (2001)	Child	Memory	-0.01	0.27
Seidman et al. (2001)	Child	Attention	0.27	0.27
Seidman et al. (2001)	Child	Inhibition	-0.92	0.27
Seidman et al. (2001)	Child	Cognitive Flexibility	-0.85	0.27
Seidman et al. (2001)	Child	Planning	-0.33	0.27
Seidman et al. (2001)	Child	Processing Speed	-0.66	0.27
Shanahan et al. (2006)	Child	Inhibition	-1.11	0.17
Shanahan et al. (2006)	Child	Cognitive Flexibility	-0.73	0.17
Shanahan et al. (2006)	Child	Processing Speed	-1.22	0.18
Shanahan et al. (2006)	Child	Vocabulary	-1.74	0.19
Shaywitz et al. (1995)	Child	Working Memory	-1.36	0.25
Shaywitz et al. (1995)	Child	Attention	-0.34	0.22
Shaywitz et al. (1995)	Child	Processing Speed	-1.72	0.26
Shaywitz et al. (1995)	Child	Vocabulary	-1.70	0.26
Shin et al. (2003)	Child	Memory	-0.41	0.34
Shin et al. (2003)	Child	Vocabulary	-0.32	0.34
Stubenrauch et al. (2014)	Child	Inhibition	-0.07	0.32
Swanson et al. (1999)	Child	Working Memory	-0.96	0.30
Swanson et al. (1999)	Child	Vocabulary	-0.38	0.29
Tannock et al. (2000)	Child	Problem-Solving	-0.54	0.30
Tannock et al. (2000)	Child	Processing Speed	-1.76	0.35
Tannock et al. (2000)	Child	Vocabulary	-1.75	0.35
Tiffin-Richards et al. (2008)	Child	Working Memory	-1.41	0.36
Tiffin-Richards et al. (2008)	Child	Cognitive Flexibility	-0.84	0.33
Tiffin-Richards et al. (2008)	Child	Vocabulary	-1.29	0.35
Toplak et al. (2003)	Child	Working Memory	-1.61	0.30
Toplak et al. (2003)	Child	Problem-Solving	-0.08	0.26
Toplak et al. (2003)	Child	Processing Speed	-1.06	0.28
Toplak et al. (2003)	Child	Vocabulary	-1.22	0.28
Turker et al. (2019)	Child	Working Memory	-1.13	0.29
Turker et al. (2019)	Child	Vocabulary	-0.74	0.28
Van de Voorde et al. (2010)	Child	Working Memory	-0.24	0.32
Van de Voorde et al. (2010)	Child	Inhibition	-1.65	0.37

Study	Age Group	Cognitive Domain	Effect Size	Standard Error
Van de Voorde et al. (2010)	Child	Processing Speed	-1.20	0.35
Wang et al. (2018)	Child	Working Memory	-1.42	0.30
Wang et al. (2018)	Child	Attention	-1.33	0.29
Wang et al. (2018)	Child	Inhibition	-1.08	0.28
Wang et al. (2018)	Child	Processing Speed	-1.63	0.31
Willcutt et al. (2001)	Child	Working Memory	-0.94	0.23
Willcutt et al. (2001)	Child	Inhibition	-0.88	0.21
Willcutt et al. (2001)	Child	Cognitive Flexibility	-0.77	0.18
Willcutt et al. (2001)	Child	Vocabulary	-2.60	0.23
Willcutt et al. (2005)	Child	Processing Speed	-1.29	0.16
Wu et al. (2002)	Child	Working Memory	-0.83	0.28
Wu et al. (2002)	Child	Attention	-0.65	0.29
Wu et al. (2002)	Child	Cognitive Flexibility	-0.58	0.28
Wu et al. (2002)	Child	Planning	-0.06	0.27
Wu et al. (2002)	Child	Processing Speed	-1.00	0.29
Zhang et al. (2022)	Child	Working Memory	-0.26	0.19
Zhang et al. (2022)	Child	Attention	-0.94	0.18
Zhang et al. (2022)	Child	Planning	-1.66	0.19
Zhang et al. (2022)	Child	Problem-Solving	0.29	0.19
Zhang et al. (2022)	Child	Processing Speed	-0.73	0.18
Zhang et al. (2022)	Child	Vocabulary	-0.10	0.19
Gregg et al. (2008)	Adult	Processing Speed	-0.80	0.14
Gregg et al. (2008)	Adult	Vocabulary	-0.46	0.16
Katz et al. (2011)	Adult	Working Memory	-0.24	0.13
Katz et al. (2011)	Adult	Attention	-0.32	0.13
Katz et al. (2011)	Adult	Inhibition	-0.99	0.14
Katz et al. (2011)	Adult	Processing Speed	-0.46	0.13
Laasonen et al. (2012)	Adult	Attention	-0.34	0.40
Laasonen et al. (2012)	Adult	Inhibition	-0.38	0.39
Laasonen et al. (2012)	Adult	Cognitive Flexibility	-0.31	0.39
Laasonen et al. (2012)	Adult	Planning	0.48	0.40
Laasonen et al. (2009)	Adult	Working Memory	-0.56	0.37
Laasonen et al. (2009)	Adult	Problem-Solving	-0.31	0.37
Laasonen et al. (2009)	Adult	Processing Speed	-1.37	0.39
Laasonen et al. (2009)	Adult	Vocabulary	-0.56	0.37
Miranda et al. (2017)	Adult	Working Memory	-1.56	0.37
Miranda et al. (2017)	Adult	Inhibition	-1.32	0.36
Miranda et al. (2017)	Adult	Cognitive Flexibility	-0.99	0.35
Miranda et al. (2017)	Adult	Planning	-1.50	0.37
Miranda et al. (2017)	Adult	Problem-Solving	-1.61	0.38
Whipple et al. (2016)	Adult	Processing Speed	-0.91	0.15

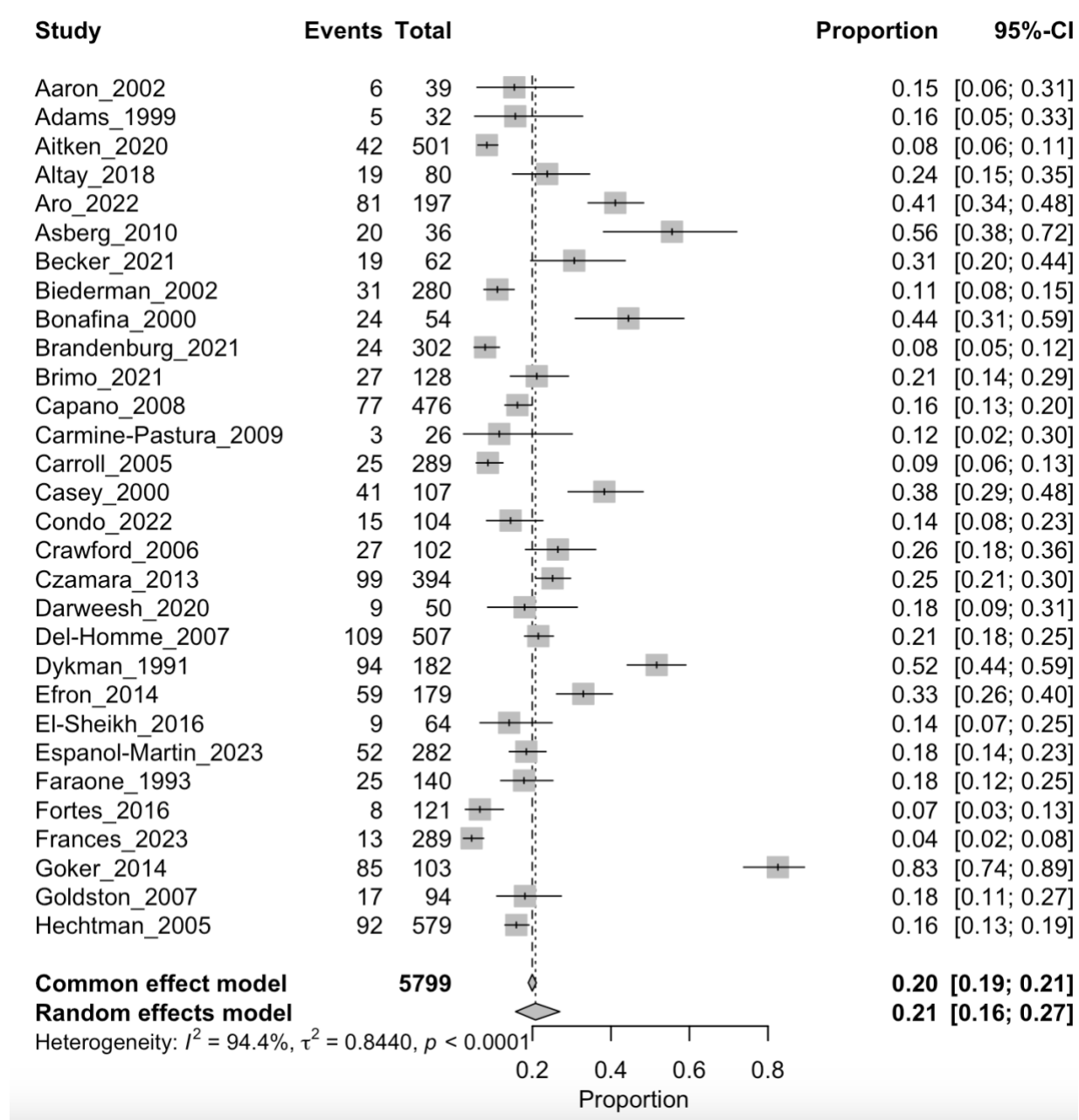
Note. Effect sizes (Cohen's *d* or Hedges' *g*) and standard errors are displayed instead of means, standard deviations and sample sizes for the following reasons: a) some studies reported multiple data for a given cognitive domain, with varying sample sizes for each measure, so a weighted average Hedges' *g* was calculated, b) some studies had missing data (i.e., unavailable control group), so published normative data were used as a substitute (with an uncertain sample size), therefore Cohen's *d* was calculated as it does not require sample size information. Note that the studies are presented in alphabetical order for child studies first, followed by adult studies.

Figure 2. Forest plot displaying proportions of ADHD+RD and 95% confidence intervals for adult studies addressing RQ1.



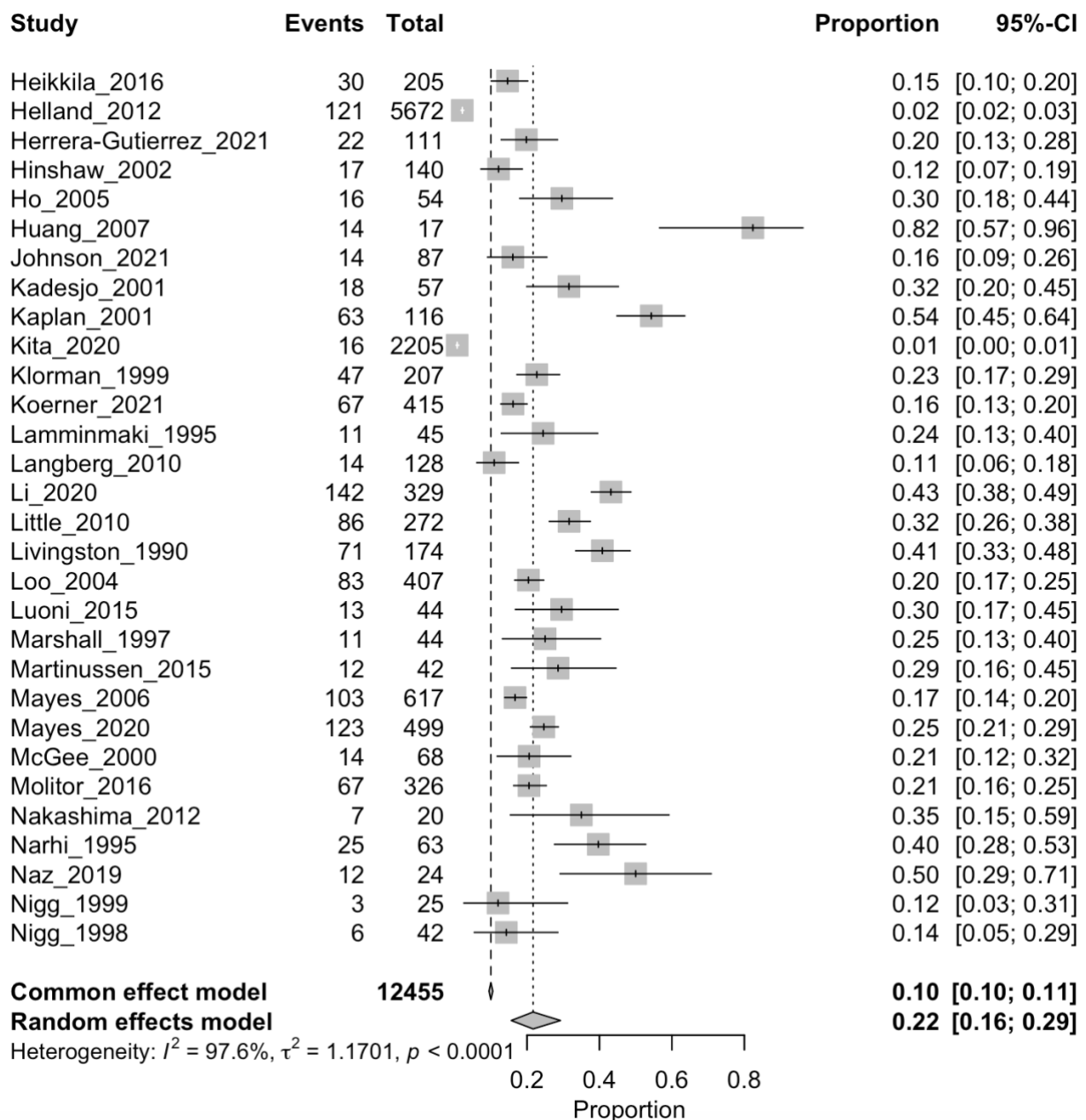
Note. ‘Events’ = number of individuals with ADHD+RD. ‘Total’ = sample from which the proportion of ADHD+RD was calculated.

Figure 3. Forest plot displaying proportions of ADHD+RD and 95% confidence intervals for the first third of child studies addressing RQ1.



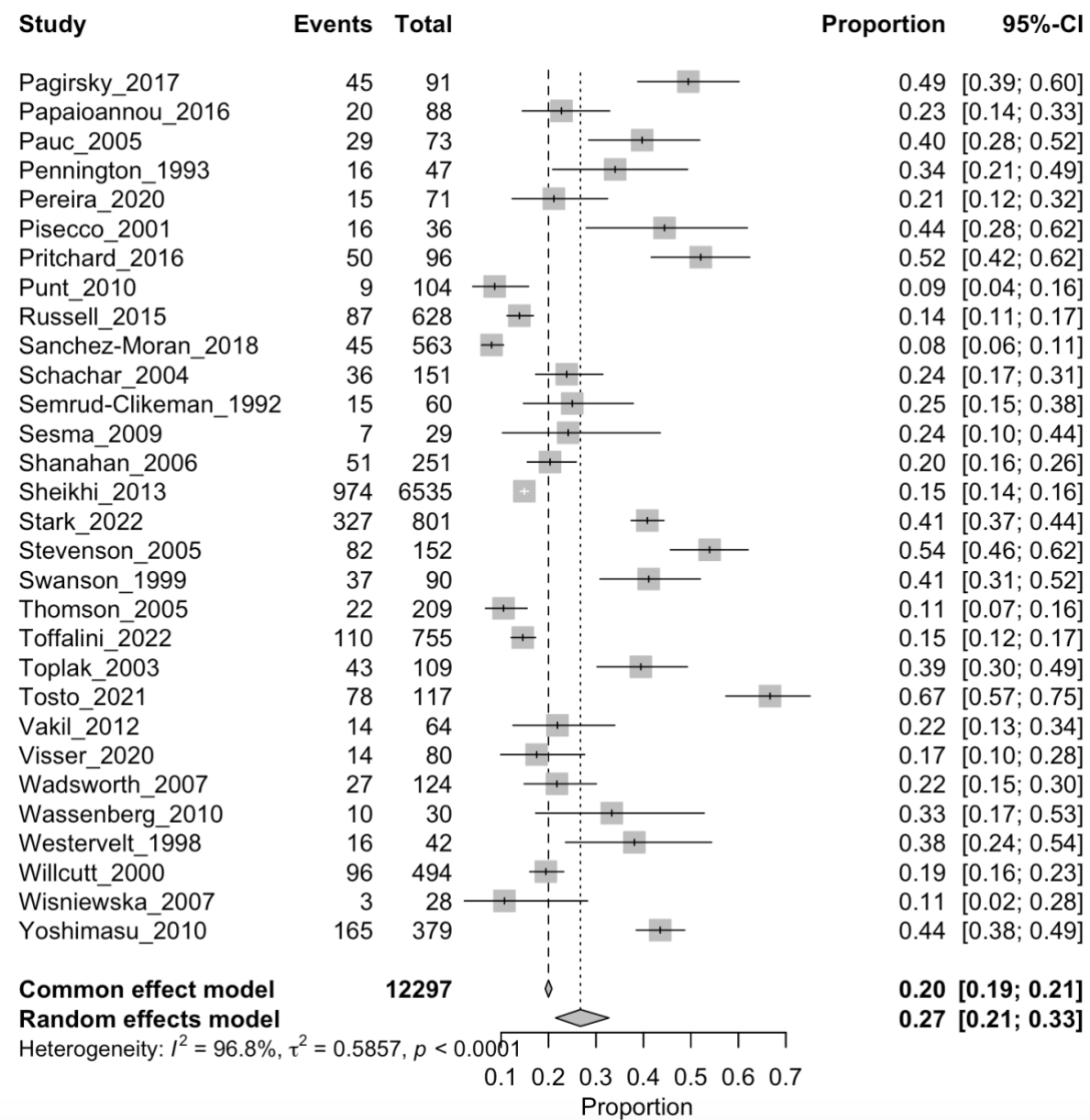
Note. ‘Events’ = number of individuals with ADHD+RD. ‘Total’ = sample from which the proportion of ADHD+RD was calculated. The ‘Common effects model’ and ‘Random effects model’ represent the pooled effect estimate for this specific subset of child studies addressing RQ1, not the overall pooled effect estimate across all studies. This also applies to the heterogeneity statistics.

Figure 4. Forest plot displaying proportions of ADHD+RD and 95% confidence intervals for the second third of child studies addressing RQ1.



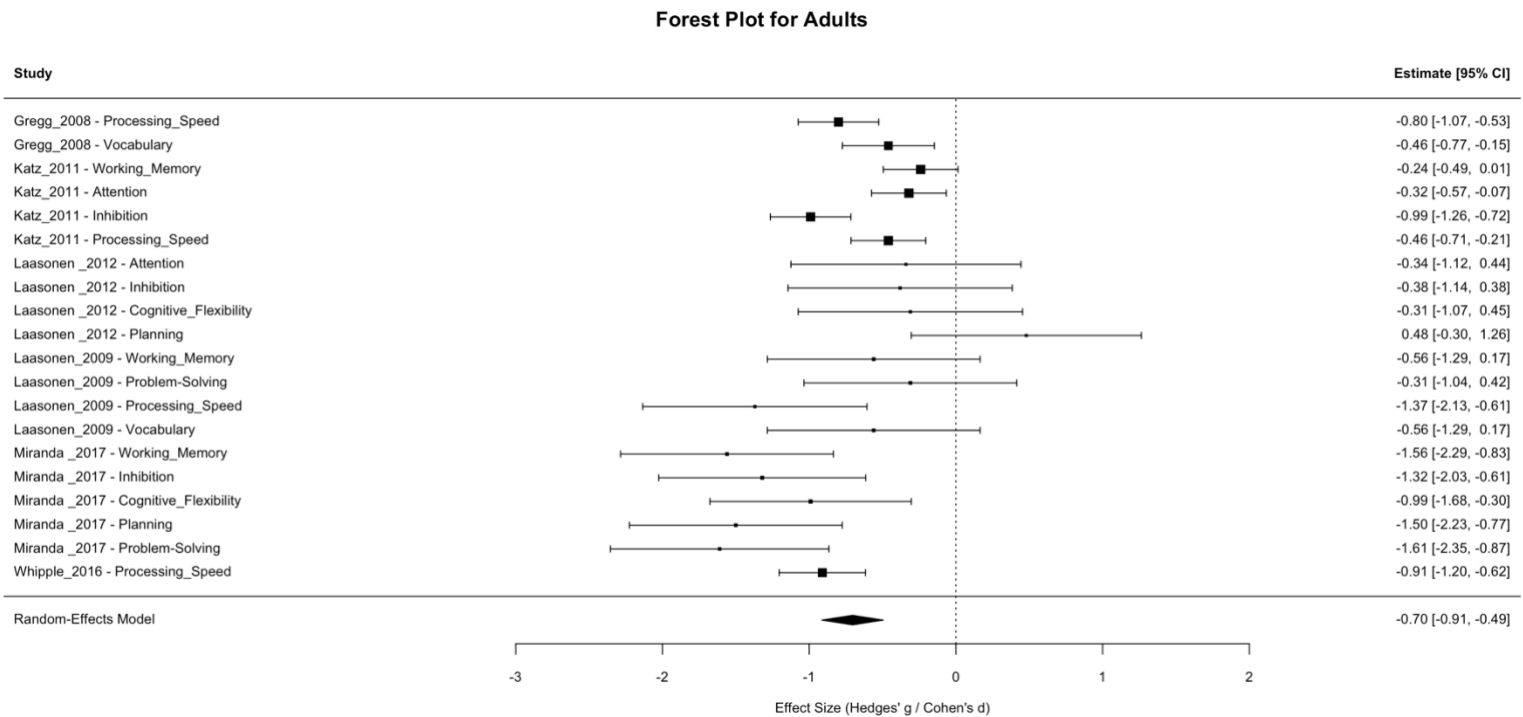
Note. ‘Events’ = number of individuals with ADHD+RD. ‘Total’ = sample from which the proportion of ADHD+RD was calculated.

Figure 5. Forest plot displaying proportions of ADHD+RD and 95% confidence intervals for the final third of child studies addressing RQ1.



Note. ‘Events’ = number of individuals with ADHD+RD. ‘Total’ = sample from which the proportion of ADHD+RD was calculated.

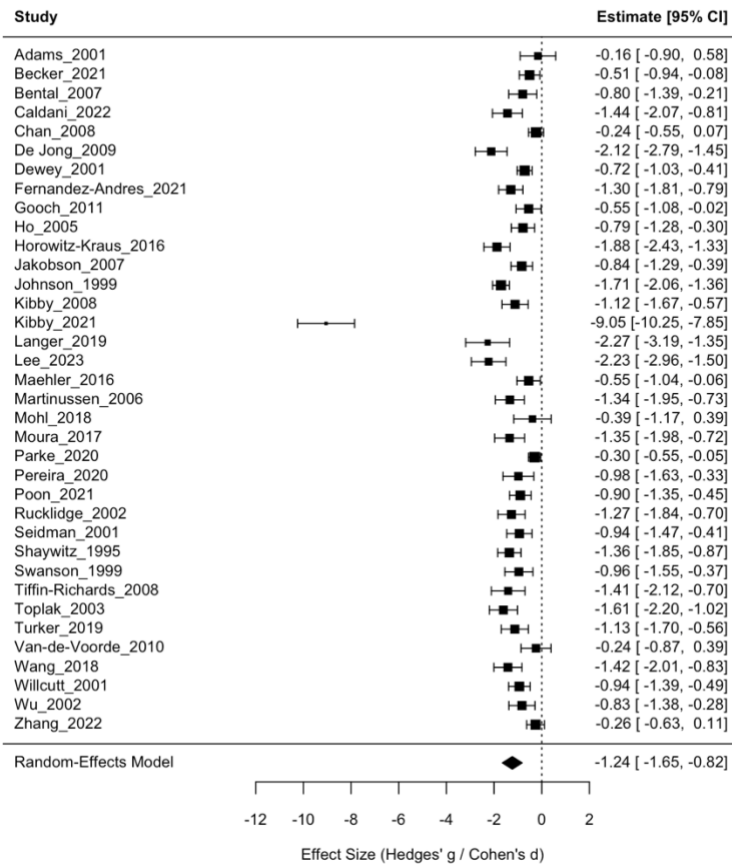
Figure 6. Forest plot displaying effect estimates and 95% confidence intervals for adult studies addressing RQ2.



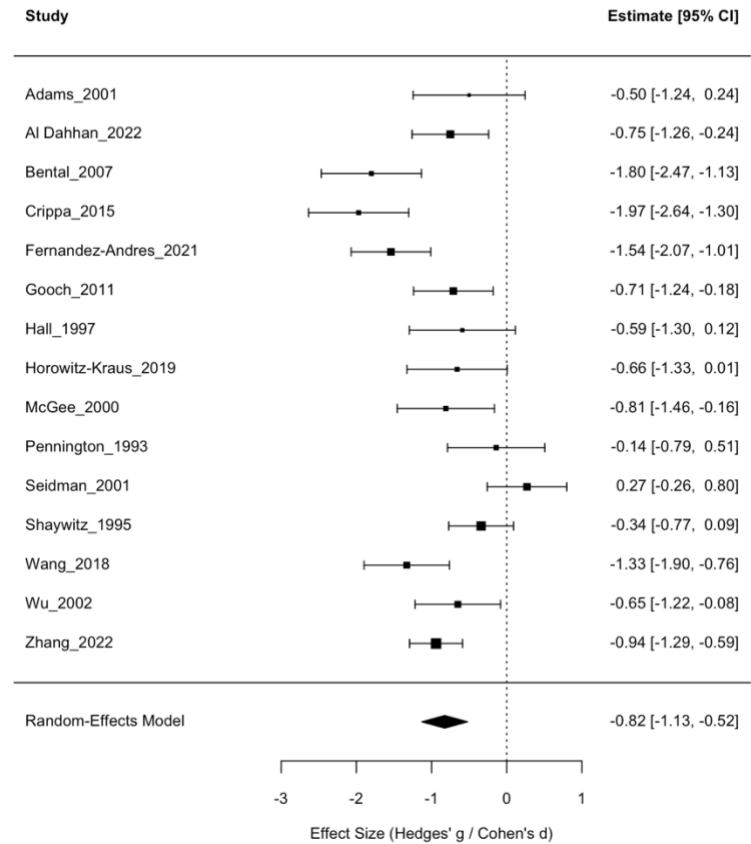
Note. Studies are presented multiple times based on the number of cognitive domains assessed by each study. The effect estimates display the magnitude of the difficulties in each cognitive domain as represented by the effect size of the difference between the ADHD+RD and control groups (or population norms) for each study.

Figure 7. Forest plots displaying effect estimates and 95% confidence intervals for child studies assessing working memory and attention.

Forest Plot for Children - Working_Memory



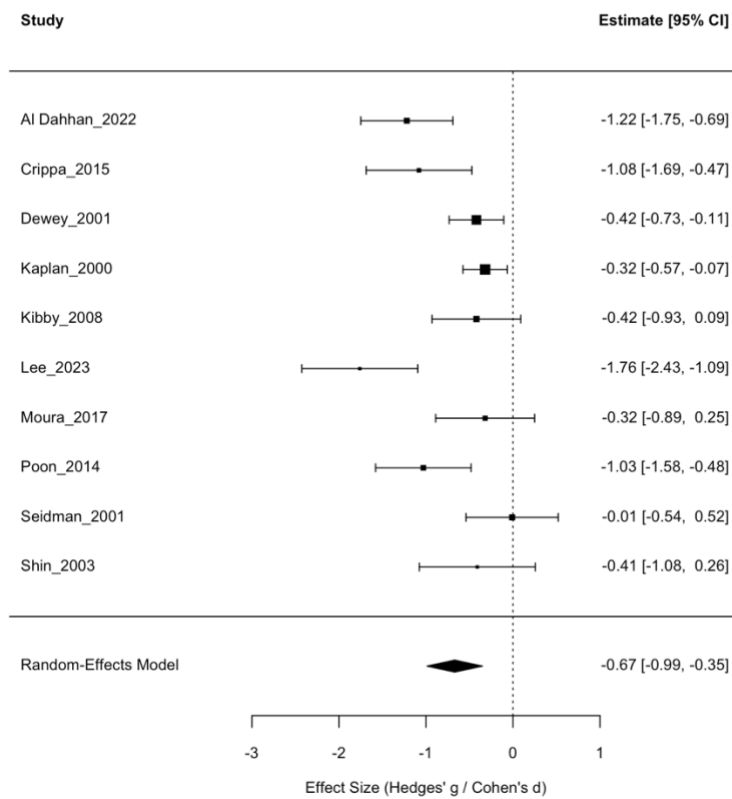
Forest Plot for Children - Attention



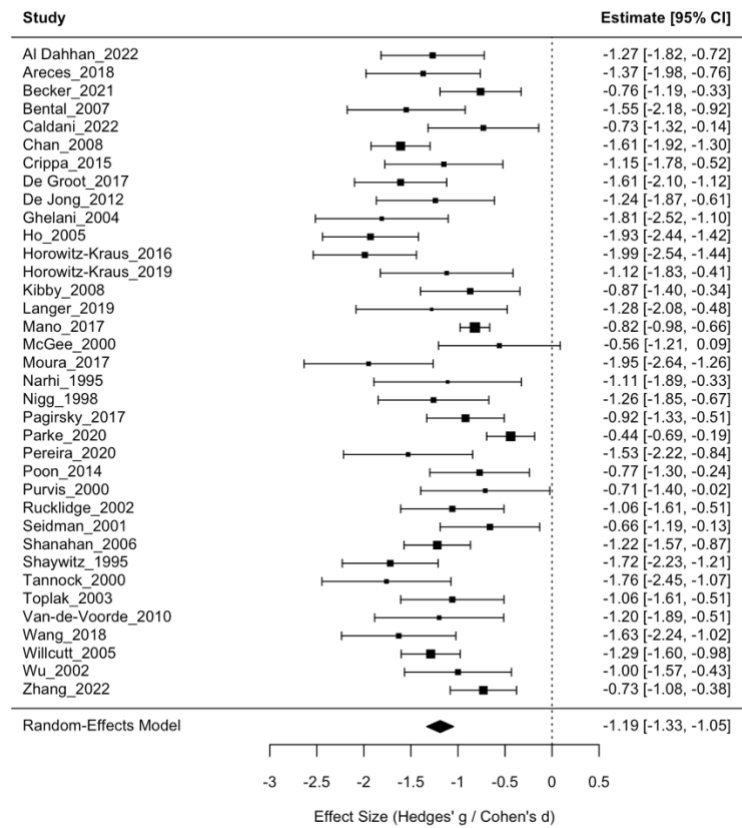
Note. The effect estimates display the magnitude of the difficulties in working memory and attention as represented by the effect size of the difference between the ADHD+RD and control groups (or population norms) for each study.

Figure 8. Forest plots displaying effect estimates and 95% confidence intervals for child studies assessing memory and processing speed.

Forest Plot for Children - Memory



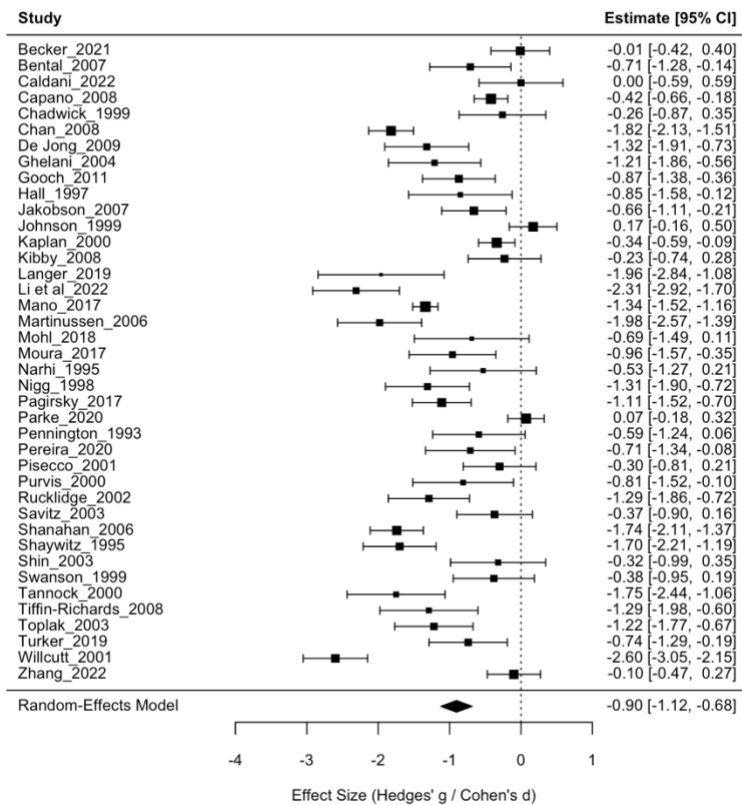
Forest Plot for Children - Processing_Speed



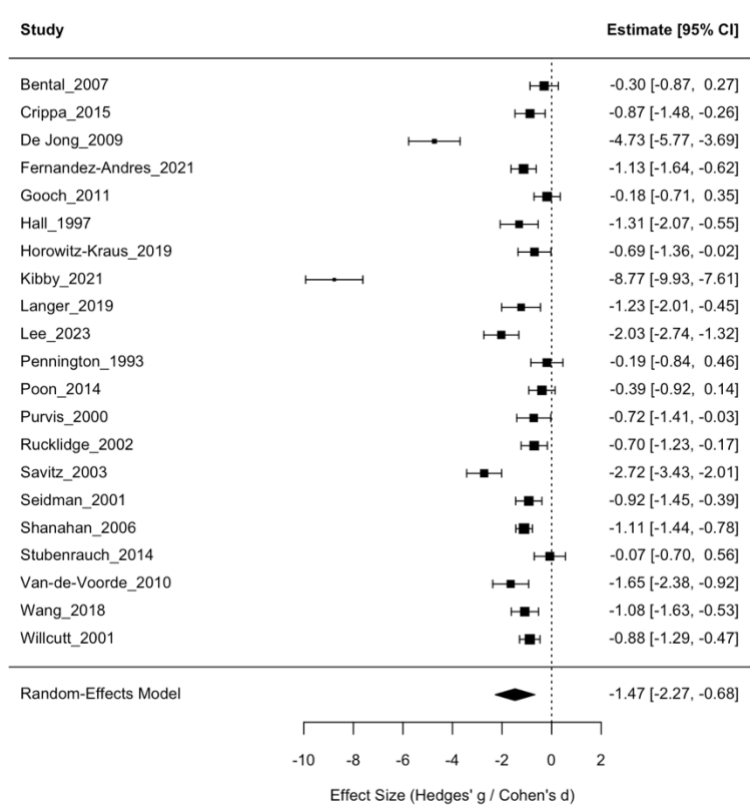
Note. The effect estimates display the magnitude of the difficulties in general memory and processing speed as represented by the effect size of the difference between the ADHD+RD and control groups (or population norms) for each study.

Figure 9. Forest plots displaying effect estimates and 95% confidence intervals for child studies assessing vocabulary and inhibition.

Forest Plot for Children - Vocabulary

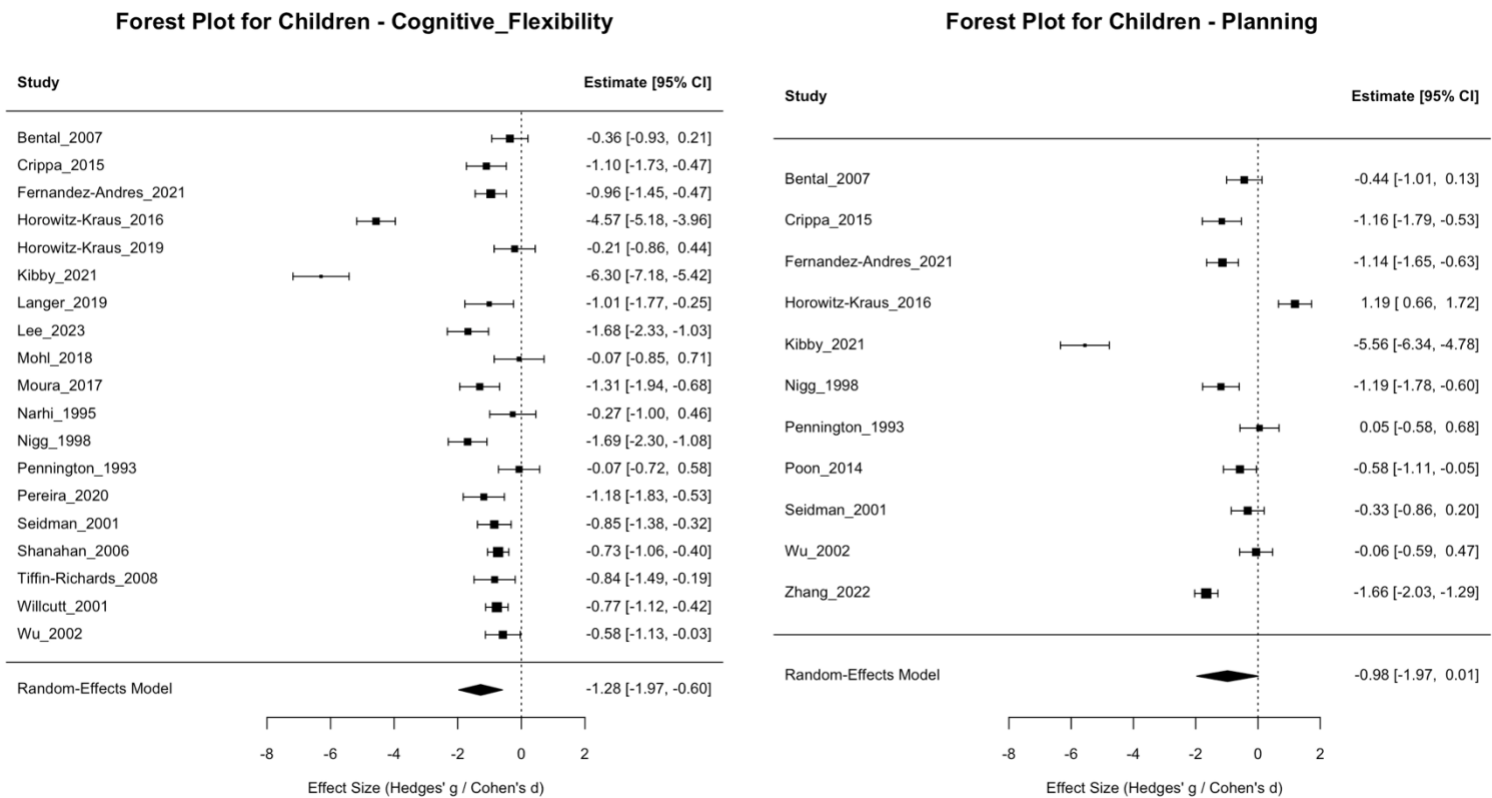


Forest Plot for Children - Inhibition



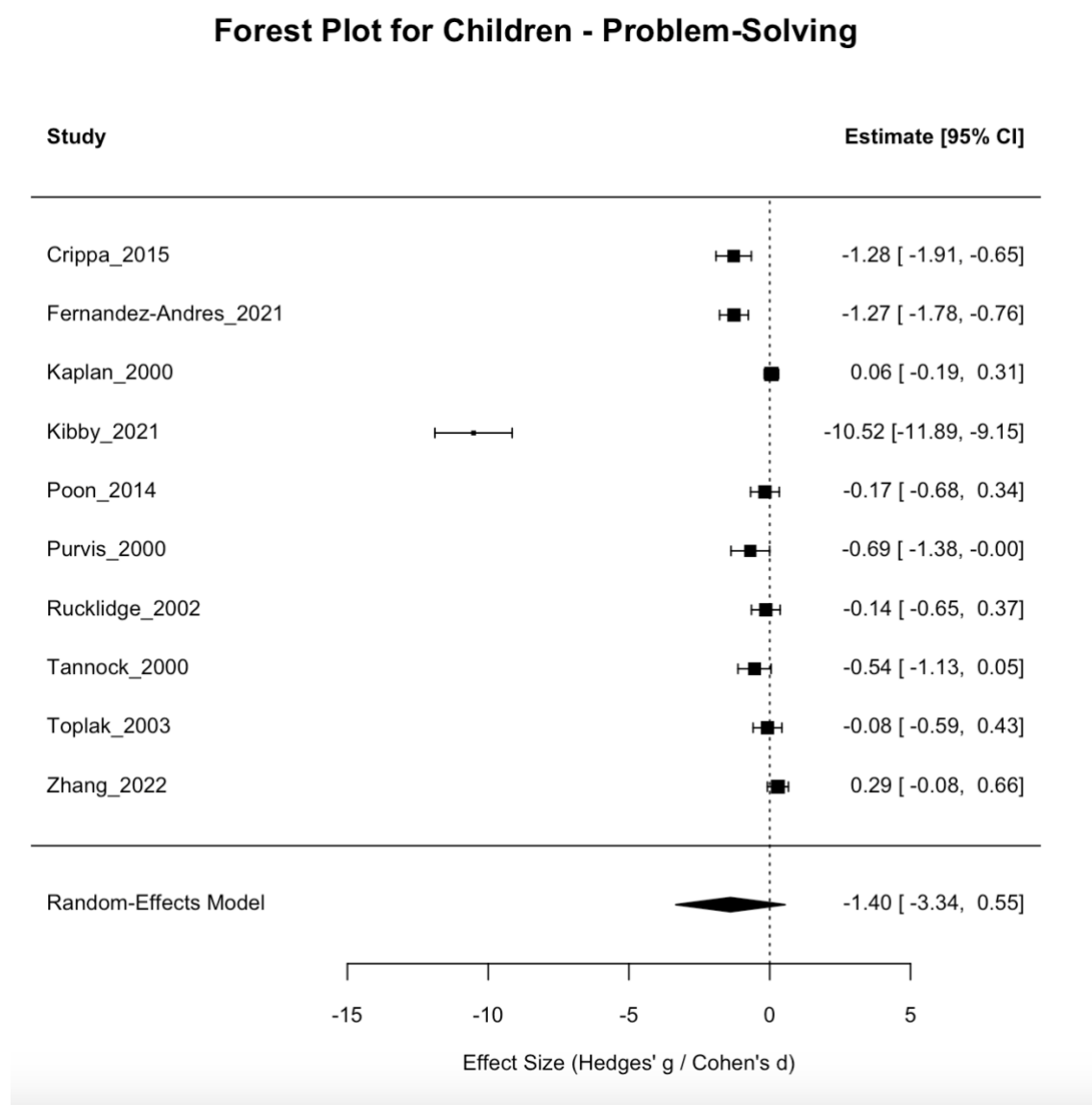
Note. The effect estimates display the magnitude of the difficulties in vocabulary and inhibition as represented by the effect size of the difference between the ADHD+RD and control groups (or population norms) for each study.

Figure 10. Forest plots displaying effect estimates and 95% confidence intervals for child studies assessing cognitive flexibility and planning.



Note. The effect estimates display the magnitude of the difficulties in cognitive flexibility and planning as represented by the effect size of the difference between the ADHD+RD and control groups (or population norms) for each study.

Figure 11. Forest plot displaying effect estimates and 95% confidence intervals for child studies assessing problem-solving.



Note. The effect estimates display the magnitude of the difficulties in problem-solving as represented by the effect size of the difference between the ADHD+RD and control groups (or population norms) for each study.

2.4.5. Results of Syntheses

2.4.5.1. Meta-Analysis of Proportions (RQ1)

The pooled proportion (i.e., prevalence rate) of co-occurring ADHD+RD across both children and adults was 0.23 (95% CI [0.20, 0.27]), with substantial heterogeneity ($\tau^2 = 0.92$, $I^2 = 97.5\%$). For child studies alone, the pooled proportion of ADHD+RD was 0.23 (95% CI [0.20, 0.27]), with relatively lower, yet still high levels of heterogeneity ($\tau^2 = 0.88$, $I^2 = 96.7\%$). For adult studies, the pooled proportion was 0.23 (95% CI [0.12, 0.40]), with substantially high levels of heterogeneity ($\tau^2 = 1.28$, $I^2 = 99.1\%$). The difference in pooled proportions between children and adults was statistically significant ($z = 30.79$, $p < .001$), with a small to moderate effect ($h = 0.33$). However, the absolute difference in proportions was negligible (0.2294 in children, vs. 0.2293 in adults), indicating limited practical significance. A separate z-test using raw event counts and sample sizes similarly found a statistically significant difference ($z = 8.71$, $p < .001$). Although these results suggest higher co-occurrence in children, the identical pooled proportions indicate that the significance is likely driven by the large overall sample size, rather than a meaningful group difference.

2.4.5.1.1. Subgroup Analyses

For subgroup analyses based on sampling method (see Table 3 for studies contributing to each subgroup), the pooled proportion of ADHD+RD in studies using mixed sampling was 0.07 (95% CI [0.03, 0.13]), with substantially high heterogeneity ($\tau^2 = 1.15$, $I^2 = 98.7\%$). For studies employing purposive sampling methods, the pooled proportion was 0.26 (95% CI [0.22, 0.29]), with slightly lower, yet still high heterogeneity ($\tau^2 = 0.65$, $I^2 = 96.8\%$). The difference in proportions of ADHD+RD between studies using mixed sampling and purposive sampling was significant ($z = -29.30$, $p < .001$), with a small to moderate effect ($h = -0.29$). The 95% CI for the difference in proportions was [-0.10, -0.09], with studies employing purposive sampling demonstrating a higher proportion of individuals with ADHD+RD.

For subgroup analyses based on definitions of RD, the pooled proportion of ADHD+RD in studies with an unclear (or not widely used) definition of RD was 0.20 (95% CI [0.14, 0.29]), with high levels of heterogeneity ($\tau^2 = 1.70$, $I^2 = 98.5\%$). Studies using the ‘discrepancy’ model to define RD demonstrated a pooled proportion of 0.24 (95% CI [0.18, 0.32]), with relatively

lower, yet still substantial heterogeneity ($\tau^2 = 0.62$, $I^2 = 95.1\%$). Further, for studies using the ‘low achievement’ model of RD, the pooled prevalence of ADHD+RD was 0.26 (95% CI [0.21, 0.31]), with even lower but still substantial levels of heterogeneity ($\tau^2 = 0.50$, $I^2 = 92.3\%$). Finally, studies using a combination of the ‘discrepancy’ and ‘low achievement’ models displayed a pooled proportion of 0.18 (95% CI [0.12, 0.25]), with high levels of heterogeneity ($\tau^2 = 0.42$, $I^2 = 95.2\%$). A Chi-squared test revealed no significant differences in proportions of ADHD+RD between studies employing different models to define RD ($\chi^2 = 0.07$, $p = .995$); therefore, pairwise comparisons were not necessary.

2.4.5.1.2. Sensitivity Analyses

A leave-one-out (LOO) sensitivity analysis of the pooled proportions of ADHD+RD across both children and adults revealed that the main analysis results were robust to the exclusion of individual studies. The LOO results indicated a stable logit-transformed effect size (ranging from -1.23 to -1.16, 95% CI [-1.39, -1.00]). This corresponds to an estimated pooled proportion of approximately 0.23 (ranging from 0.22 to 0.24), with a back-transformed 95% CI ranging from 0.20 to 0.27. No study changed the effect size by more than the threshold of 0.1 when removed, therefore no individual studies disproportionately affected the results of the analysis. Additionally, heterogeneity remained high across all iterations (mean $I^2 = 97.2\%$), suggesting that variability was not driven by any single influential study.

A LOO sensitivity analysis of the child subgroup revealed that the logit-transformed effect sizes remained consistent across iterations (ranging from -1.24 to -1.17), which corresponds to pooled proportions ranging from 0.22 to 0.24. Both the lower [-1.44, -1.35] and upper [-1.05, -0.98] 95% CIs (i.e., back-transformed 95% CIs ranging from 0.19 to 0.27) remained stable across individual study exclusions. Additionally, no single study was responsible for the high levels of heterogeneity, as these remained high across all exclusions ($I^2 = 95-97\%$). There was one study (Kita et al., 2020) that influenced a change in effect size above the 0.1 threshold, although the results remained relatively robust despite this effect. Conversely, results for the adult subgroup showed less stability compared to child studies. The logit-transformed pooled effect size varied from -1.37 to -0.89 (corresponding to approximately 0.20 to 0.29 pooled proportions), with 95% CIs ranging from 0.10 to 0.45 after back-transformation, showing some uncertainty. Heterogeneity remained consistently high across all iterations ($I^2 = 92-99\%$), and there was one study (Cederlof et al., 2017) that substantially impacted the results.

Further, results for the mixed sampling group were relatively stable, with logit-transformed pooled effect sizes ranging from -2.81 to -2.35 (corresponding to around 0.06 to 0.09 pooled proportions), with 95% CIs showing relatively small variation [0.03, 0.15] after back-transformation. Heterogeneity levels remained high ($I^2 = 97-99\%$) across iterations, indicating consistent between-study variability. One study (Kita et al., 2020) was found to be influential, but its removal had minimal impact on the analysis as results remained consistent. Similarly, results from the purposive sampling subgroup were relatively stable, with logit-transformed effect sizes ranging from -1.09 to -1.03 (corresponding to approximately 0.25 to 0.26 pooled proportions), with 95% CIs showing good stability [0.22, 0.30]. Again, heterogeneity levels remained consistently high across exclusions ($I^2 = 94-97\%$), suggesting that no study considerably influenced variance between studies. One study (Goker et al., 2014) was flagged as influential, but its removal had minimal impact on the results.

Moreover, the LOO analysis showed that results for RD definition subgroup analyses were also robust. For studies with an unclear (or not widely used) definition of RD, the logit-transformed pooled effect sizes were stable and ranged from -1.47 to -1.24 (corresponding to around 0.19 to 0.22 pooled proportions), with 95% CIs also showing relatively good stability [0.13, 0.31]. Heterogeneity levels remained consistently high across study exclusions ($I^2 = 98-99\%$). Kita et al. (2020) was found to be an influential study again, yet its impact on the overall results was minimal. Results of the ‘discrepancy’ subgroup were also stable, with pooled logit effect sizes ranging from -1.20 to -1.06 (corresponding to 0.23 to 0.26 in back-transformed pooled proportions), and relatively narrow 95% CIs ranging from 0.17 to 0.33 (after back-transformation). Heterogeneity also remained high ($I^2 = 95\%$) across iterations. One study was found to be influential again (Stevenson et al., 2005), but it did not substantially destabilise the results.

Lastly, studies employing the ‘low achievement’ model to define RD also displayed stability in logit effect sizes (ranging from -1.10 to -1.01, corresponding to 0.25-0.27 in pooled proportions), with relatively narrow back-transformed 95% CIs [0.21, 0.31]. Again, heterogeneity remained high across iterations ($I^2 = 91-92\%$), showing that no single study significantly influenced between-study variance. One study was found to be particularly influential (Frances et al., 2023), but it did not disproportionately affect the results. Similarly, for studies using a combination of the ‘discrepancy’ and ‘low achievement’ definitions, the

pooled proportions showed good stability (varying from -1.71 to -1.42, corresponding to 0.15-0.20 in back-transformed pooled proportions). To this end, the 95% CIs were relatively narrow, ranging from 0.11 to 0.27. There was some variation in heterogeneity ($I^2 = 85\text{-}96\%$), suggesting that the study flagged as influential (Yoshimasu et al., 2010) may have contributed to between-study variance.

2.4.5.1.3. Risk of Reporting Bias

For visualisations of Doi plots, please refer to Appendix K (Supplementary Materials 1). The overall pooled proportion of ADHD+RD across child and adult studies showed minor asymmetry (LFK = 1.59), suggesting potential publication bias (Figure 1, Appendix K, Supplementary Materials 1). For child-only studies, the asymmetry was very minor (LFK = 1.1; Figure 2), while adult-only studies showed major asymmetry (LFK = 4.99), indicating substantial risk of publication bias (Figure 3). Mixed-sampling studies also showed major asymmetry (LFK = -4.51; Figure 4), indicating substantial publication bias. In contrast, studies using purposive sampling showed no asymmetry (LDK = 0.97; Figure 5). Regarding RD definitions, studies with an unclear (or not widely used) definition showed minor asymmetry (LFK = 1.91; Figure 6), indicating potential publication bias. Those using the ‘discrepancy’ model showed no asymmetry and thus no risk of publication bias (LFK = -0.17; Figure 7), while studies using the ‘low achievement’ model showed minor asymmetry (LFK = 1.78; Figure 8), indicating potential publication bias. Finally, studies using a combination of both models showed minor asymmetry (LFK = -1.87; Figure 9), suggesting potential publication bias.

2.4.5.2. Meta-Analysis of Standardised Mean Differences (RQ2)

A meta-analysis of standardised mean differences revealed that the pooled effect estimate of overall cognitive performance across child studies was -1.11 (SE = 0.09, 95% CI [-1.28, -0.95]), with a significantly strong negative effect ($z = -13.07$, $p < .001$). There were substantially high levels of heterogeneity among these results ($\tau^2 = 1.35$, $I^2 = 95.7\%$). For adult studies, the pooled effect estimate was -0.70 (SE = 0.11, 95% CI [-0.91, -0.49]), with a significantly strong negative effect ($z = 6.59$, $p < .001$). Levels of heterogeneity were lower among adult studies, yet still significant ($\tau^2 = 0.15$, $I^2 = 75.9\%$). The results suggest that children displayed more difficulties in overall cognitive performance compared to adults; however,

statistical analyses for direct comparisons were not conducted given that adult data were not available for every cognitive domain.

2.4.5.2.1. Meta-Regression and Independent Samples T-Test

Results of the meta-regression using cognitive domain as a moderator revealed that attention significantly decreased the pooled effect size estimate in child studies (see Table 5). No other cognitive domains were found to significantly contribute to the overall variability in pooled effect sizes across child and adult studies. The results showed that differences in cognitive domain accounted for 0% (R^2) of the total heterogeneity in both child and adult studies (see Tables 5-6). Therefore, residual heterogeneity remained high in both children ($\tau^2 = 1.38$, $I^2 = 95.7\%$) and adults ($\tau^2 = 0.24$, $I^2 = 80.3\%$), after accounting for differences in cognitive domain.

Results of the independent samples *t*-test (Mann-Whitney U) indicated that there were no significant differences in effect size between children and adults in the domain of attention ($t(15) = 4.5$, $p = .136$), despite children ($M = -0.83$, $SD = 0.61$) demonstrating higher effect sizes than adults ($M = -0.33$, $SD = 0.01$). Similar non-significant differences were observed for cognitive flexibility ($t(19) = 15.0$, $p = .675$), although children ($M = -1.29$, $SD = 1.56$) did display slightly higher effect sizes than adults ($M = -0.65$, $SD = 0.48$). Results of the Mann-Whitney U test were also not significant for the domain of inhibition ($t(22) = 31.0$, $p = 1.000$), despite children ($M = -1.51$, $SD = 1.96$) showing higher effect sizes than adults ($M = -0.90$, $SD = 0.48$). Further, no significant differences in effect size were found between children ($M = -0.99$, $SD = 1.70$) and adults ($M = -0.51$, $SD = 1.40$) in planning ($t(11) = 10.0$, $p = .923$). Similarly, differences in effect sizes between children ($M = -1.43$, $SD = 3.24$) and adults ($M = -0.96$, $SD = 0.92$) were not significant for the domain of problem-solving ($t(10) = 6.00$, $p = .485$). The results of the independent samples *t*-test also revealed no significant differences in effect sizes between children ($M = -1.21$, $SD = 0.42$) and adults ($M = -0.89$, $SD = 0.38$) in processing speed ($t(38) = 43.5$, $p = .207$). Between-group differences for the vocabulary domain were also not significant ($t(40) = 27.0$, $p = .460$), with children ($M = -0.91$, $SD = 0.69$) displaying slightly higher effect sizes than adults ($M = -0.51$, $SD = 0.07$). Finally, there were no significant differences found between children and adults in the domain of working memory ($t(37) = 40.0$, $p = .477$), despite children ($M = -1.28$, $SD = 1.45$) demonstrating slightly higher effect sizes than adults ($M = -0.79$, $SD = 0.69$).

Table 5. Meta-regression results with cognitive domain as moderator for child studies.

Domain	Effect Estimate	SE	<i>z</i>	<i>p</i>	95% CI LB	95% CI UB
Attention (Reference Domain)	-0.83	0.31	-2.65	0.01*	-1.44	-0.22
Cognitive Flexibility	-0.45	0.42	-1.06	0.29	-1.27	0.38
Inhibition	-0.61	0.41	-1.47	0.14	-1.41	0.20
Memory	0.14	0.49	0.27	0.78	-0.83	1.10
Planning	-0.14	0.48	-0.29	0.77	-1.08	0.80
Problem-Solving	-0.39	0.50	-0.78	0.43	-1.36	0.58
Processing Speed	-0.38	0.37	-1.02	0.31	-1.11	0.35
Vocabulary	-0.08	0.37	-0.22	0.83	-0.80	0.64
Working Memory	-0.40	0.37	-1.08	0.28	-1.13	0.33

Note. * $p < 0.01$. *SE* = Standard Error. *CI* = Confidence Interval. *LB* = Lower Bound. *UB* = Upper Bound. Attention was the baseline domain to which the estimated effect sizes of other cognitive domains were compared.

Table 6. Meta-regression results with cognitive domain as moderator for adult studies.

Domain	Effect Estimate	SE	<i>z</i>	<i>p</i>	95% CI LB	95% CI UB
Attention (Reference Domain)	-0.33	0.39	-0.83	0.41	-1.10	0.45
Cognitive Flexibility	-0.34	0.59	-0.57	0.57	-1.48	0.81
Inhibition	-0.59	0.51	-1.15	0.25	-1.60	0.42
Planning	-0.21	0.59	-0.36	0.72	-1.37	0.95
Problem-Solving	-0.63	0.59	-1.06	0.29	-1.78	0.53
Processing Speed	-0.51	0.48	-1.07	0.28	-1.44	0.42
Vocabulary	-0.17	0.56	-0.31	0.76	-1.27	0.92
Working Memory	-0.38	0.51	-0.75	0.45	-1.39	0.62

Note. *SE* = Standard Error. *CI* = Confidence Interval. *LB* = Lower Bound. *UB* = Upper Bound. Attention was the baseline domain to which the estimated effect sizes of other cognitive domains were compared. Note that general memory data were not available for adult studies.

2.4.5.2.2. Sensitivity Analyses

A leave-one-out (LOO) sensitivity analysis of the child meta-analysis indicated that the results were robust, with stable effect size estimates ranging from -1.13 to -1.07 (95% CI [-1.29, -0.92]) across individual study exclusions. Heterogeneity levels also remained consistently high across iterations ($I^2 = 94-96\%$). The removal of one study (Kibby et al., 2021) was found to result in a slightly higher effect size compared to the pooled effect estimate, but this study had minimal impact on the overall results. Similarly, results of the adult meta-analysis remained stable across study exclusions, with effect estimates varying from -0.74 to -0.66, with relatively narrow 95% CIs ranging from -0.95 to -0.46. Again, heterogeneity levels remained consistently high across iterations ($I^2 = 72-78\%$), and no single study disproportionately affected the results.

Further, a LOO analysis of the child meta-regression suggested that the results of the analysis were robust to the removal of individual studies. The effect estimates remained stable (varying from -0.91 to -0.75), with all 95% CIs remaining below zero (ranging from -1.55 to -0.11). Heterogeneity remained consistently high across iterations ($I^2 = 94-96\%$), yet no single study disproportionately affected the overall results. Finally, as expected due to the limited number of studies, the results of the adult meta-regression were not as stable. The effect size estimates varied from -1.28 to 0.81, suggesting that the removal of at least one study changed the direction of the effect. Similarly, the 95% CIs were fairly wide (ranging from -2.64 to 2.01), indicating some uncertainty. The levels of heterogeneity showed some variability, fluctuating between substantial and non-substantial with individual study exclusions ($I^2 = 69-83\%$). The removal of one study (Miranda et al., 2017) seemed to have a significant impact on the pooled estimate.

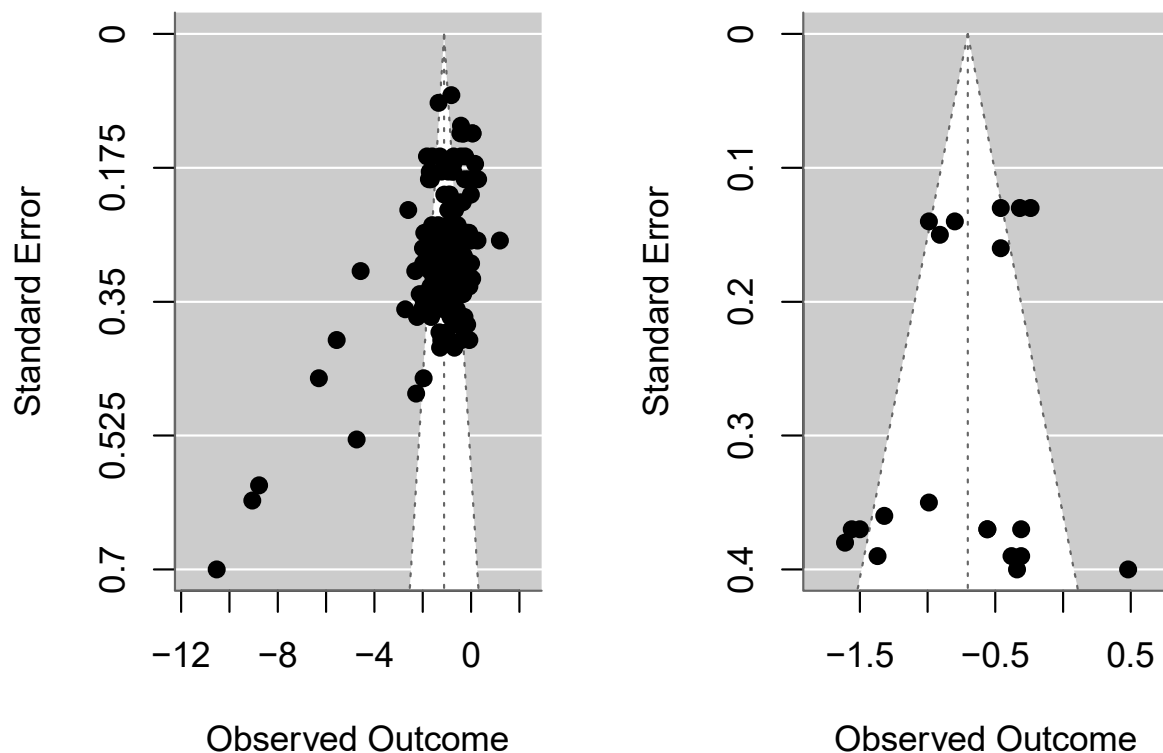
2.4.5.2.3. Risk of Reporting Bias

Egger's test for funnel plot asymmetry (see Figures 12-13) indicated publication bias among child studies in both the main meta-analysis ($p < .001$) and the meta-regression ($p < .001$). Conversely, no publication bias was observed for the adult meta-analysis ($p < .399$), nor for the adult meta-regression ($p < 0.312$).

Since publication bias was detected among child studies, the trim-and-fill method was used to estimate the number of missing studies and adjust the pooled effect estimate accordingly. The

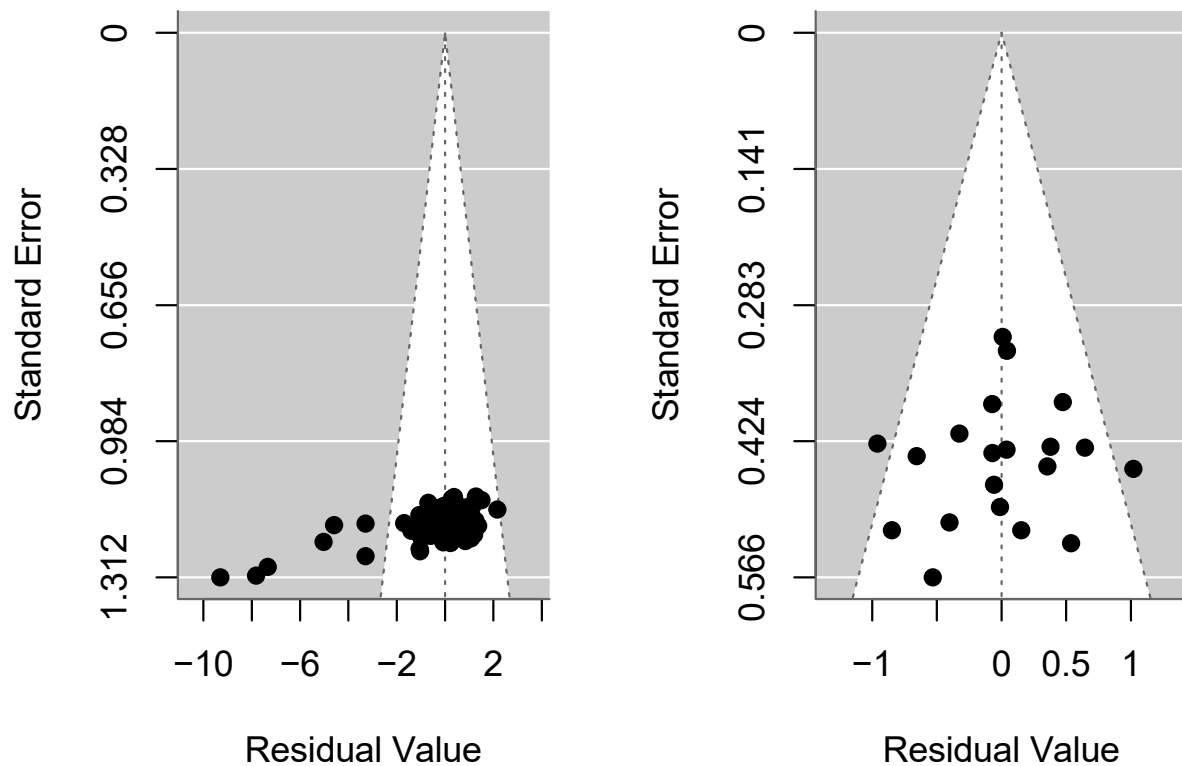
estimated number of missing studies on the right-hand side was 0, with a standard error of 5.74. After adjusting for publication bias, the pooled effect size estimate for child cognitive performance was -1.11 (SE = 0.09, 95% CI [-1.28, -0.95], with a significantly strong negative effect ($z = -13.07$, $p < .001$). Heterogeneity also remained high ($\tau^2 = 1.35$, $I^2 = 95.7\%$). The corrected results were identical to the results of the primary meta-analysis.

Figure 12. Funnel plots displaying risk of publication bias for child (left) and adult (right) studies in the main meta-analysis results.



Note. Each plot displays individual study effect sizes ('Observed Outcome') against their standard errors. Asymmetry in the plot suggests potential small-study effects or publication bias, where studies with non-significant or negative results may be underreported. Studies outside the white funnel (95% confidence region) may be potential outliers.

Figure 13. Funnel plots displaying risk of publication bias for child (left) and adult (right) studies in the meta-regression results (after accounting for cognitive domains as moderators).



Note. The 'Residual Value' represents the difference between observed and model-predicted effect sizes against their standard errors. Asymmetry in the distribution of points may indicate potential publication bias, where studies with non-significant or negative effect sizes may be underreported. Studies outside the white funnel (95% confidence region) may be potential outliers.

2.4.6. Certainty of Evidence

Table 7. Simplified assessment of certainty in the body of evidence for each outcome assessed.

Outcomes	Study Limitations	Consistency of Effect	Imprecision of Results	Publication Bias	Evidence Certainty
Child Prevalence of ADHD+RD	High risk of bias in: a) Sampling procedures b) Methods used to confirm ADHD+RD	$I^2 = 96.7\%$	95% CI [0.20, 0.27]	LFK Index = 1.1 Minor publication bias	Low
Adult Prevalence of ADHD+RD	Some concerns in: a) Appropriateness of statistical analyses	$I^2 = 99.1\%$	95% CI [0.12, 0.40]	LFK Index = 4.99 Major publication bias	Low
Cognitive Performance in Children with ADHD+RD	Some concerns in: a) Exclusion criteria for sampling b) Methods used to confirm ADHD+RD	$I^2 = 95.7\%$	95% CI [-1.28, -0.95]	Egger's Test = $p < .001$ Major publication bias	Low
Cognitive Performance in Adults with ADHD+RD	Low risk of bias in: a) Appropriateness of statistical analyses	$I^2 = 75.9\%$	95% CI [-0.91, -0.49]	Egger's Test = $p < .399$ No publication bias	Moderate

Note. The assessment of certainty was inspired by the GRADE framework (Balshem et al., 2011). A standard GRADE assessment was not appropriate due to the non-intervention/non-clinical nature of this review. Note that the 'Study Limitations' section overlaps between child and adult studies for prevalence and cognitive performance (i.e., the same study limitations apply to both child and adult studies addressing each research question).

2.5. Discussion

2.5.1. Prevalence of Co-Occurring ADHD and RD

The first part of this systematic review aimed to explore the prevalence rates of co-occurring ADHD+RD across development. Results of the proportional meta-analysis revealed that the overall co-occurrence of ADHD+RD across the lifespan was estimated at around 23% (ranging from 20-27%). The prevalence estimates were identical for children and adults (both at 23%), with child estimates ranging from 20% to 27%, whilst adult estimates showed more variation (ranging from 12-40%). These results are in line with previous literature suggesting that around 25-40% of individuals with ADHD or RD will also meet criteria for the other condition (e.g., DuPaul et al., 2013).

Further, results of subgroup analyses revealed that the co-occurrence estimates in studies using ‘mixed’ or broader sampling methods were around 7%, whilst those employing ‘purposive’ sampling have reported significantly higher rates of co-occurring ADHD+RD (approximately 26%). The significantly lower co-occurrence rates in studies employing broader sampling methods are in line with recent literature suggesting that individuals with cognitive profiles which do not directly align with traditional diagnostic criteria may be less likely to get identified in both research and clinical settings (Astle et al., 2022). These results also support the notion that studies which only include participants based on the presence of specific characteristics that meet strict diagnostic thresholds may result in a loss of important information regarding the true prevalence or complexity of cognitive presentations associated with neurodiverse conditions (Astle et al., 2022). Nonetheless, it is also possible that individuals in broader samples simply do not meet criteria for both conditions due to a lack of meaningful co-occurrence. Thus, the higher rates in purposive samples may reflect more severe or specific cases that are not representative of the general population captured by broader samples.

Moreover, the prevalence estimates of co-occurring ADHD+RD ranged from 18-26% between different RD definitions, with studies employing the ‘low achievement’ model to define RD demonstrating the highest pooled proportions, and studies with an unclear (or not widely used) definition showing the lowest rates. Despite the variations in estimates, no statistically significant differences were found. These results are surprising, given that the agreement

between the IQ-achievement discrepancy definition and the low achievement definition has previously been found to be as low as 32% (Brown Waesche et al., 2011). Therefore, based on previous literature, the two main definitions of RD would be expected to identify a significantly different number of individuals with ADHD+RD. However, it has recently been suggested that neither the discrepancy, nor the low achievement definition, provide an accurate method for the identification of individuals with reading difficulties (Wagner et al. 2020). This is because individuals with RD can be found across the reading continuum, and cannot simply be characterised by poor or discrepant reading performance (Wagner et al. 2020). Instead, it has been proposed that ‘hybrid’ models, which incorporate different criteria for defining RD (e.g., genetic or environmental influences, and cognitive correlates of dyslexia) may be a more accurate approach for identifying the condition (Fletcher et al., 2019; Wagner et al., 2020). To this end, none of the included studies adopted such an approach, suggesting that in order to expand on the current findings, future research may explore such hybrid models to increase the accuracy of co-occurrence estimates.

This uncertainty in the estimated co-occurrence rates is further exacerbated by the significantly high levels of heterogeneity observed for each of the abovementioned syntheses (all I^2 values above 90%). The main sources of uncertainty for the majority of studies addressing RQ1 were sampling procedures and methods used to identify and/or confirm ADHD+RD. These limitations are in line with the abovementioned issues associated with the current state of neurodevelopmental research, in regards to sampling and identification methods (e.g., Astle et al., 2022). Further, it is important to note that there were very few epidemiological studies that directly assessed the prevalence of co-occurring ADHD+RD, with the majority of included studies simply reporting the proportion of participants with the combined condition for sample characterisation purposes. This may have contributed to the observed high levels of heterogeneity between studies, given the variation in sampling frames and sample sizes. Consequently, this raises further questions around the precision of the reported estimates, and therefore, the findings of this review should be interpreted with caution.

Although the leave-one-out sensitivity analysis of the overall co-occurrence estimates of ADHD+RD revealed that the findings were robust to the exclusion of individual studies, there was one study for both child (Kita et al., 2020) and adult (Cederlof et al., 2017) subgroup analyses that seemed to affect the results. Whilst Kita et al. (2020) was also found to influence the ‘mixed sampling’ and ‘unclear/other RD definition’ subgroup results, it seemed to have

minimal impact on these analyses as the results remained relatively stable despite its removal. Upon closer inspection, this study displayed relatively low risk of bias across domains, but may have been an outlier due to its large community sample size in comparison with the other smaller-scale studies included in the review. Similarly, the study by Cederlof et al. (2017) showed minimal risk of bias across domains, but was based on data from a Swedish registry, and therefore, the sample size was significantly larger than that of other adult studies included in the review. This may explain why the removal of this study may have disproportionately affected the results, specifically the relatively wide confidence intervals.

Further, one study (Goker et al., 2014) in the ‘purposive sampling’ subgroup was flagged as influential, but did not seem to disproportionately impact the results of the sampling subgroup analyses. This study seemed to demonstrate some uncertainties across several domains, specifically potential bias regarding convenience sampling procedures (i.e., all children were recruited from a single clinic), as well as methods used to identify and/or confirm ADHD and RD. These study limitations further emphasise the need for improved sampling and identification procedures in neurodiversity research. Similarly, Stevenson et al. (2005) was found to be an influential study in the ‘discrepancy’ RD definition subgroup, but it did not substantially destabilise the results. Upon closer inspection, this study displayed some concerns in several risk of bias domains, including unclear sampling methods, small sample size, and appropriateness of statistical analyses for estimating co-occurrence rates.

Additionally, two studies – Frances et al. (2023) and Yoshimasu et al., (2010) – were found to be particularly influential in the ‘low achievement’ and ‘discrepancy and low achievement’ RD definition subgroup analyses. The study by Frances et al. (2023) exerted minimal influence as the results remained relatively robust despite its removal. This study showed minimal risk of bias across most domains, although participants were consecutively recruited from a registry until the desired sample size was reached, which does not entirely control for bias. On the other hand, it is possible that the removal of Yoshimasu et al. (2020) substantially contributed to changes in between-study variance, with heterogeneity levels fluctuating between 85-96% (I^2). Despite these fluctuations, this study demonstrated minimal risk of bias across several domains, with some concerns regarding the methods used to confirm ADHD+RD. It is possible that this study may have also been an outlier due to its large sample size compared to the smaller-scale studies included in this review.

Finally, results of publication bias analyses indicated that child studies demonstrated a minor risk of reporting bias, whilst the risk was significantly high for adult studies. In light of these uncertainties, it is important to highlight that there were only a limited number of eligible adult studies with available proportional data (precisely 8 studies were included). This does not only highlight the current lack of research into neurodiversity in adulthood, but also suggests that available data may not be reliable enough to accurately estimate the co-occurrence rates of ADHD+RD, especially in adults. Therefore, while the results of the proportional meta-analysis should be interpreted with caution, the findings appear to be an accurate reflection of the current uncertainty regarding the true prevalence of ADHD+RD across development.

2.5.2. Cognitive Manifestations of Co-Occurring ADHD and RD

The second part of this systematic review aimed to explore the cognitive manifestations of co-occurring ADHD+RD across development. The results revealed that the pooled effect estimate of the overall cognitive performance was -1.11 for children, and -0.70 for adults, with a significantly strong negative effect (compared to control data). These results suggest that both children and adults show substantial weaknesses in cognitive function, and are in line with previous research suggesting that individuals with ADHD+RD experience severe cognitive difficulties and display complex cognitive profiles (e.g., Mayes et al., 2000; Wang & Chung, 2018).

Although overall child and adult cognitive performance were not statistically compared due to the unavailability of adult data for the general memory domain, the findings suggest that children may demonstrate more difficulties in overall cognitive function compared to adults. This supports the notion that the characteristics of ADHD and RD may decline with age (Faraone et al., 2006; MacCullagh et al., 2017). However, while this may be due to strategies that have been developed throughout the lifespan to compensate for areas of difficulty in cognitive function, it is important to note that these difficulties likely persist into adulthood, but may present differently (van der Putten et al., 2024; Asherson & Agnew-Blais, 2019; Reis et al., 2020). This raises potential questions around the appropriateness of traditional cognitive measures in assessing the cognitive functioning of adults with ADHD and/or RD. Much of the current literature has attempted to pinpoint a ‘core cognitive deficit’ underpinning ADHD and/or RD. For instance, the idea that the co-occurrence of ADHD and RD may be attributed to a ‘shared difficulty’ in processing speed or working memory (McGrath et al., 2011; Tiffin-

Richards et al., 2008), or that ADHD+RD may simply present as a combination of the ‘core’ attentional control and phonological processing difficulties associated with ADHD and RD alone (e.g., Barkley, 1997; Lyon et al., 2003; Willcutt et al., 2001). Through the use of a restrictive range of ‘gold standard’ measures which simply assess the cognitive domains that are frequently associated with ADHD and/or RD, it may be difficult to identify potential mechanisms through which the conditions may present differently across development. It has recently been suggested that the use of more comprehensive cognitive assessments that go beyond the ‘core deficits’ and assess areas of functioning that are not typically associated with a given condition, may allow for a deeper understanding of the complexities associated with neurodiversity (Astle & Fletcher-Watson, 2020). This is especially important for the assessment of cognition in adults, given that the cognitive presentations of co-occurring ADHD+RD in adulthood may be even more likely to deviate from what is considered the ‘norm’ based on current diagnostic criteria.

This systematic review focused on synthesising evidence across 9 cognitive domains (attention, cognitive flexibility, inhibition, planning, problem-solving, processing speed, vocabulary, working memory, and memory), with the aim to gain a more comprehensive understanding of the complex cognitive profiles associated with ADHD+RD. Child and adult results were compared on domains for which both data were available (i.e., all apart from memory). Although children displayed slightly higher negative effect sizes across all domains, no significant differences were found between the two age groups. These results suggest that there may in fact be no substantial differences in the cognitive presentations of ADHD+RD across development. These findings are not in line with current literature on the deteriorating course of difficulties associated with ADHD (e.g., Biederman & Faraone, 2000), but do align with research suggesting that the underlying cognitive differences associated with RD remain constant across the lifespan (Reis et al., 2020). It is possible that since the co-occurrence of ADHD+RD has been associated with more complex cognitive profiles (Wang & Chung, 2018), the interaction between the two conditions may make it more challenging for the difficulties to subside or develop into more adaptive functioning with age. However, it should be noted that the lack of observed differences between child and adult cognitive presentations may have been due to insufficient power for detecting such differences, resulting from the small number of eligible adult studies. Therefore, the results of this analysis should be interpreted with caution.

Further, it should also be considered that both children and adults with ADHD and/or RD may show relative strengths in several areas of cognition given the potential overreliance on specific cognitive abilities to compensate for areas of difficulty (e.g., Hobson & Lee, 2023; van der Putten et al., 2024). This is important in light of the current review's aims to explore the cognitive manifestations of ADHD+RD, with the majority (if not all) of the included literature focusing on assessing cognitive difficulties, without considering the relative strengths that may also be associated with the two conditions. As a result, it appears that current research does not provide a comprehensive account of ADHD and/or RD, since this would also involve understanding potential strengths, not just weaknesses. This issue has previously been raised in the area of mental health and behavioural presentations of ADHD. Specifically, it has been highlighted that the majority of studies have traditionally focused on assessing the 'atypical' end of the spectrum only, through employing measures which do not have the capacity to identify factors related to resiliency in clinical populations (Alexander et al., 2020). A similar issue applies in the area of cognition, with an evident lack of dimensional measures with the capacity to capture cognitive abilities on a broader continuum, encompassing both strengths and weaknesses in a given domain (Astle et al., 2022). As a result, not assessing the relative strengths associated with ADHD and/or RD limits the ability to inform interventions of potentially effective strategies that could harness individual strengths to support areas of difficulty (Astle et al., 2022).

It should be noted that substantially high levels of heterogeneity were observed for both child and adult studies (I^2 above the 75% threshold for both syntheses). Although results of the meta-regression indicated that attention significantly decreased the pooled effect size estimate in child studies, differences in cognitive domain did not account for any of the observed heterogeneity in child or adult studies. Whilst high variability may be characteristic of the complexity associated with the diverse cognitive profiles of both ADHD and RD (Vos et al., 2022; Chalmepe & Vlachos, 2025), such high levels of between-study heterogeneity raise questions around the certainty in the body of evidence included in the review. A potential source of uncertainty could be concerns regarding the methods used to identify ADHD+RD in the included studies, although the majority of studies demonstrated relatively low risk of bias across most domains. Such concerns were also evident in studies addressing the first part of the review, and are in line with the well-known limitations of neurodiversity research in regards to the appropriateness of current sampling and identification procedures (Astle et al., 2022).

Additionally, substantial publication bias was identified among child studies. This may be related to the abovementioned criticism that many studies often fail to assess relative strengths in cognitive functioning in neurodiverse populations. Similarly, it is possible that studies which have failed to identify any significant differences in cognitive performance between ‘cases’ and ‘controls’ often do not report such results, or that these results simply do not get published. This creates uncertainty around the precision of effect estimates in the current body of evidence, as it is possible that the effect sizes may be inflated due to reporting bias or missing results. Given that this systematic review was not focused on interventions or randomised controlled trials, protocols were not available for the included studies, and thus it was not possible to examine reporting bias from this perspective. Therefore, the results of this synthesis should be interpreted with caution.

Despite these uncertainties, the leave-one-out sensitivity analysis revealed that the results remained stable across individual study exclusions for both child and adult analyses. The removal of one study (Kibby et al., 2021) was found to result in a slightly higher effect size compared to the pooled effect estimate in children, but this study had minimal impact on the overall results. Upon closer inspection, this study demonstrated minimal risk of bias across all assessed domains. Additionally, while the results of the child meta-regression were relatively robust, the adult meta-regression was not as stable, with the removal of individual studies changing the direction of the effect, fairly wide confidence intervals, and heterogeneity levels fluctuating from significant to non-significant. These results were as expected given the small number of adult studies included in this synthesis (specifically 6), with research suggesting that there should be at least 10 studies for every factor modelled to prevent misleading results due to insufficient data (Higgins et al., 2024). Therefore, the lack of eligible adult studies has substantially contributed to the uncertainty in the body of evidence addressing RQ2.

Finally, one adult study was flagged as particularly influential (Miranda et al., 2017), with the main methodological limitation of this study being that cognition was not assessed directly via cognitive tasks, but through a standardised self and informant report assessment (specifically the Behaviour Rating Inventory of Executive Function; BRIEF; Gioia et al., 2000). Whilst the extraction of data from direct cognitive tasks was prioritised over indirect assessments of cognition in this review, this study was included despite the unavailability of performance-based data. This is because the BRIEF is a validated and well-established rating scale which has been shown to effectively identify children with ADHD (Davidson et al., 2016). However,

research suggests that there is a dissociation between the BRIEF and direct assessments of cognition, potentially as a result of performance-based tasks assessing the underlying cognitive construct, while informant reports measure the application of cognitive skills in different settings (McAuley et al., 2010). Therefore, despite the standardisation of extracted data, it is possible that the inclusion of both performance-based and questionnaire-based cognitive data may have substantially contributed to the high heterogeneity levels observed between studies.

2.5.3. Strengths and Limitations

2.5.3.1. Strengths

This review has several strengths. First, 50% of full text records were independently screened by a second reviewer, who also critically appraised approximately 33% of the included studies. This is in line with research suggesting that multiple-reviewer full text screening is recommended in order to reduce risk of bias (Stoll et al., 2019). To this end, there was a high level of consistency between the two reviewers, with above 80% agreement across different stages of the review, which has been proposed as the minimum acceptable level of interrater agreement (McHugh, 2012). Thus, a methodological strength of this review was that all efforts were made to reduce bias through independent screening and appraisal, increasing the reliability of the review process.

Another strength of this review was the transparent and systematic approach to addressing and handling missing data. When control data were unavailable, published normative data were used as a substitute with clearly documented assumptions to maintain transparency. Where sample size information was available, Hedges' g was used to compute standardised effect sizes as it provides a more accurate estimate by correcting for small sample bias. In cases where sample size data for the normative comparison group were unavailable, Cohen's d was calculated instead, as it allowed for the estimation of effect sizes without requiring control group sample sizes for standard error calculations. This approach ensured that standardised effect sizes could be computed even when the exact sample size was unknown for normative data. This approach ensured that meaningful and comparable effect sizes could still be included in the meta-analysis despite missing data, thus maximising the available evidence base and overall representativeness of the review.

Further, leave-one-out sensitivity analyses were conducted for both the meta-analysis of proportions and standardised mean differences (including subgroup analyses). This approach allowed for the identification of potential outliers or highly influential studies, and provided a robust assessment of the stability and reliability of the results. Additionally, this systematic review applied a novel approach to assessing publication bias. Specifically, Doi plots and the LFK index were used in the meta-analysis of proportions, in line with recent recommendations suggesting that these methods offer greater accuracy than traditional procedures for evaluating publication bias in meta-analyses of proportions (Furuya-Kanamori et al., 2018; Cheema et al., 2022). This represents an improvement over previous systematic reviews, which have typically relied on conventional funnel plots and Egger's test, despite these methods being shown to perform poorly when applied to proportional data (Barker et al., 2021). Thus, the interpretability of the proportional meta-analysis results was enhanced through the use of appropriate publication bias detection tools.

Another strength of this systematic review is that it assessed the impact of sampling methods on co-occurrence estimates. This is important since recruitment methods have been suggested to result in a loss of important information regarding the true prevalence or complexity of cognitive presentations associated with neurodiverse conditions (Astle et al., 2022). This review therefore allowed for an exploration of the ways in which different sampling strategies (e.g., mixed or purposive sampling) may influence reported rates of co-occurrence. By accounting for these methodological differences through subgroup analyses, the present review provided a more accurate understanding of co-occurrence estimates, and further underscored the importance of recruitment strategies when interpreting findings in neurodiversity research.

Finally, a core strength of this review is its comprehensive approach to evidence synthesis, through gathering evidence across a broad range of cognitive domains. This is in line with the emerging dimensional approaches to neurodiversity, where broader assessments of cognition are recommended to capture a range of dimensions relevant to individual wellbeing across diagnostic categories (e.g., Insel et al., 2010; Kotov et al., 2017; Astle et al., 2022). By encompassing multiple cognitive domains, this review has contributed to a more comprehensive understanding of the cognitive profiles associated with ADHD+RD, and supported a shift towards a transdiagnostic dimensional approach in evidence synthesis. Through adopting this approach, the present review was able to highlight gaps in current ADHD+RD literature, for instance, the lack of research into several cognitive domains in the

adult population, thus providing potential objectives for future dimensional research within this field.

2.5.3.2. Limitations

There are several limitations concerning the processes of this review. First, the screening of titles and abstracts was conducted by the first author only due to the unavailability of a second reviewer for this stage of review. This may have introduced bias, as it has recently been shown that single-reviewer abstract screening may miss up to 13% of relevant studies (Gartlehner et al., 2020). Similarly, all quantitative data were extracted by the first author only, also due to the unavailability of a second reviewer. This may have introduced potential risk of error, as the data extraction process was complex given the nature of the data included in this review. Specifically, this process involved well-informed subject-specific decision making regarding the collation of relevant data; for instance, in cases where a single study reported multiple performance scores from different measures for a given cognitive domain, or in cases where different data were reported for multiple cognitive abilities relating to the same cognitive construct (see Methods section). Other complex decision-making involved the prioritisation of more widely used measures or larger effect sizes, where multiple studies by a given author employed the same sample and reported similar data. Although a second reviewer was not available for a direct review of title and abstract screening, or data extraction, all decisions were discussed with a senior researcher, and efforts were made to ensure accuracy and consistency (e.g., through maintaining detailed documentation of all decisions made).

Further, due to time constraints, study authors were not contacted to clarify any information. This may have resulted in the exclusion of potentially eligible studies. For instance, several studies with potentially relevant data were excluded if precise figures were difficult to extract (e.g., from graphs/bar charts). Additionally, since there were no language restrictions in the search process, several databases retrieved potentially eligible articles, which were not written in English. These records were excluded despite their potential eligibility due to limited resources in effectively translating such articles into English. Moreover, there were a total of 127 potentially eligible records which were inaccessible via institutional login. Although efforts were made to retrieve these papers through open access sources, alternative databases, and reference lists where the studies were previously cited, the records were excluded from the review. These studies may have had the capacity to provide important information regarding

the prevalence or cognitive manifestations of ADHD+RD, and their exclusion may have therefore affected the representativeness of the review findings, and/or increased the risk of publication bias and/or missing evidence. Therefore, future systematic reviews should ensure to contact study authors for additional information, or for access to articles which are not readily inaccessible via institutional login. Similarly, future studies may benefit from publishing articles in English as well as their home language, while future systematic reviews could benefit from additional resources to enable the translation of articles published in different languages.

Moreover, some of the planned analyses outlined in the pre-registered protocol were not carried out. Specifically, studies addressing RQ2 were not divided into subgroups based on measures used to assess cognitive ability in order to investigate the influence of methodology on identified differences between children and adults. This was not feasible for the following reasons: a) there was extremely high variability in measures used to assess different cognitive domains, b) some studies reported data from multiple cognitive measures assessing the same domain, which had to be averaged for the purposes of data syntheses, thus data could not be separated based on measures used. Further, it was not feasible to divide child and adult studies into subgroups based on clinical or subclinical presentations of ADHD+RD for the purposes of investigating differences in cognitive ability. This is because of the uncertainty in methods used to define the two conditions in a large proportion of the included studies. Therefore, future systematic reviews should focus on exploring the influence of methodology (i.e., cognitive measures used) on observed effect estimates, while future empirical studies may focus on more transparent reporting of methods used to confirm/define diagnoses, in order to enable for the comparison of cognitive functioning between clinical and subclinical presentations of ADHD+RD.

Additionally, the impact of methods used to identify ADHD on overall co-occurrence estimates was not investigated, although this may have contributed to the observed heterogeneity in the incidence of ADHD+RD reported across studies. This information was not included in the synthesis since the majority of studies employed DSM-5 diagnostic criteria to confirm ADHD. However, the risk of bias evaluation indicated that diagnostic assessment was identified as a moderate to high risk of bias domain, suggesting that the method used to confirm ADHD may not have been as clear or consistent across studies. Likewise, ADHD subtypes were not explored since the majority of studies included a combined-type sample. However, research

suggests that different ADHD subtypes may be characterised by differing patterns of cognitive function (e.g., Molavi et al., 2020), suggesting that differences in ADHD subtype may have affected the results in the present synthesis. Thus, the systematic review would have benefitted from additional subgroup analyses based on methods used to confirm ADHD, similar to the subgroup analyses performed to assess the impact of model used to define RD, in order to further investigate potential sources of heterogeneity and/or bias. Future reviews may benefit from distinguishing between ADHD subtypes to further explore how different presentations of ADHD may impact co-occurrence rates with RD, or the cognitive profiles of ADHD+RD.

Another limitation of this review is that a distinction was not made between children, adolescents, and adults. Although this review was concerned with how the co-occurrence of ADHD+RD and associated cognitive manifestations differ between children and adults, it would be worth exploring whether this may also differ for adolescents. For instance, research has shown that cognitive difficulties associated with ADHD may differ between children, adolescents and adults (Marx et al., 2010). It is possible that such differences may also contribute to differing cognitive profiles of co-occurring ADHD+RD across these age groups. Similarly, such differences in cognition may contribute to variable co-occurrence rates across the lifespan. Thus, future systematic reviews may benefit from performing subgroup analyses of child, adolescent, and adult data. This could potentially reveal whether adolescent data are more similar to that of children, or adults.

Finally, this systematic review did not distinguish between studies reporting the overall prevalence of ADHD+RD in a given population, and those reporting the prevalence of RD in individuals with ADHD (and vice versa). Separate meta-analyses for the proportion of ADHD in RD (and RD in ADHD) may have provided a more nuanced understanding of co-occurrence patterns, with important implications for clinical practice. However, the available data were often inconsistently reported across studies, limiting the ability to conduct separate syntheses. Therefore, future empirical studies and/or systematic reviews may focus on exploring bidirectional patterns of co-occurrence to provide further insights into the prevalence of ADHD+RD.

2.5.4. Implications for Practice and Future Research

The findings of this review have several important implications for both clinical practice and research. First, significantly lower levels of ADHD+RD were identified in studies employing broader sampling methods (e.g., struggling learners in schools or community samples), compared to those using purposive sampling strategies (e.g., clinical referrals or prior formal diagnoses). These findings emphasise the recently highlighted need for enhanced diagnostic sensitivity in identifying ADHD and RD in individuals whose cognitive profiles may not align with current diagnostic criteria, yet may still experience difficulties that warrant professional attention (Astle et al., 2022). This is important to consider in clinical practice as the failure to identify or diagnose individuals with milder difficulties and/or more complex cognitive profiles has a direct impact on the type of support they may be able to access.

Further, the results of this review also revealed that the co-occurrence of ADHD+RD remained consistently high across development. This suggests that although difficulties associated with ADHD+RD may be more pronounced and/or easier to identify in childhood, their co-occurrence remains high and clinically relevant in adulthood, and should therefore receive an equal amount of attention. This is especially important since current diagnostic criteria are based on childhood presentations of the two conditions (APA, 2013), which highlights the need for an evidence-based system for a more comprehensive assessment of ADHD+RD in adults. The findings also showed that both children and adults demonstrated significant difficulties in overall cognitive function, whilst no significant age group differences were found in any specific cognitive domain. This suggests that the same level of cognitive difficulty observed in children with ADHD+RD may persist into adulthood, which not only further emphasises the need for improved diagnostic assessment tools that are appropriate for use with adults, but also the need for support strategies that are well suited for all developmental stages.

This review did not identify any specific areas of cognition in which adult ADHD+RD may present differently to children. Since it is possible that this may be due to a lack of eligible adult studies resulting in insufficient power to detect significant differences, this review highlights the critical need for further research into the cognitive functioning of adults with ADHD+RD. Such research should focus on the comprehensive and dimensional assessment of both strengths and weaknesses across multiple cognitive domains, in order to yield empirical

evidence that allows for a more accurate identification of ADHD+RD across development, and provides potential targets for more effective support strategies.

Future systematic reviews should acknowledge and harness the methodological limitations of the current review. For instance, through identifying a limited set of widely used standardised assessments of specific cognitive domains, future systematic reviews could attempt to investigate the influence of methodology (i.e., assessments used) on pooled effect estimates, or any potential differences in the cognitive manifestations of children and adults with ADHD+RD. Similarly, future reviews may benefit from assessing ADHD subtypes or methods used to diagnose and/or confirm ADHD in order to better account for sources of heterogeneity or bias. Future reviews should also aim to distinguish more clearly between different co-occurrence estimates (i.e., the prevalence of RD among individuals with ADHD, and vice versa), in order to provide a more nuanced understanding of how these conditions co-occur. Additionally, future systematic reviews may focus on distinguishing the co-occurrence rates and cognitive manifestations of ADHD+RD between children, adolescents, and adults, in order to gain a deeper understanding of age-related differences. Finally, due to the lack of published prevalence/epidemiological studies investigating the co-occurrence of ADHD+RD at the time the database searches were performed (August 2023), the current review included any eligible studies with available information on the proportion of children or adults with co-occurring ADHD+RD (see Methods section). As the number of published prevalence studies increases with time, future systematic reviews could restrict their inclusion criteria to prevalence studies in order to increase the precision of pooled co-occurrence estimates.

2.5.5. Conclusion

In conclusion, this systematic review was one of the first to assess the co-occurrence of ADHD+RD across the lifespan, alongside a synthesis of cognitive functioning across a broad range of cognitive domains. The results of this systematic review suggested that the co-occurrence rates of ADHD+RD and the level of cognitive difficulty experienced by individuals with ADHD+RD do not differ substantially between children and adults. The findings also demonstrated the impact of sampling methods on co-occurrence estimates, and highlighted the high variability in reported co-occurrence rates and cognitive performance scores across studies. Importantly, this review underscored the need for further research into the co-occurrence of ADHD+RD in adults, and added to the emerging pool of transdiagnostic

literature on neurodiverse conditions. Finally, this systematic review highlighted several methodological considerations for future research, and has important implications for the assessment, diagnosis and support of ADHD+RD across the lifespan.

2.6. Other Information

2.6.1. Registration and Protocol

This systematic review has been pre-registered in the international prospective register of systematic reviews (PROSPERO) under the registration number CRD42023463062, and can be accessed using the following link:

<https://www.crd.york.ac.uk/PROSPERO/view/CRD42023463062>

The following amendments to the protocol were made at the full text screen stage:

- 1) The initial protocol focused on the co-occurrence of ADHD with both reading and maths difficulties. This was changed to focus on the co-occurrence of ADHD with RD only in order to make the review more specific and to make data synthesis more feasible.
- 2) The initial protocol included studies with any information on ADHD and RD (e.g., those where samples were grouped into separate ADHD and RD groups), with the aim to calculate the extent of cognitive overlap between the two conditions. This was amended to only include studies which assessed cognitive ability in samples with co-occurring ADHD+RD. This change was made to focus the analyses on differences in cognitive ability between children and adults with ADHD+RD, as opposed to attempting to calculate the extent of cognitive overlap between the two conditions from groups with single diagnoses, as this plan proved to be too complex for data synthesis/analysis.
- 3) The protocol was amended to include studies with standardised cognitive assessments only. This change was made with the aim to investigate the influence of methodology on reported cognitive performance scores. However, this proved to be impractical during the data synthesis stage since variability in measures used across studies remained too high, despite the exclusion of unstandardised assessments.

The following amendments to the protocol were made at the data analysis stage:

- 1) The initial protocol stated that means and standard deviations would be used as a common metric and converted into z -scores for data synthesis. Instead, means and standard deviations were converted into effect sizes to enable meta-analyses of standardised mean differences.
- 2) The initial protocol stated that differences in cognitive ability between children and adults would be compared using a Wilcoxon test because data were expected to be non-parametric. Instead, an independent samples t -test was used as data were parametric after log-transformation.
- 3) The initial protocol stated that a Chi-squared test would be used to determine any significant differences in prevalence percentages of co-occurring ADHD+RD between children and adults. Instead, a z -test was used as co-occurrence rates were estimated using proportions, and a z -test is well suited for statistically comparing proportions between two independent groups.

Chapter 3: Embracing Complexity in the Characterisation and Tracking of Neurodevelopment: A Transdiagnostic Protocol

3.1. Introduction

This chapter outlines the protocol of the current study, providing a detailed overview of the methods employed to build on the emerging quantitative framework in the field of neurodiversity research. Chapter 1 (narrative review) and Chapter 2 (systematic review) highlighted the need for further data-driven accounts of how the interplay between cognitive and mental health processes contributes to the complexity of neurodiverse conditions, with a particular focus on adults. The main objectives of the current study were to explore a) the ways in which neurodivergent adults differ on key cognitive and mental health dimensions, b) how individual profiles may best be represented in a multidimensional space of strengths and difficulties, c) whether data-driven clusters may be used to better characterise individual wellbeing compared to traditional diagnostic categories (i.e., whether they map directly onto diagnostic labels), and d) the ways in which transdiagnostic dimensional research frameworks may help to elucidate the mechanisms underlying the complexity of NDs in adults (in terms of between-diagnosis overlaps, individual variation, and co-occurring conditions).

The protocol of this study has been developed in light of the recent ‘transdiagnostic revolution’ in neurodiverse conditions, through which the shortcomings of standard categorical approaches have been highlighted (Astle et al., 2022). In order to address these shortcomings (described in Chapter 1) and to fulfil the aims of the current research (see above), this study adopted a combination of inclusive sampling methods, dimensional assessment measures, and novel analytical approaches. The rationale for the methodological choices is provided below (Sections 3.1.1 to 3.1.3), followed by a detailed overview of the methodology in Section 3.2.

3.1.1. Transdiagnostic Recruitment

In line with the transdiagnostic nature of this research, this study adopted a relaxed approach to recruitment, with the aim to include the full developmental spectrum of abilities (from ‘typical’ to ‘atypical’ functioning), as well as a broad range of neurodiverse conditions. This involved the inclusion of a) participants with formal (i.e., clinically diagnosed) neurodiverse conditions, b) individuals with suspected yet undiagnosed neurodiverse conditions (e.g., those

with milder difficulties that fail to meet diagnostic thresholds, or those who were awaiting diagnostic assessment), c) individuals with more complex profiles of co-occurring conditions (whether diagnosed or undiagnosed), and d) a small proportion of neurotypical individuals. This recruitment strategy enabled the investigation of cognitive and mental health processes across a wide range of neurodivergent profiles, creating the opportunity to capture between-condition overlaps and within-condition heterogeneity. It also allowed for the assessment of individuals with milder difficulties, who have the capacity to provide valuable information, yet are often excluded from ‘case-control’ studies, where participants are typically recruited based on the presence or absence of a specific diagnosis (Astle & Fletcher-Watson, 2020). The study sample is described in further detail in Section 3.2.1.

3.1.2. Dimensional Assessment

This study employed a combination of dimensional cognitive and mental health measures. Dimensional measures are broader assessments, which allow for the identification of both strengths and weaknesses across a variety of domains, offering a more comprehensive understanding of individual functioning (Astle et al., 2022). Such assessments generate continuous scales, along which individuals may be located, with the particular combination of strengths and weaknesses across multiple dimensions reflecting the nature and complexity of individual profiles (Astle et al., 2022). Since dimensional measures assess a broad range of processes, as opposed to attempting to pinpoint a ‘core deficit’ underpinning a given diagnosis, they are well suited for use with transdiagnostic samples (Astle & Fletcher-Watson, 2020). Similarly, given their continuous nature, dimensional measures offer the opportunity to reliably distinguish individuals from one another even in subclinical (i.e., less severe) parts of the population (Alexander et al., 2020), making them appropriate for use with more inclusive samples (e.g., individuals with milder, subthreshold difficulties). Consequently, dimensional assessments have the capacity to identify areas of need that may be common across diagnoses, or mechanisms that may contribute to within-diagnosis heterogeneity. Since this study employed a combination of broader assessments of both cognition and mental health, it offered the potential to explore how the interplay between cognitive and mental health dimensions may contribute to individual variation, or underpin the co-occurrence of mental health problems in neurodivergent adults, allowing for a more comprehensive and holistic understanding of individual wellbeing. The dimensional measures selected for this study are described in detail in Sections 3.2.3. and 3.2.4.

3.1.3. Exploratory Data Analysis

This study applied a variety of data reduction methods, with the aim to explore the underlying structure of the dataset, without confinement to any predefined grouping criteria (i.e., singular diagnostic categories). Data reduction methods are well suited for use with multivariate data yielded from heterogeneous samples and broader assessments, as they can be applied to generate simplified models of complex datasets (e.g., van de Velden et al., 2019; Borsboom et al., 2021). Such techniques may be used to identify dimensions from broader assessments that best define a transdiagnostic sample, or to identify naturally occurring subgroups through hypothesis-free data-driven grouping, based on shared variance across multiple measures (Astle et al., 2022). In this way, data reduction methods can generate more comprehensive data-driven accounts of neurodiverse conditions, which are not restricted to characteristics that would typically be associated with a specific diagnostic category (Astle et al., 2022). The two main data reduction techniques applied in this study included cluster analysis and network analysis, which are both described in further detail in Section 3.2.5. of this chapter, and discussed extensively in the individual empirical chapters that exploit these techniques (Chapters 4 and 5, respectively).

3.2. Methods

3.2.1. Participants

A cohort of 175 adults (aged 18-51, M_{age} 21.7), who had been referred for assessment and/or sought help from support services for suspected or previously diagnosed neurodiverse conditions, were recruited using convenience sampling. The sample size was established based on previous research suggesting that studies should aim for $n = 20\text{-}30$ observations per expected subgroup when conducting cluster analysis, as this provides sufficient power for detecting number of clusters and individual cluster membership (Dalmaijer et al., 2022). This study aimed to explore a total of 8 core variables (5 cognitive and 3 mental health domains) on which individuals may differ; thus, a sample of 160 to 240 participants was deemed desirable. Participants were predominantly recruited from Aston University, including both staff and students (undergraduate and postgraduate). The study was advertised via SONA (online portal for participant recruitment), recruitment posters (see Appendix A, Supplementary Materials 2),

academic talks, and mailouts to special interest groups. A smaller proportion of participants were recruited from external sources. To this end, the study was advertised by neurodiversity charities (including Embracing Complexity, Dyspraxia Foundation, UK Adult ADHD Network, ADHD Adult UK, and All Age Autism), research databases (the Cambridge Autism Research Database), local support groups (The Recovery College), and via social media (LinkedIn, X, Instagram and Facebook). All Aston University Psychology students participated in exchange for course credit and a £10 Amazon voucher, whilst any external participants and Aston University staff were reimbursed with a £20 Amazon voucher for their participation.

The exclusion criteria were as follows:

- a) Non-native English speakers (English did not exclusively have to be their first language).
- b) Persons with a history of traumatic brain injury with long-term effects on mental functioning.
- c) Adults above 50 years of age.
- d) Adolescents below the age of 16 who could not provide independent informed consent.
- e) Adults with reduced capacity to provide informed consent on their own behalf (e.g., due to pervasive intellectual disability).
- f) Individuals with uncorrected primary sensory impairments (e.g., hearing loss).

These exclusionary criteria were put in place as the abovementioned factors have the capacity to affect performance on the measures administered. The characteristics and diagnostic status of the sample are presented in Tables 1-2.

Table 1. Demographic characteristics of the sample ($n = 175$).

	<i>n</i>	%
Sex		
Male	36	20.6
Female	139	79.4
Race		
Caucasian	49	28.0
Asian	81	46.3
African/Caribbean	35	20.0
Mixed Heritage	10	5.7
First Language		
English	143	81.7
Other	32	18.3
Current/Completed Education Level		
Secondary School	2	1.1
Sixth Form/College	2	1.1
Undergraduate Degree	152	86.9
Postgraduate Degree	19	10.9
Referred for Difficulties		
No	98	56.0
Yes (Type of Difficulty):	77	44.0
Attention	46	59.7
Memory	18	23.4
Learning	22	28.6
Reading	25	32.5
Writing	1	1.3
Language/Speech	2	2.6
Visual Processing	2	2.6
Movement Co-Ordination	3	3.9
Anxiety/Depression/Stress	4	5.2
Impulsivity	1	1.3
Socialisation/Interaction	1	1.3
Referred By		
Self	36	46.8
Parents/Family Member/Friends	27	35.1
School/College	20	26.0
GP/Therapist/Psychiatrist	7	9.1
Not Specified	1	1.3
Referred To		
Education Services	34	44.2
Health Services	43	55.8
Local Support Services	4	5.2
Diagnostic Assessment	2	2.6
Private Support/Specialist	3	3.9
Not Specified	1	1.3

Note. In the ‘Difficulties With’ subsection, the majority of participants reported multiple difficulties. Therefore, *n* does not correspond to the total number of referred individuals. The percentages in this section indicate the number of participants who reported a given difficulty, from the total number of referred participants. In the ‘Referred By’ and ‘Referred To’ sections, some individuals reported being

referred by/to multiple sources. Therefore, *n* does not correspond to the total number of referred participants. Table 1 is continued below.

Table 1 Continued. Demographic characteristics of the sample (*n* = 175).

	<i>n</i>	%
Birth Complications		
No	163	93.1
Yes	12	6.9
Family History of Difficulties		
No	102	58.3
Yes (Type of Difficulty):	73	41.7
Neurodivergent	37	50.7
Mental Health Condition	39	53.4
Learning Difficulty	18	24.7
Not Specified	2	2.7
Difficulties Reported		
No	24	13.7
Yes (Type of Difficulty):	151	86.3
Communicating Needs	47	31.1
Understanding People's Behaviour	32	21.2
Functioning Independently	21	13.9
Self-Care	47	31.1
Understanding New Information	63	41.7
Co-ordinating Movements	19	12.6
Controlling Behaviour	29	19.2
Learning New Skills	27	17.9
Attention/Concentration	16	10.6
Organisation	4	2.6
Memory	8	5.3
Emotion Regulation	2	1.3
Overstimulation	5	3.3
Rigidity to Routine	2	1.3
Hyperactivity/Impulsivity	3	2.0
Reading	4	2.6
Writing	27	17.9
Maths	3	2.0

Note. In the “Type of Difficulty” subsection of “Family History of Difficulties”, *n* does not correspond to the total number of participants with a family history of difficulties, as most participants reported a history of multiple difficulties. The percentages indicate the number of individuals who reported a given difficulty out of the total number of participants with a family history of difficulties. The final “Difficulties Reported” section refers to the general difficulties reported by participants, for which they may or may not have been referred in the past. The majority of participants reported having experienced multiple difficulties. Therefore, in the “Type of Difficulty” subsection, *n* does not correspond to the total number of participants who reported having experienced difficulties. The percentages indicate the number of individuals who reported each type of difficulty, out of the participants who reported having experienced difficulties.

Table 2. Formal diagnostic status of the sample ($n = 175$).

	<i>n</i>	%
Formal ND	49	28.0
ADHD	21	42.9
Dyslexia	15	30.6
Autism	8	16.3
Dyspraxia	3	6.1
Dyscalculia	1	2.0
Dysgraphia	1	2.0
Intellectual Disability	3	6.1
Specific Language Impairment	2	4.1
Tourette Syndrome	1	2.0
Learning Difficulty (Not Specified)	3	6.1
Perceptual Processing Difficulty	1	2.0
Co-occurring Conditions	24	49.0
Homotypic	5	20.8
Heterotypic	17	70.8
Homotypic and Heterotypic	2	8.3
Primary Formal ND	49	28.0
ADHD	20	40.8
Autism	7	14.3
Dyslexia	14	28.6
Dyspraxia	1	2.0
Intellectual Disability	3	6.1
Specific Language Impairment	1	2.0
Tourette Syndrome	1	2.0
Learning Difficulty (Not Specified)	1	2.0
Perceptual Processing Difficulty	1	2.0
Diagnosed By		
Educational Psychologist	14	28.6
Clinical Psychologist	9	18.4
Psychiatrist	9	18.4
Paediatrician	2	4.1
GP	5	10.2
Diagnostic Specialist	3	6.1
Not Specified	7	14.3
Age at Diagnosis of ND		
Under 18	22	44.9
18 or Over	20	40.8
Not Specified	7	14.3
Other Diagnoses (Without ND)	22	12.6
Anxiety	10	5.7
Depression	4	2.3
Anxiety and Depression	8	4.6
Suspected ND	108	61.7
No ND	18	10.3

Note. ND = Neurodiverse Diagnosis. ADHD = Attention Deficit Hyperactivity Disorder. In the 'Formal ND' section, the percentages indicate how many individuals out of those with a formal diagnostic label had received a given diagnosis. The *n* does not add up to the total number of participants with a formal ND, as co-occurring diagnoses were included (i.e., some participants reported more than one diagnosis).

The total percentage of ‘Co-occurring Conditions’ refers to the number of participants with a formal ND who had received an additional diagnosis, which was either ‘*homotypic*’ (within the same diagnostic category – i.e., another ND), or ‘*heterotypic*’ (from a different diagnostic category – e.g., a mental health condition). The corresponding percentages indicate the number of individuals with each type of co-occurring condition out of the total number of participants with an additional diagnosis.

Note that participants with multiple diagnostic labels were categorised into ‘**Primary Formal ND**’ groups based on the age at which they received their diagnoses. For instance, if an individual with co-occurring conditions received a diagnosis of ADHD first, this was regarded as their ‘primary’ formal diagnosis. The corresponding percentages indicate how many individuals out of the participants with a formal ND had received a given diagnosis. The percentages in the ‘Diagnosed By’ section indicate how many of those with a formal ND were diagnosed by each type of professional. The percentages in the ‘Other Diagnoses’ section were calculated from the number of participants out of the total sample who had received a formal diagnostic label other than ND. The ‘**Suspected ND**’ group consists of individuals who had reported one or more difficulties (see Table 1), been referred to support services and/or assessment (but never received a formal diagnosis), or are currently awaiting assessment. The ‘**No ND**’ group consists of participants who had not reported any difficulties, referrals or any ND. This group may include some individuals with a mental health condition (e.g., anxiety), who did not report any difficulties associated with NDs. The percentages in these sections indicate how many participants out of the total sample reported a suspected ND or no neurodiverse diagnosis.

3.2.2. Procedure

This study received ethical approval in the year 2022 by the Aston Research Ethics Committee (#HLS21066; see Appendix B, Supplementary Materials 2). All participants were required to provide informed consent (Appendices C-D, Supplementary Materials 2) and complete a short demographic questionnaire via Qualtrics prior to taking part in this study, in order to provide information about any difficulties experienced and/or prior referrals or diagnoses. All participants reconsented upon arrival on the day of their assessment to ensure that they were happy to participate, and to confirm that participant identity was being linked with corresponding data across all measures. All participants were debriefed upon study completion, and received a brief assessment report (Appendices E-F, Supplementary Materials 2). The cognitive and mental health batteries administered are described in the following sections.

3.2.3. Cognitive Assessment: NIH Toolbox Cognition Battery (Weintraub et al., 2013)

The National Institutes of Health (NIH) Toolbox Cognition Battery is a neuropsychological measure consisting of seven standardised assessments, tapping the overarching cognitive domains of **attention**, **executive function**, **memory**, **processing speed** and **language** (Weintraub et al., 2013). The battery is a computerised tool, administered to participants via an iPad, and takes approximately 36 minutes to complete. The NIH Toolbox was selected for this study due to its broad coverage of cognitive abilities, which have been shown to be relevant to a wide range of NDs (see Chapter 1). The specific cognitive functions assessed by each task are outlined in sections 3.2.3.1. to 3.2.3.4. Further, the battery has been validated for use with adults from a variety of ethnic and educational backgrounds, as well as both neurotypical and neurodivergent populations (Mungas et al., 2014; Beaumont et al., 2013; Jones et al., 2022), making it appropriate for use with more inclusive, heterogeneous samples. Additionally, the toolbox automatically generates norm-referenced continuous scores on each measure, capturing both strengths and weaknesses in cognitive functioning, making it well aligned with dimensional research frameworks. The following information has been retrieved from the NIH Toolbox Administrator's Manual and Weintraub and colleagues (2013). *All NIH Toolbox-related materials are ©2015 Northwestern University and the National Institutes of Health.*

3.2.3.1. Attention and Executive Function

The *Flanker Inhibitory Control and Attention Test* (Zelazo et al., 2014) is an adapted version of the Eriksen flanker task (Eriksen & Eriksen, 1974). The measure assesses the allocation of an individual's limited capacities to deal with environmental stimulation, through inhibiting visual attention to irrelevant task dimensions. Thus, the task assesses inhibitory control, a component of executive function. Each trial consists of a central directional target, which is flanked by similar stimuli on each side. The participant is required to indicate the direction of the central stimulus and ignore the direction to which the arrows are pointing. During congruent trials, the flankers and target face the same direction; during incongruent trials, the target and flankers point to opposite directions. Adult performance is assessed via reaction time (RT), where scores are yielded from 0 to 10. Overall, the measure consists of 40 trials, averaging around 4 minutes to complete.

The *Dimensional Change Card Sort Test* (Zelazo et al., 2014) is a measure of set shifting (i.e., cognitive flexibility), an executive function component. The test requires the participant to

match a visual stimulus to one of two choices of stimuli, according to shape or colour. Adults receive a mixed block of trials, in which colour is relevant in most trials, with occasional/unpredictable shifts to shape. The appropriate criterion word (i.e., 'colour' or 'shape') is presented on the screen, according to which participants must match the stimuli. There is a total of 40 trials which require a total of 4 minutes to complete. Performance is assessed via RT.

3.2.3.2. *Memory*

The *Picture Sequence Memory Test* (Dikmen et al., 2014) is a measure of episodic memory. The stimuli used in this measure are pictured objects/activities, which may be thematically related but have no set order. At the beginning of each trial, pictures are displayed in the centre of a computer screen and moved one at a time into a box (in a fixed order), while an audio describes the content of the picture (e.g., "Go for a hike"). This continues until the entire sequence of activities is presented on the screen. The pictures are then returned to the centre of the screen in a random display, at which point the participant is required to move them into the sequence previously demonstrated. The score is calculated from the cumulative number of adjacent picture pairs recalled correctly by the participant over three learning trials. Levels of task difficulty vary according to age group. For participants aged 9 to 60 years, 15 pictures are presented. This task takes approximately 10 minutes to complete.

The *List Sorting Working Memory Test* (Tulsky et al., 2014) is a measure of working memory. A series of stimuli are, one at a time, displayed on a computer screen both visually and orally. Participants must repeat the stimuli in size order (smallest to largest). In condition one, all stimuli presented come from one category. In the second condition, stimuli are presented from two categories. Here, the participant is required to first report all stimuli from one category, then from the other, in order of size within each. For example, if foods (category one) and animals (category two) are presented – e.g., 'elephant', 'pumpkin', 'strawberry' and 'duck' – the participant would respond with "strawberry, pumpkin, duck, elephant". The number of items in a series increases with trials, and the test must be discontinued if the participant fails two trials of the same length. The score is derived from the total items answered correctly across trials, as marked by the examiner. This measure takes approximately 7 minutes to administer.

3.2.3.3. *Processing Speed*

The *Pattern Comparison Processing Speed Test* (Carlozzi et al., 2014) assesses choice reaction time (i.e., processing speed). Participants must identify whether two visual patterns are the “same” or “not the same”. The type, complexity and number of stimuli vary depending on the age spectrum from 3 to 85 years, in order to ensure adequate variability of performance. Performance is assessed via calculating the score from the total number of correct items (of a possible 130) completed in 90 seconds. This task takes approximately 3 minutes to administer.

3.2.3.4. *Language*

The *Picture Vocabulary Test* (Gershon et al., 2014) is a measure of receptive vocabulary. Single words are presented via an audio recording, and simultaneously paired with four images of different objects, actions and/or depictions of concepts. Participants are required to select the picture that most closely matches the meaning of a given word. The test items are administered in a variable-length computer-adaptive test (CAT) format, reducing the time taken to identify individual performance levels. The test uses education level to determine which items will initially be presented. This means that some participants see fewer items than others, with the specific words presented depending on the participant’s age and performance. The majority of participants will see approximately 25 items and take up to 5 minutes to complete the task. Performance is assessed based on the percentage of correct responses.

The *Oral Reading Recognition Test* (Gershon et al., 2014) assesses reading decoding skills through testing the ability to accurately pronounce single words and/or recognise letters. The test operates on a bank of English items (words/letters), controlled for orthographic typicality, frequency of word use and complexity of letter-sound relationships. The items are administered using a CAT format, which determines the items that will be presented to each participant. The participant is required to read aloud each item that is presented to them one by one on a computer screen; these responses are then entered by the examiner, who indicates whether or not the response was correct. Performance is assessed based on the number of items answered correctly, and the measure takes approximately 4 minutes to administer.

3.2.3.5. Scoring of the NIH Toolbox

For the purposes of this study, age-corrected standard scores were extracted from the automatically generated NIH Toolbox output. This score compares individual performance to people of the same age within the NIH Toolbox normative sample (based on the population of the United States). A score of 100 indicates that the participant performed at the national average level for their age. Scores of 15 points above or below 100 indicate that the individual's performance was 1 standard deviation (SD) away from the average (Weintraub et al., 2013). The age-corrected standard scores were selected over uncorrected scores generated via the toolbox, as these have been shown to account for variance that is associated with non-clinical factors, providing a more accurate reflection of condition-related changes in cognitive function (Casaletto et al., 2015).

The following summary scores were also automatically generated via the NIH Toolbox: Cognitive Function Composite Score, Fluid Cognition Composite Score and Crystallised Cognition Composite Score. The Fluid Composite Score is calculated using the average of standard scores across the following measures: Flanker Test, Dimensional Change Card Sort Test, Picture Sequence Memory Test, List Sorting Working Memory Test and Pattern Comparison Processing Speed Test. It can be used as a global assessment of fluid cognitive functioning. The Crystallised Cognition Composite Score is calculated using the average of standard scores across the Picture Vocabulary Test and the Oral Reading Recognition Test, and can be interpreted as a global assessment of verbal cognition. The Cognitive Function Composite Score is derived from the average of the Fluid and Crystallised Composites, providing an overall assessment of general cognitive functioning. The summary scores were extracted for the purposes of providing overall sample descriptive statistics, but not included in subsequent analyses to prevent multicollinearity (see Chapter 4).

3.2.4. Mental Health Assessment

The following questionnaires have been selected to form the mental health battery part of the assessment. Each measure is dimensional in nature, assessing the broad spectrum of strengths and difficulties associated with the following correlates of mental health: **internalising**,

externalising and **emotion regulation**. All questionnaires were administered on paper and scored manually.

3.2.4.1. The Extended Strengths and Weaknesses Assessment of Normal Behaviour (E-SWAN; Alexander et al., 2020)

The E-SWAN (Appendix G, Supplementary Materials 2) is an extended version of the Strengths and Weaknesses Assessment of ADHD Symptoms and Normal Behaviour (SWAN; Swanson et al., 2012). It reconceptualises DSM-5 diagnostic criteria for specific conditions as abilities ranging from high to low (i.e., strengths and weaknesses), through converting each symptom into a behaviour. The E-SWAN scales used in the current study include measures assessing ADHD, Panic Disorder, Social Anxiety and Major Depressive Disorder (MDD). The Social Anxiety and MDD scales include items assessing internalising behaviour, while the ADHD and Panic Disorder scales assess externalising symptomatology. The E-SWAN generates bidirectional distributions, reflecting the dimensional nature of psychopathology. There is a strong correspondence between the E-SWAN scores and traditional scale scores at the pathological end for each trait, with good discrimination and reliability across the full latent trait (Alexander et al., 2020). The E-SWAN was originally developed for parent-report of child behaviour, but it has been demonstrated that the framework is also valid for adult self-report (Alexander et al., 2020). Thus, the original scales have been adapted for the purposes of this study. For example, item 18 of the ADHD scale has been adapted from “Enters into conversation and games without interrupting or intruding” to “I enter into conversation without interrupting or intruding”. The E-SWAN is a 7-point scale. For the purposes of this study, the E-SWAN was reverse scored from 7 – ‘Far Below Average’ to 1 – ‘Far Above Average’. A mean score was calculated for each subscale. A score of 4 or above represents above average weaknesses in each area assessed, i.e., a higher score is indicative of more difficulties experienced.

3.2.4.2. The Emotion Regulation Skills Questionnaire (ERSQ; Grant et al., 2018)

The ERSQ (Appendix H, Supplementary Materials 2) is an English version of a self-report measure assessing emotion regulation, originally developed and validated in German (Berking & Znoj, 2008). The questionnaire assesses 9 dimensions of emotion regulation, including awareness, sensations, clarity, understanding, acceptance, tolerance, readiness to confront, self-support and modification. Scores for each item/subscale are derived along a continuum, with

the total average of items reflecting successful emotion regulation skills. The ERSQ is consistent with established measures of emotion regulation, such as the Emotion Regulation Questionnaire (Gross & John, 2003), shows good to excellent internal reliability, and has been validated for use with both typically developing and clinical adult samples (Grant et al., 2018). The ERSQ is scored on a 5-point scale ranging from 0 – ‘Not At All’ to 4 – ‘Almost Always’. The questionnaire consists of 27 items. It is scored by calculating the mean score for each subscale, with each subscale being made up of 3 items. A higher score indicates higher emotion regulation skills.

3.2.4.3. The Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997)

The SDQ (Appendix I, Supplementary Materials 2) is a behavioural screening questionnaire validated for adult self-report, assessing emotional symptoms, conduct problems, hyperactivity/inattention, peer relationship problems and prosocial behaviour (Goodman, 1997). The SDQ assesses both strengths and difficulties, generating a total score for general difficulties, and internalising and externalising problems, respectively. The measure has good internal consistency, test-retest stability and inter-rater agreement (Goodman, 2001), and has the capacity to predict the onset of psychopathology over time (Goodman & Goodman, 2009). The SDQ has recently been found to be equally as effective in identifying existing mental health problems in children with developmental difficulties without a specific diagnosis, as it has been in typically developing children and those with formal diagnoses, demonstrating its transdiagnostic utility (Bryant et al., 2020).

The SDQ consists of 25 items. It is a 3-point scale ranging from 0 – ‘Not True’ to 2 – ‘Certainly True’ (with items 7, 11, 14, 21 and 25 being reverse scored). The Emotional Difficulties, Conduct Problems, Hyperactivity, Peer Problems and Prosocial subscales each consist of 5 items and are scored out of 10 (via calculating the sum). The Externalising scale is the sum of the Conduct Problems and Hyperactivity scales, while the Internalising scale is the sum of the Emotional and Peer Problems subscales. The Total Difficulties scale is the sum of all subscales except the Prosocial scale. The Impact score represents overall distress and impairment as a result of any difficulties experienced. A higher score on any subscale except the Prosocial scale represents a higher level of self-reported difficulties.

3.2.5. Statistical Analysis: An Overview

This study employed two complementary data reduction methods to address the research aims outlined in Section 3.1. The statistical analyses consisted of cluster analysis and network analysis. The following sections offer a brief overview of these techniques, with the full methodological details (including data pre-processing, specific software packages used, and robustness checks) being described in Chapters 4 and 5, respectively.

3.2.5.1. Cluster Analysis (Chapter 4)

A *k*-means cluster analysis was conducted to identify data-driven groups of individuals based on shared patterns in cognitive and mental health functioning. Given the multivariate nature of the mental health dataset, a principal component analysis (PCA) was initially performed to reduce highly correlated mental health variables into fewer, orthogonal components. The identified components were then entered into the cluster analysis, along with the seven cognitive variables assessed. A one-way analysis of variance (ANOVA) was performed to explore group differences in cognition and mental health between the identified data-driven clusters, followed by Games-Howell (Games & Howell, 1976) post-hoc comparisons. This test is robust to violations of normality assumptions, and thus suitable for use with mental health data generated via rating scales. All initial analyses were conducted in jamovi (Version 2.3.28; The jamovi project, 2023). Additionally, a linear discriminant analysis (LDA) was performed in SPSS (Version 29.0.2.0; IBM SPSS Statistics, 2023) to define the underlying structure of the dataset through characterising the processes (i.e., dimensions) that best differentiate between the identified clusters. Finally, the composition of the data-driven clusters was inspected to determine whether they corresponded to traditional diagnostic categories.

3.2.5.2. Network Analysis (Chapter 5)

The network analysis provided a complementary, variable-level perspective by exploring the associations between individual cognitive and mental health dimensions. This approach involved estimating and visualising a Gaussian Graphical Model (GGM) using partial correlations, and evaluating the structure, centrality, and stability of the resulting network (Epskamp et al., 2018). Two network models were compared (regularised and non-regularised) to enable a more nuanced understanding of the subtle interactions between cognitive and mental health dimensions. Community detection (using the Walktrap algorithm) and bridge centrality metrics were computed to explore cross-domain interactions and potential ‘bridging

mechanisms’ (Pons & Latapy, 2006; Jones et al., 2021). All network analyses were conducted in R Studio (Version 2024.12.1; R Core Team, 2024).

Together, these analyses allowed for a deeper understanding of a) the ways in which neurodivergent adults differ on key cognitive and mental health dimensions (via cluster analysis and ANOVA), b) how individual profiles can best be represented in a multidimensional space of strengths and difficulties (via LDA of data-driven clusters), c) whether data-driven clusters map directly onto traditional diagnostic categories (via inspection of individual cluster composition), and d) the potential mechanisms underlying within-diagnosis heterogeneity, between-diagnosis overlaps, and/or co-occurrence of conditions (via LDA of data-driven clusters, and network analysis).

3.2.5.3. Descriptive Statistics and Preliminary Analyses

As mentioned above, the primary analyses reported in Chapters 4 and 5 were based on clusters derived following dimensionality reduction using PCA. To aid interpretation and provide additional context, overall descriptive statistics for the sample (means and SDs of individual cognitive and mental health measures, including composite scores) are presented in Appendix A (Supplementary Materials 3). The appendix also includes descriptive statistics and one-way ANOVA results for group differences on cognitive and mental health measures across the broad diagnostic groupings presented in Table 2 (ND, Suspected ND, and No ND). Preliminary cluster analyses were also conducted prior to dimensionality reduction to illustrate the impact of PCA on the clustering solution. To this end, clusters were initially derived from cognitive data only, then from both cognitive and mental health data, in order to explore whether the inclusion of mental health variables would influence cluster structure and/or membership. Moreover, separate cluster analyses were conducted using data from the ND group only (i.e., those with formal diagnostic labels), with the aim to explore the extent to which data-driven clusters mapped directly onto formal diagnostic labels. Here, the clusters were first derived from cognitive data, then a combination of both cognitive and mental health data, with the aim to explore whether the optimal cluster solution and/or cluster membership of different diagnostic categories shifted as a result of interactions between cognitive and mental health dimensions. These exploratory analyses are also presented in Appendix A (Supplementary Materials 3). All analyses were conducted in jamovi (Version 2.3.28; The jamovi project, 2023), unless stated otherwise in the appendix.

3.3. Conclusion

This chapter outlined the transdiagnostic protocol employed to explore the interplay between cognitive and mental health processes in a neurodivergent adult sample. A dimensional framework was adopted to capture strengths and weaknesses across a broad range of cognitive and mental health dimensions, whilst data reduction techniques were applied to identify data-driven groups and explore complex between-variable relationships. Together, these methodological choices provided a robust foundation for investigating the mechanisms underpinning the complexity of neurodiverse conditions (e.g., within-diagnosis heterogeneity and between-diagnosis overlaps). The following chapters present the results of cluster analysis and network analysis, and explore the implications of the findings in the context of the study's aims.

Chapter 4: Cluster Analysis

This chapter aims to adopt a data-driven approach to characterising a transdiagnostic sample of neurodivergent adults, based on patterns of strengths and weaknesses in cognitive and mental health functioning. This chapter will employ a combination of multiple data reduction methods, including principal component analysis (PCA), cluster analysis, and linear discriminant analysis (LDA). The rationale for each technique, as well as the aims and hypothesis will be outlined in Section 4.1. The specific methods and results of each analysis will be reported in Sections 4.2. to 4.4. This will be followed by a discussion of the results in Section 4.5.

4.1. Introduction

4.1.1. Cluster Analysis

Cluster analysis is an unsupervised data reduction technique used to identify naturally occurring groups within a given dataset (Aldenderfer & Blashfield, 1984). It enables the discovery and characterisation of participant groups from multivariate data through capturing distinct patterns of correlation between large sets of variables that vary across groups of individuals (Kaufman & Rousseeuw, 2009). The clustering algorithm groups data into subsets based on similarity or dissimilarity, meaning the patterns of data that co-exist within a valid cluster are more similar to each other than they are to patterns belonging to different clusters within a dataset (Frades & Matthiesen, 2010). This allows for the exploration of variability across participants and measures, and the identification of variables that are the most significant differentiators between clusters.

In the process of clustering, data are not initially classified into groups based on predefined properties, which allows for any identified differences between groups to be completely data-driven (Astle et al., 2022). In this way, cluster analysis helps to elucidate how different cognitive and mental health variables may interact to form data-driven clusters, and how such processes may be associated with varying presentations of neurodiverse diagnoses (NDs). Importantly, empirically derived groups may or may not directly map onto existing diagnostic categories. To this end, data-driven transdiagnostic clusters may be compared with traditional diagnostic categories, offering the potential to detect inaccurate diagnostic labels, challenge supposed boundaries between patterns of difficulties, or identify novel ND subtypes (Astle et

al., 2022). Ultimately, clustering has the capacity to allow for a more accurate characterisation of individual strengths and weaknesses, above and beyond diagnostic status.

The application of such data-driven grouping methods has recently gained traction in the field of neurodiversity research. For example, Rivard and colleagues (2023) performed a cluster analysis to identify meaningful clinical profiles of children with suspected NDs that transcend diagnostic labels. The authors identified three clusters which significantly differed from one another, with only one of these groups aligning with a specific diagnostic category (i.e., autism). The other clusters were associated with a mix of diagnoses (e.g., ADHD, learning difficulties), and diverse difficulties (e.g., in intellectual and social functioning) that did not directly map onto specific diagnostic labels (Rivard et al., 2023). Similar results were observed by Mareva and colleagues (2024), who applied a *k*-means cluster analysis to explore data-driven profiles of executive function in a transdiagnostic sample of neurodivergent children. They identified three clusters with differing profiles, which were not directly related to diagnostic categories or to ‘core’ dimensions associated with specific diagnoses (Mareva et al., 2024). Additionally, Zdorovtsova and colleagues (2023) used cluster analysis to explore associations between inattention and hyperactivity behaviours and features of the structural brain connectome in a transdiagnostic sample of neurodivergent children. The results of this study revealed two clusters with invariant behavioural profiles (i.e., inattentive and hyperactive), which displayed significant differences in cognitive abilities (e.g., executive function). The authors suggested that inattention and hyperactivity may show overlaps across neurodiverse conditions because they can emerge through multiple trajectories of brain development (Zdorovtsova et al., 2023). Together, these findings demonstrate the insights that can be gained from data-driven grouping of transdiagnostic samples, in terms of exploring within-diagnostic heterogeneity, and the mechanisms that may underpin overlaps across NDs.

4.1.2. Principal Component Analysis (PCA)

Principal component analysis is often used in conjunction with clustering to aid in dimensionality reduction. PCA is an unsupervised data reduction technique which captures the maximum amount of variance within high-dimensional datasets through creating new uncorrelated variables (i.e., principal components), whilst minimising information loss (Jolliffe & Cadima, 2016; Greenacre et al., 2022). It is specifically useful for simplifying complex datasets in preparation for cluster analysis, as it highlights the key patterns between variables,

making it easier for the algorithm to efficiently identify meaningful clusters (e.g., Ding & He, 2004). Research has shown that removing irrelevant features (i.e., those with low variance) enables the filtering of statistical ‘noise’ and results in more robust clustering solutions (Ben-Hur & Guyon, 2003). Similarly, through creating a set of uncorrelated components, PCA directly addresses the issue of multicollinearity (high correlation between several variables), which can often distort clustering algorithms and lead to unstable and/or misleading groupings (Jolliffe, 2002). PCA has been effectively employed as a pre-processing step for cluster analysis in neurodiversity research. For instance, Zheng and colleagues (2020) performed a hierarchical cluster analysis on principal components to explore developmental and behavioural heterogeneity among autistic preschoolers. This approach enabled the identification of meaningful subgroups based on multiple measures of cognition, language, and adaptive/social skills (Zheng et al., 2020). These findings highlight the utility of PCA in extracting core dimensions from highly dimensional multivariate datasets, particularly for enhancing the reliability and interpretability of subsequent clustering solutions.

4.1.3. Linear Discriminant Analysis (LDA)

Linear discriminant analysis is another (supervised) machine learning method used for dimensionality reduction, which explores linear combinations of features that best separate classes of data (e.g., Zhao et al., 2024). Similarly to PCA, LDA can also be utilised in conjunction with cluster analysis; for instance, it can be used to define the underlying structure of a dataset, through characterising the dimensions (i.e., discriminant functions) that best differentiate between data-driven clusters (Méndez et al., 2002). This method has recently been adopted by Zhang and colleagues (2022), who explored transdiagnostic symptom subtypes across autism and ADHD. In their study, LDA was applied to identify the cognitive and behavioural processes that best characterised data-driven clusters, providing evidence for precise phenotyping of the two NDs (Zhang et al., 2022). These findings demonstrate the value of employing LDA in conjunction with cluster analysis for investigating heterogeneity in neurodivergent populations, and for providing a more nuanced characterisation of individual profiles.

4.1.4. Aims

This chapter aims to explore a) the ways in which neurodivergent adults may differ on key cognitive and mental health dimensions, b) how individual profiles may be best represented in a multidimensional space of strengths and difficulties, c) whether data-driven clusters may be more effective at characterising individual functioning compared to traditional diagnostic categories, and d) how transdiagnostic dimensional frameworks may help to elucidate the mechanisms underpinning individual variation, between-diagnosis overlaps, and/or the co-occurrence of conditions.

The combination of PCA, cluster analysis, and LDA is therefore well suited for addressing the aims of this chapter. First, PCA will be used as a pre-processing step to simplify the high-dimensional dataset, enhancing the stability and interpretability of subsequent analyses. Cluster analysis will then be employed to identify naturally occurring groups, enabling a data-driven examination of whether distinct cognitive and/or mental health profiles exist among a mixed sample of neurodivergent adults, beyond the boundaries of traditional diagnostic labels. This will address the chapter's aim to explore whether individual functioning can be better characterised by data-driven clusters rather than diagnostic categories. Finally, LDA will be used to interpret the resulting clusters by identifying the combinations of variables that best distinguish between them. This will allow for a deeper understanding of the strengths and difficulties along key cognitive and mental health dimensions that define each subgroup. Together, these analyses have the capacity to highlight the cognitive and/or mental health mechanisms that may underpin the complexity associated with NDs (in terms of heterogeneity, overlaps, and/or co-occurrence). For instance, if a cluster predominantly composed of individuals with diagnoses of ADHD and dyslexia is characterised by a particular pattern of cognitive and/or mental health strengths and difficulties, this could point to shared underlying processes that contribute to the co-occurrence of these conditions, or reveal overlapping areas of functioning across the two ND profiles. Similarly, if individuals with the same diagnostic label are distributed across multiple data-driven clusters – each defined by a distinct profile of cognitive and/or mental health functioning – this may indicate the presence of underlying mechanisms that contribute to individual variability within diagnostic categories.

Given the exploratory and data-driven nature of cluster analysis, there were no *a priori* hypotheses regarding the number or structure of potential clusters. The main aim of this

analysis was to uncover naturally occurring groups within the transdiagnostic dataset, without imposing predefined assumptions. Nonetheless, based on previous research, it was predicted that the data-driven groups would not reliably map onto the primary diagnostic labels present within the dataset.

4.2. Principal Component Analysis (PCA): Methods and Results

A principal component analysis was performed on the 19 mental health variables assessed, with the aim to reduce multivariate data to a set of dimensions, which together explain the largest amount of variance within the self-reported mental health measures. The variables entered into the PCA are presented in Table 1. Prior to the analysis, the Strengths and Difficulties Questionnaire (SDQ) and the Extended Strengths and Weaknesses Assessment of Normal Behaviour (E-SWAN) variables were rescaled such that low scores represented more difficulties (or less adaptive functioning) across all measures. Any mental health composite scores (i.e., the total score of the ERSQ, and the Total Difficulties, Externalising and Internalising subscales of the SDQ) were excluded from the analysis. Data were standardised across measures via converting all values to *z*-scores (referenced to the sample, not questionnaire population norms). The correlations between all variables assessed are presented in Table 1, Appendix B (Supplementary Materials 3).

Bartlett's Test of Sphericity was conducted to assess whether the correlation matrix of the mental health variables was significantly different from an identity matrix (i.e., whether the correlation coefficients between the variables were different from zero). The results of the test were significant ($\chi^2(171, N = 175) = 1563, p < .001$), indicating the variables significantly covaried and were therefore suitable for PCA. The PCA was conducted with no statistical rotation to maximise the amount of variance explained by each component. A minimum factor loading of 0.5 was applied to highlight variables with only moderate or strong correlations to a given component. The optimal number of components retained from the analysis was determined using parallel analysis, which suggested a 4-component solution. This was validated by the finding that the first four observed eigenvalues exceeded those generated from randomly simulated data, indicating that these components explained more variance than would be expected by chance. The variable loadings on each component are presented in Table 1. The threshold of variance explained for a component was set to 60%, as increasing this threshold resulted in the inclusion of components with factor loadings below 0.5 (i.e., no more

added value). The percentage of variance explained by each principal component is presented in Table 2. Together, the first four components explained 61.1% of the total variance, with PC1 and PC2 accounting for majority of the variance in the dataset (45.7%).

The component loadings indicated that PC1 was primarily associated with variables relating to emotion regulation difficulties, hyperactivity, impact of difficulties and specific mental health conditions, while PC2 displayed strong correlations with variables relating to emotion regulation skills. Additionally, PC3 was strongly associated with variables relating to antisocial behaviour ('Conduct Problems' and 'Prosocial Skills'), while PC4 was primarily related to ADHD and hyperactivity. It is important to note that there was an overlap in the loadings of several variables. For instance, 'Awareness', 'Clarity' and 'Understanding' were strongly correlated with PC1 and PC2, with 'Awareness' being more strongly related to PC2, and 'Clarity' and 'Understanding' having a stronger relationship with PC1. Further, 'Hyperactivity' and 'ADHD' were associated with both PC1 and PC4, with 'Hyperactivity' being more strongly correlated with PC4, and 'ADHD' showing a stronger association with PC1.

Since the variables 'Modification', 'Emotional Difficulties' and 'Depression' had the highest loadings onto PC1, this component was defined as the '*Emotional Difficulties*' dimension. Further, 'Sensations' and 'Clarity' had the highest loadings onto PC2; this component was described as the '*Emotional Clarity*' dimension. Additionally, 'Prosocial Skills' had the highest loading on PC3; this component was characterised as the '*Social Skills*' dimension. Finally, 'Hyperactivity' had the highest loading onto PC4; this component was labelled as the '*Hyperactive Behaviour*' dimension.

Table 1. The loadings of mental health variables onto principal components.

Measure	Component			
	1	2	3	4
ERSQ				
Awareness	0.528	0.550	-0.156	0.098
Sensations	0.498	0.611	-0.016	0.148
Clarity	0.613	0.582	-0.096	0.052
Understanding	0.627	0.541	-0.113	-0.061
Acceptance	0.593	0.061	-0.077	-0.348
Tolerance	0.520	0.060	-0.102	-0.494
Confrontation	0.604	0.071	0.083	-0.075
Self-Support	0.624	0.126	0.182	-0.159
Modification	0.740	-0.007	0.237	-0.203
SDQ				
Emotional Difficulties	0.702	-0.391	-0.048	-0.060
Conduct Problems	0.247	0.046	0.535	0.356
Hyperactivity	0.522	-0.133	-0.154	0.633
Peer Problems	0.378	-0.192	0.441	-0.195
Prosocial Skills	0.279	0.071	0.806	0.097
Impact	0.653	-0.428	0.142	0.120
E-SWAN				
ADHD	0.606	-0.151	-0.183	0.538
Depression	0.796	-0.331	-0.137	0.070
Panic Disorder	0.694	-0.321	-0.340	0.002
Social Anxiety	0.633	-0.368	-0.105	-0.243

Note. *ERSQ* = Emotion Regulation Skills Questionnaire. *SDQ* = Strengths and Difficulties Questionnaire. *E-SWAN* = Extended Strengths and Weaknesses Assessment of Normal Behaviour. *ADHD* = Attention Deficit Hyperactivity Disorder. Strong correlations (above 0.5) are highlighted in bold. These correlations were used to define each component. The component scores were derived from the full set of loadings, with variables contributing proportionally based on the strength of their loadings.

Table 2. Percentage of variance explained by each principal component.

Component	SS Loadings	% of Variance	Cumulative %
1	6.58	34.61	34.6
2	2.10	11.07	45.7
3	1.52	7.98	53.7
4	1.42	7.49	61.1

Note. *SS* = Sum of Squares. The *SS* loading is the sum of squared loadings for all variables contributing to a given component. This indicates how much of the variance in the original dataset is accounted for by that component, based on the contributions of each variable. An *SS* loading close to 1 indicates that the component explains a larger amount of variance in the data.

Note

PCA was also applied to cognitive variables, as well as a combination of mental health and cognitive variables. However, these principal components were not deemed appropriate for further analyses as retaining separate variables representing distinct cognitive processes allowed for more meaningful cluster separation. Please refer to Appendix B (Supplementary Materials 3) for a more detailed explanation. Appendix B also includes a comparison of PCA and exploratory factor analysis (EFA) models for dimension reduction, providing a justification for the use of PCA over EFA for this dataset. All PCA and EFA analyses were conducted in jamovi (Version 2.3.28; The jamovi project, 2023).

4.3. Cluster Analysis: Methods and Results

A *k*-means cluster analysis was conducted in jamovi (Version 2.3.28; The jamovi project, 2023) to investigate the structure of the dataset through identifying distinct data-driven groups based on patterns of strengths and weaknesses in cognitive and mental health functioning. The four identified mental health components (Emotional Difficulties, Emotional Clarity, Social Skills and Hyperactive Behaviour) were entered into the analysis (representing individual scores), along with seven cognitive variables, each represented by the NIH Toolbox task administered: 1) Flanker Inhibitory Control and Attention Test, 2) Dimensional Change Card Sort Test, 3) Picture Sequence Memory Test, 4) List Sorting Working Memory Test, 5) Pattern Comparison Processing Speed Test, 6) Picture Vocabulary Test, and 7) Oral Reading Recognition Test. Cognitive composite scores (i.e., the Fluid Cognition, Crystallised Cognition and Cognitive Function composites generated by the NIH Toolbox) were excluded from the analysis. This is because the inclusion of composite scores could result in the misleading increase of homogeneity between and/or within clusters due to the inherent correlations between individual measures and their respective composite variables. All data were standardised (i.e., converted to *z*-scores) when entered into the cluster analysis. All *z*-scores were referenced to the sample mean.

A three-cluster structure was suggested by the gap statistic (Tibshirani et al., 2001) and elbow method (Thorndike, 1953) as the optimal solution (see Figure 1, Appendix C, Supplementary Materials 3). The clusters consisted of 50, 73 and 52 participants, respectively (i.e., all individuals were placed in only one cluster). Table 3 shows that Cluster 2 captured the majority of individuals with a formal neurodiverse diagnosis (ND; 44.9% of all individuals with a formal

diagnosis), followed by Cluster 3 (36.7%) and Cluster 1 (18.4%). Individuals with a suspected (but unconfirmed) ND were also most often members of Cluster 2 (40.7% of all individuals with a suspected ND), followed by Cluster 1 (31.5%), and Cluster 3 (27.8%). Finally, individuals with no ND (i.e., no formal/suspected diagnosis) were equally represented in Cluster 1 and Cluster 2 (38.9% of all individuals with no ND, respectively), followed by Cluster 3 (22.2%). Further, Cluster 3 captured 65.0% of all participants with a primary diagnosis of ADHD, while 71.4% of all autistic individuals were members of Cluster 2. Interestingly, individuals with a primary diagnosis of dyslexia were almost evenly distributed across the three clusters (35.7% in Clusters 1 and 2, 28.6% in Cluster 3). The distributions of other, less prevalent formal diagnostic labels are presented in Table 3.

Table 3. Frequency of diagnostic groups and types of primary diagnoses within individual clusters derived from cognitive data and mental health principal components ($n = 175$).

	Cluster 1 ($n = 50$)	Cluster 2 ($n = 73$)	Cluster 3 ($n = 52$)
Group	n (%)	n (%)	n (%)
Diagnostic Group			
ND	9 (18.0)	22 (30.1)	18 (34.6)
Suspected ND	34 (68.0)	44 (60.3)	30 (57.7)
No ND	7 (14.0)	7 (9.6)	4 (7.7)
Type of Diagnosis			
n	9	22	18
ADHD	1 (11.1)	6 (27.3)	13 (72.2)
Autism	1 (11.1)	5 (22.7)	1 (5.6)
Dyslexia	5 (55.6)	5 (22.7)	4 (22.2)
Dyspraxia	-	1 (4.5)	-
ID	1 (11.1)	2 (9.1)	-
SLI	-	1 (4.5)	-
TS	-	1 (4.5)	-
PPD	1 (11.1)	-	-
SLD	-	1 (4.5)	-

Note. ND = Neurodiverse Diagnosis. ADHD = Attention Deficit Hyperactivity Disorder. ID = Intellectual Disability. SLI = Specific Language Impairment. TS = Tourette Syndrome. PPD = Perceptual Processing Difficulty. SLD = Specific Learning Difficulty (but not specified). The frequency of primary diagnosis type was calculated for individual clusters from the total number of participants with a formal ND found within each cluster.

The descriptive statistics (mean z -scores) across the cognitive and mental health variables are presented in Table 4. A one-way analysis of variance (ANOVA) was conducted to explore group differences between the three clusters on these variables. The results revealed significant group differences on the Emotional Difficulties, Emotional Clarity and Hyperactive Behaviour dimensions (see Table 4). Note that in the following results, a higher score on any variable

indicates more adaptive functioning (or fewer difficulties). To this end, although the original PCA component labels (e.g., ‘Emotional Difficulties’ and ‘Hyperactive Behaviour’) reflect the original variable names, they should be interpreted as representing positive functioning. For example, higher scores on the ‘Emotional Difficulties’ dimension indicate greater emotional wellbeing.

Post-hoc comparisons using the Games-Howell test (Games & Howell, 1976) indicated that Cluster 1 reported significantly fewer Emotional Difficulties on average than Cluster 2 ($t(106) = 3.90, p < .001$) and Cluster 3 ($t(99.6) = 3.90, p < .001$), with a mean difference of 0.69 and 0.73, respectively. Interestingly, Cluster 1 also displayed significantly lower Emotional Clarity scores than Cluster 2 ($t(116) = -2.98, p = .010$) and Cluster 3 ($t(91.9) = -3.00, p = .010$), with a mean difference of 0.48 and 0.59. Additionally, Cluster 1 reported significantly lower Hyperactive Behaviour than Cluster 3 ($t(93.6) = 3.89, p < .001$), with a mean difference of 0.75; similarly, Cluster 2 reported significantly fewer hyperactive symptoms than Cluster 3 ($t(98.8) = 2.67, p = .024$), with a mean difference of 0.51. Overall, Cluster 1 displayed low emotional difficulty, low emotional clarity, and low hyperactivity (relative to Clusters 2 and 3). Cluster 2 displayed high emotional difficulty, high emotional clarity, and moderate hyperactivity (relative to Clusters 1 and 3). Finally, Cluster 3 displayed high emotional difficulty, high emotional clarity, and high hyperactivity (relative to Clusters 1 and 2).

Further, a one-way ANOVA also revealed significant group differences on all of the cognitive variables. Post-hoc analyses using the Games-Howell test indicated that Cluster 1 scored significantly higher than Cluster 2 on the Flanker Inhibitory Control and Attention Test ($t(93) = 7.84, p < .001$), with a mean difference of 1.05; while Cluster 2 also displayed significantly lower scores than Cluster 3 on the same task ($t(85.5) = -9.57, p < .001$), with a mean difference of -1.43. Significant differences were also observed on the Dimensional Change Card Sort Test, with Cluster 1 scoring significantly higher than Cluster 2 ($t(88.1) = 12.5, p < .001$) and significantly lower than Cluster 3 ($t(100) = -2.44, p = .043$), with a mean difference of 1.41 and -0.33, respectively. Similarly, Cluster 2 displayed significantly lower scores than Cluster 3 on this task ($t(90.6) = -15.26, p < .001$), with a mean difference of -1.73. Further, Cluster 1 ($t(99.1) = -3.34, p = .003$) and Cluster 2 ($t(104.9) = -4.50, p < .001$) scored significantly lower than Cluster 3 on the Picture Sequence Memory Test, with a mean difference of -0.63 and -0.80, respectively. Post-hoc comparisons indicated similar results on the List Sorting Working Memory Test, with both Cluster 1 ($t(97.4) = -6.72, p < .001$) and Cluster 2 ($t(118.9) = -5.83, p$

< .001) displaying significantly lower scores than Cluster 3, with a mean difference of -1.14 and -0.91. Significant differences were also found on the Pattern Comparison Processing Speed Test, with Cluster 1 scoring significantly higher than Cluster 2 ($t(109) = 10.7, p < .001$), with a mean difference of 1.46, and Cluster 2 scoring significantly lower than Cluster 3 ($t(116) = -9.96, p < .001$), with a mean difference of -1.31. Additionally, Cluster 1 ($t(96.8) = -8.80, p < .001$) and Cluster 2 ($t(106.2) = -6.77, p < .001$) scored significantly lower than Cluster 3 on the Picture Vocabulary Test, with a mean difference of -1.41 and -1.07, respectively. Finally, Cluster 1 scored significantly lower than Cluster 2 ($t(99.9) = -2.97, p = .010$) and Cluster 3 ($t(93.9) = -8.71, p < .001$) on the Oral Reading Recognition Test, with a mean difference of -0.48 and -1.43, respectively. To this end, Cluster 2 also scored significantly lower than Cluster 3 ($t(118.2) = -6.70, p < .001$), with a mean difference of -0.95.

Summary of Cluster Characteristics

Based on the above results, the three clusters can be described as below (in comparison to one another). The differences in cluster profiles across the cognitive and mental health variables are visually presented in Figure 1. For reference, the primary cognitive domains assessed by each cognitive task are presented in Table 5.

Cluster 1

Mental Health: Low emotional difficulty, low emotional clarity, low hyperactivity.

Cognition: Moderate inhibitory control, moderate cognitive flexibility, weak episodic memory, weak working memory, strong processing speed, weak vocabulary, very weak reading skills.

Cluster 2

Mental Health: High emotional difficulty, high emotional clarity, moderate hyperactivity.

Cognition: Weak inhibitory control, weak cognitive flexibility, weak episodic memory, weak working memory, weak processing speed, weak vocabulary, weak reading skills.

Cluster 3

Mental Health: High emotional difficulty, high emotional clarity, high hyperactivity.

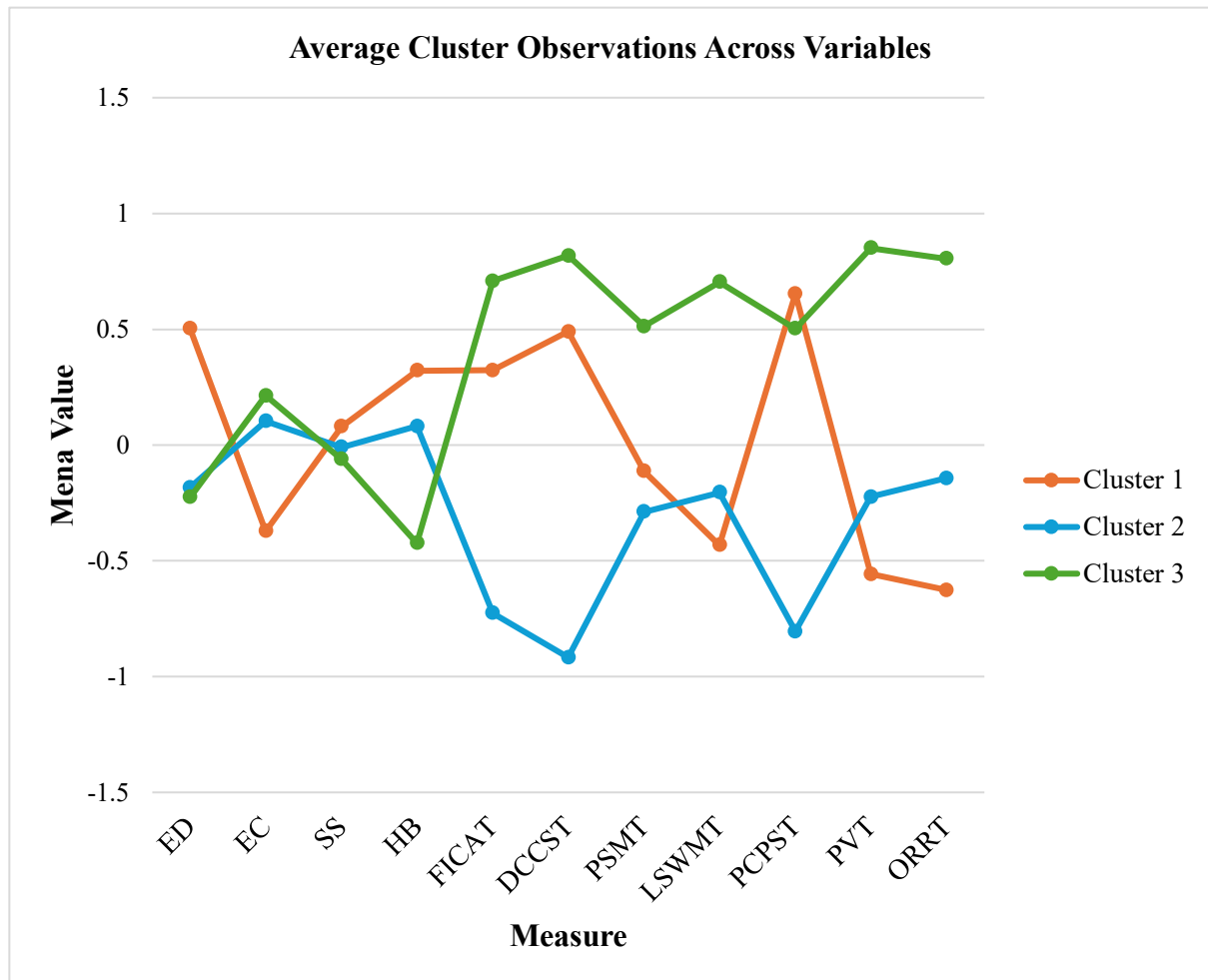
Cognition: Strong inhibitory control, strong cognitive flexibility, strong episodic memory, strong working memory, moderate processing speed, strong vocabulary, strong reading skills.

Table 4. Mean z-scores and one-way ANOVA results for group differences across cognitive tasks and mental health dimensions for clusters derived from cognitive variables and mental health principal components ($n = 175$).

	Cluster 1 ($n = 50$)	Cluster 2 ($n = 73$)	Cluster 3 ($n = 52$)			
Variable	$Z (SD)$	$Z (SD)$	$Z (SD)$	F	$df2$	p
Emotional Difficulties	0.50 (0.95)	-0.18 (0.97)	-0.23 (0.93)	9.75	108.0	<.001
Emotional Clarity	-0.37 (0.81)	0.10 (0.96)	0.21 (1.14)	6.27	107.0	.003
Social Skills	0.08 (1.00)	-0.01 (0.97)	-0.06 (1.05)	0.25	106.2	.779
Hyperactive Behaviour	0.32 (0.81)	0.08 (0.94)	-0.42 (1.11)	7.53	107.1	<.001
Flanker Inhibitory Control and Attention Test	0.32 (0.78)	-0.73 (0.65)	0.71 (0.93)	59.36	99.6	<.001
Dimensional Change Card Sort Test	0.49 (0.67)	-0.92 (0.52)	0.82 (0.69)	150.43	99.5	<.001
Picture Sequence Memory Test	-0.11 (0.88)	-0.29 (0.94)	0.51 (1.01)	10.42	107.4	<.001
List Sorting Working Memory Test	-0.43 (0.91)	-0.20 (0.94)	0.70 (0.80)	27.07	109.2	<.001
Pattern Comparison Processing Speed Test	0.65 (0.73)	-0.80 (0.76)	0.50 (0.69)	73.63	109.1	<.001
Picture Vocabulary Test	-0.56 (0.72)	-0.22 (0.85)	0.85 (0.89)	40.15	108.7	<.001
Oral Reading Recognition Test	-0.63 (0.91)	-0.14 (0.84)	0.80 (0.73)	42.70	107.4	<.001

Note. *PCA* = Principal Component Analysis. *Z* = Z-score. *SD* = Standard Deviation. *df* = degrees of freedom. Note that *df1* = 2 for all variables. The mental health dimensions of ‘Emotional Difficulty’, ‘Emotional Clarity’, ‘Social Skills’ and ‘Hyperactive Behaviour’ were derived from a PCA of 19 mental health variables, and are therefore a combination of data from the ERSQ, SDQ and E-SWAN questionnaires. Age-corrected standard scores were generated via the NIH Cognition Toolbox. A higher score is indicative of more adaptive functioning (or fewer difficulties) across all variables. Therefore, despite the label ‘Emotional Difficulties’, higher scores on this component indicate greater emotional wellbeing. The same applies to ‘Hyperactive Behaviour’, with higher scores indicating greater behavioural regulation, despite the negative direction of the component label.

Figure 1. A line graph showing average observations across cognitive and mental health variables for the three-cluster solution.



Note. *ED* = Emotional Difficulties. *EC* = Emotional Clarity. *SS* = Social Skills. *HB* = Hyperactive Behaviour. *FICAT* = Flanker Inhibitory Control and Attention Test. *DCCST* = Dimensional Change Card Sort Test. *PSMT* = Picture Sequence Memory Test. *LSWMT* = List Sorting Working Memory Test. *PCPST* = Pattern Comparison Processing Speed Test. *PVT* = Picture Vocabulary Test. *ORRT* = Oral Reading Recognition Test.

Table 5. Primary cognitive domains assessed by each cognitive task.

Task	Domains Assessed	Description
Flanker Inhibitory Control and Attention Test	Inhibitory Control	The ability to filter out irrelevant stimuli in the environment
Dimensional Change Card Sort Test	Cognitive Flexibility	The ability to efficiently switch attention in response to changing demands
Picture Sequence Memory Test	Episodic Memory	The ability to acquire, store and retrieve new information about personal experiences
List Sorting Working Memory Test	Working Memory	The ability to store information in the mind
Pattern Comparison Processing Speed Test	Processing Speed	The rate at which information can be received, processed and responded to
Picture Vocabulary Test	Vocabulary (Receptive Language)	The ability to understand the meaning of language
Oral Reading Recognition Test	Reading Decoding (Expressive Language)	The ability to translate written language into spoken words

Note. There is substantial overlap between the cognitive domains tapped by each of the above tasks as cognitive skills can rarely be assessed in isolation due to their interdependence. For example, both the Flanker Inhibitory Control and Attention Test and the Dimensional Change Card Sort Test measure executive function as they both measure different aspects of attention. However, each of the NIH Toolbox tasks have been designed to primarily tap a specific cognitive domain (displayed above with descriptions). Therefore, each of these tasks have been retained as separate variables representing each of the cognitive processes.

4.4. Linear Discriminant Analysis (LDA): Methods and Results

A linear discriminant analysis (LDA) was conducted in SPSS (Version 29.0.2.0; IBM SPSS Statistics, 2023) on the three clusters derived from the four mental health components and seven cognitive variables. This was performed with the aim to characterise the discriminant functions (i.e., dimensions) that best statistically differentiate between the data-driven clusters, and therefore help to define the underlying structure of the dataset. Since LDA is a parametric method and assumes that data are normally distributed, Box's M test was conducted to evaluate homogeneity of variance-covariance across clusters, given the heterogeneous nature of the sample. The result was not significant ($p = .547$), confirming that the assumption of equal variance-covariance was not violated.

The LDA revealed that the dataset was best defined by two discriminant functions, suggesting that the clusters were best represented in a two-dimensional space. Combined, the first two discriminant functions significantly distinguished between the three clusters ($\chi^2(22) = 358.48$, $p < .001$), with a Wilk's Lambda of .117. Additionally, the second discriminant function alone significantly contributed to the separation of the clusters ($\chi^2(10) = 138.85$, $p < .001$), with a Wilk's Lambda of .435. Function 1 explained 67.8% of the variance, while Function 2 explained the remaining 32.3% of the variance in the dataset.

The Structure Matrix (Table 6) revealed that the Dimensional Change Card Sort Test showed the strongest correlation with Function 1 ($r = .77$), followed by the Pattern Comparison Processing Speed Test ($r = .54$) and the Flanker Inhibitory Control and Attention Test ($r = .50$). Additionally, the Canonical Coefficients table (Table 7) indicated that the Dimensional Change Card Sort Test ($\beta = .70$, $p < .001$) and Pattern Comparison Processing Speed Test ($\beta = .49$, $p < .001$) had the largest weighting on Function 1, suggesting they were the most influential predictors in significantly differentiating between the clusters. The Flanker Inhibitory Control and Attention Test had a relatively low weighting on Function 1 ($\beta = .19$, $p < .001$), suggesting that although significant, it was not a strong contributor to the final classification among the other variables. Therefore, the first discriminant function (i.e., **Dimension 1**) could be defined by a combination of **cognitive flexibility** and **processing speed** abilities (see Table 5 for cognitive domains tapped by each task).

Further, the Structure Matrix revealed that the Oral Reading Recognition Test ($r = .53$), Picture Vocabulary Test ($r = .51$), List Sorting Working Memory Test ($r = .38$), Emotional Difficulties ($r = -.29$) and Hyperactive Behaviour ($r = -.24$) displayed the strongest correlations with Function 2. The Canonical Coefficients table indicated that the Oral Reading Recognition Test ($\beta = .49, p < .001$) and the Picture Vocabulary Test ($\beta = .49, p < .001$) had the largest weighting on Function 2, followed by Emotional Difficulties ($\beta = -.33, p < .001$), List Sorting Working Memory Test ($\beta = .32, p < .001$) and Hyperactive Behaviour ($\beta = -.30, p < .001$). These results imply that **language** and **working memory** abilities, as well as **emotional difficulties** and **hyperactive behaviour** were the most influential contributors in significantly differentiating between clusters along **Dimension 2**.

The classification table indicated an overall original accuracy rate of 97.1%, with 98.0% of Cluster 1 correctly classified, 95.9% of Cluster 2 correctly classified, and 98.1% of Cluster 3 correctly classified. The overall cross-validated accuracy rate was 96.0%, suggesting that the model also performed well on new, unseen data. Figure 2 visualises the extent to which the discriminant functions separated the data points into the three data-driven clusters.

Examination of the cluster centroids on each discriminant function revealed distinct profiles across the three clusters. Dimension 1 best differentiated Cluster 2 (weaker cognitive flexibility and processing speed abilities) from Cluster 3 (stronger cognitive flexibility and processing speed), with Cluster 1 falling in the middle. It should be noted that the Emotional Difficulties and Hyperactive Behaviour variables were negatively correlated with Dimension 2. Thus, clusters scoring high on this dimension should be interpreted as having stronger language and working memory skills, but more emotional and behavioural difficulties, whilst clusters scoring low on this dimension should be interpreted as demonstrating weaker language and working memory skills, but stronger emotional and behavioural functioning. To this end, Dimension 2 primarily separated Cluster 1 from Cluster 3, whilst Cluster 2 displayed moderate discriminant scores on this function. Specifically, Cluster 1 demonstrated lower scores than Cluster 3; i.e., Cluster 1 showed weaker language and working memory abilities, but fewer emotional difficulties and hyperactive behaviour problems, whilst Cluster 3 displayed stronger language and working memory abilities, but more emotional and behavioural difficulties.

Table 6. Structure matrix displaying pooled within-groups correlations between discriminating variables and standardised canonical discriminant functions.

Variable	Function 1	Function 2
Dimensional Change Card Sort Test	.773*	-.085
Pattern Comparison Processing Speed Test	.541*	-.260
Flanker Inhibitory Control and Attention Test	.495*	-.001
Picture Sequence Memory Test	.189*	.164
Oral Reading Recognition Test	.181	.534*
Picture Vocabulary Test	.229	.513*
List Sorting Working Memory Test	.186	.381*
Emotional Difficulties	.055	-.285*
Hyperactive Behaviour	-.085	-.240*
Emotional Clarity	-.020	.216*
Social Skills	-.003	-.048*

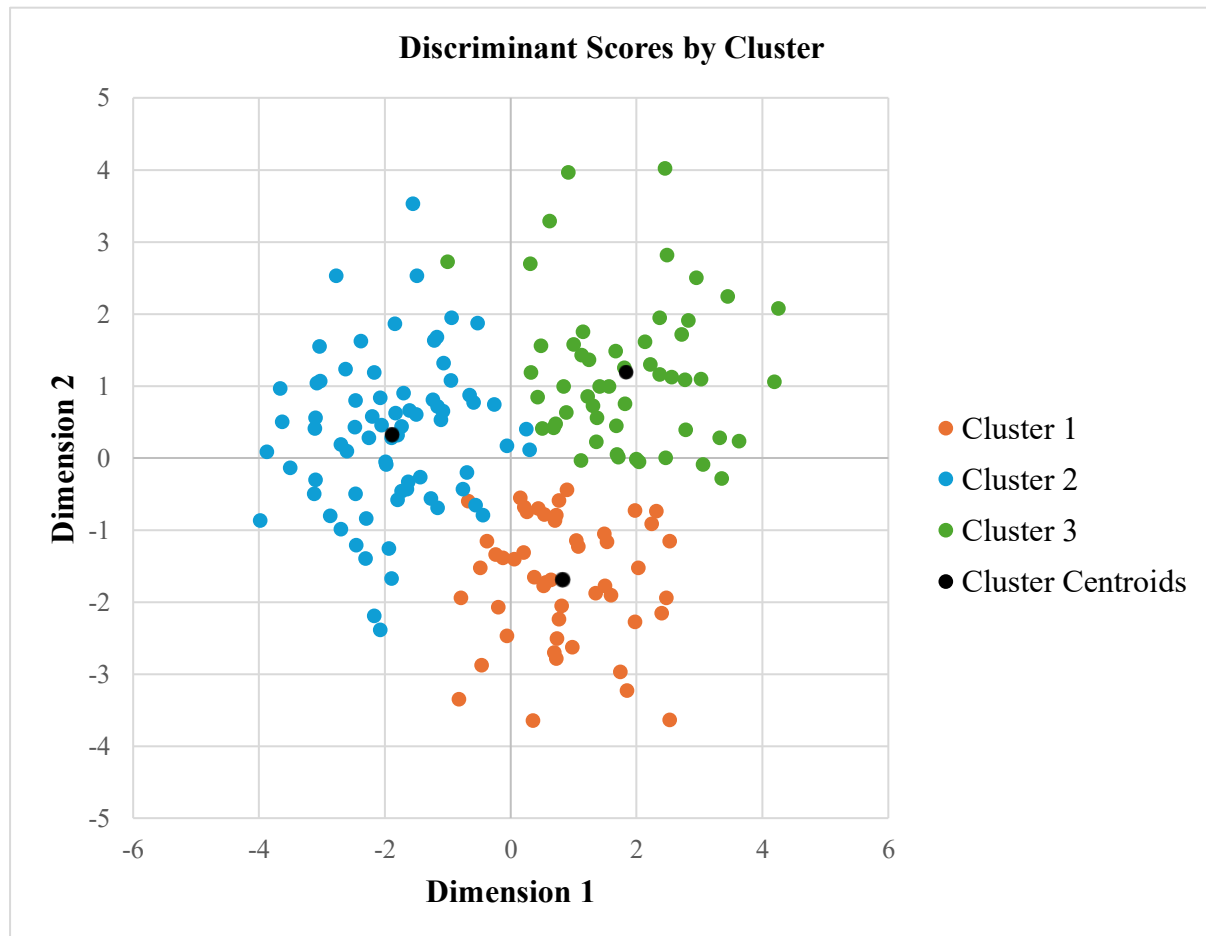
Note. * Largest absolute correlation between each variable and any discriminant function. The variables are ordered by absolute size of correlation within function.

Table 7. Standardised canonical discriminant function coefficients.

Variable	Function 1	Function 2
Emotional Difficulties	-.018	-.328
Emotional Clarity	-.195	.278
Social Skills	.064	-.099
Hyperactive Behaviour	-.076	-.301
Flanker Inhibitory Control and Attention Test	.185	.068
Dimensional Change Card Sort Test	.702	-.153
Picture Sequence Memory Test	.133	.135
List Sorting Working Memory Test	.050	.321
Pattern Comparison Processing Speed Test	.489	-.377
Picture Vocabulary Test	.009	.491
Oral Reading Recognition Test	.306	.490

Note. Standardised canonical coefficients represent the relative importance of each variable in distinguishing between groups, with higher absolute values indicating stronger contributions to the discriminant functions.

Figure 2. A scatterplot of discriminant function scores by data-driven cluster.



Note. The axes represent discriminant scores, derived from the linear combinations of input variables that best separate the clusters. Dimension 1 = cognitive flexibility and processing speed. Dimension 2 = language, working memory, emotional difficulties and hyperactivity.

Note

A multinomial logistic regression was also conducted (in jamovi, Version 2.3.28; The jamovi project, 2023) and compared with results of the LDA, given the convergence of the two methods in solving multi-classification problems (e.g., Antonogeorgos et al., 2009). The logistic regression model did not perform well when the four mental health components and seven cognitive variables were entered into the analysis simultaneously. Therefore, LDA was ultimately chosen as the preferred method for defining the structure of the dataset. Please refer to Appendix B (Supplementary Materials 3) for more detail.

4.5. Discussion

This chapter aimed to adopt a data-driven approach to grouping a transdiagnostic sample of neurodivergent adults based on patterns of strengths and difficulties across cognitive and mental health dimensions. First, a principal component analysis (PCA) was applied as a data-reduction technique to reduce multicollinearity of mental health variables and simplify the high-dimensional dataset in preparation for further analyses. The results of the PCA indicated that the sample was best defined by four mental health dimensions (i.e., principal components) relating to 1) Emotional Difficulties, 2) Emotional Clarity, 3) Social Skills, and 4) Hyperactive Behaviour. The four principal components were subsequently entered into a cluster analysis, along with seven cognitive variables corresponding to the domains of inhibitory control, cognitive flexibility, episodic memory, working memory, processing speed, vocabulary and reading decoding. The *k*-means cluster analysis revealed three data-driven groups which differed significantly from each other on all measures, except the Social Skills dimension. A linear discriminant analysis (LDA) was conducted to characterise the dimensions that best differentiate between the data-driven clusters in order to further define the underlying structure of the dataset. The results of the LDA revealed that the sample was best defined by two core dimensions (i.e., discriminant functions). Here, Dimension 1 was primarily defined by a combination of cognitive flexibility and processing speed abilities, suggesting that differences in these cognitive domains underpinned the most significant distinctions between the clusters. On the other hand, language and memory skills, as well as emotional difficulties and hyperactive behaviour, were the most influential contributors in significantly differentiating between clusters along Dimension 2. Together, these results suggest that the data-driven clusters reflect meaningful and interpretable profiles, driven by distinct cognitive and mental health dimensions.

More specifically, Cluster 1 included the lowest number of participants with a formal neurodiverse diagnosis (ND), with 5/9 of these individuals reporting a diagnosis of dyslexia. Given that Cluster 1 displayed only moderate discriminant scores on Dimension 1, results of the LDA indicated that this group was best defined by Dimension 2. Specifically, the group was characterised by weak language (i.e., vocabulary, reading decoding) and working memory abilities, as well as low emotional difficulties and low hyperactive behaviour. In other words, individuals in this group displayed specific weaknesses in cognition, but showed strengths in mental health functioning when compared with the other groups. Interestingly, the profile of

this group aligned closely with the cognitive difficulties often associated with dyslexia. For example, the results are in line with recent evidence suggesting a significant association between vocabulary, reading fluency and dyslexia (Maassen et al., 2022), as well as with research suggesting that working memory difficulties in dyslexia likely persist into adulthood (Smith-Spark & Fisk, 2007). Such close alignment with the cognitive profile associated with a formal diagnostic label is interesting, given that Cluster 1 was comprised of the lowest number of individuals with a formal ND. These findings may suggest that whilst Cluster 1 may not have captured many individuals meeting diagnostic thresholds, those individuals with suspected NDs who were included in this group may have presented with significant difficulties associated with dyslexia, which did not align with current diagnostic criteria, and may have therefore gone unidentified in clinical settings. This would align with the recently highlighted challenges associated with the identification of neurodiverse conditions in current clinical practice (outlined in the Introduction chapter of this thesis), and provide further support for the notion that data-driven approaches have the capacity to provide a more comprehensive understanding of individual functioning, compared to diagnostic processes grounded in categorical research (Astle et al., 2022).

Additionally, Cluster 1 also demonstrated relative strengths in mental health functioning, scoring low on emotional difficulties and hyperactive behaviour. These results are both expected and unexpected. First, given that this cluster captured the lowest number of individuals with formal NDs, the low scores on emotional and behavioural difficulties would be as anticipated, since many neurodiverse diagnoses have frequently been associated with mental health problems (e.g., van Steensel et al., 2013, Kouvava et al., 2022; Jensen & Steinhausen, 2015). Thus, since the other two clusters included more individuals with formal NDs, it is as expected that Cluster 1 would show relative strengths in mental health functioning compared to these groups. Specifically, Cluster 1 only included one individual with a diagnosis of ADHD (whilst Clusters 2 and 3 included substantially more); therefore, the low levels of self-reported hyperactive behaviour characterising Cluster 1 are as predicted, given that high levels of hyperactivity are characteristic of ADHD (APA, 2013). On the other hand, the results are not in line with research evidence suggesting that individuals with dyslexia are likely to experience elevated levels of internalising problems, such as anxiety or depression (Nelson & Harwood, 2011). Since the Emotional Difficulties dimension was highly correlated with scales assessing such mental health problems, and the cognitive difficulties characterising Cluster 1

were highly aligned with the cognitive profile associated with dyslexia, these results are not as expected.

Therefore, it is possible that Cluster 1 may have uncovered a unique profile of mental health strengths that is specific to neurodivergent adults with language and memory difficulties. Although emotional difficulties and hyperactive behaviour may be common among children with language and memory problems (Zuppardo et al., 2021; Martinussen et al., 2005), research has shown that the mental health profiles of neurodivergent individuals are inclined to change throughout development. For example, neurodivergent adults have been shown to demonstrate an improvement in emotional wellbeing over time (e.g., Scheeren et al., 2022), whilst research has also shown that neurodivergent adults may develop effective strategies to cope with their difficulties (e.g., Carawan et al., 2016). Therefore, the characteristics of Cluster 1 provide some insight into the potential emotional processing strengths of neurodivergent adults with specific cognitive difficulties in language and memory domains, which could provide targets for future intervention strategies; for example, through leveraging these mental health strengths to support individual wellbeing.

Further, Cluster 2 contained the highest number of individuals with a formal ND out of the three data-driven groups. This cluster was also the most heterogeneous in terms of diagnostic labels, capturing all but one formal ND within the dataset (Perceptual Processing Difficulty). Additionally, results of the LDA indicated that Cluster 2 was best characterised by cognitive flexibility and processing speed difficulties. In conjunction with the ANOVA results, it could be argued that Cluster 2 displayed the most ‘severe’ cognitive profile, scoring significantly low across all cognitive domains. These results are as expected, given that this data-driven group captured the highest number of individuals with formal NDs, and encompassed a wide range of diagnostic labels. Previous literature has shown that the co-occurrence of conditions often results in more severe difficulties and/or more complex cognitive profiles (e.g., Mayes et al., 2000; Wang & Chung, 2018), thus a highly heterogeneous transdiagnostic cluster displaying the most pronounced cognitive difficulties is in line with such evidence. Interestingly, whilst Cluster 2 showed relatively high emotional difficulty and moderate hyperactivity (as well as weak language and working memory abilities), its discriminant scores along Dimension 2 were moderate, suggesting that the observed weaknesses in cognitive flexibility and processing speed were more important for distinguishing this cluster from the other data-driven groups.

The above findings highlight a transdiagnostic group of neurodivergent adults with a unique profile of cognitive flexibility and processing speed weaknesses. Given the diversity of Cluster 2, it is worth questioning whether the combination of cognitive flexibility and processing speed difficulties may be a potential mechanism underpinning the co-occurrence of neurodiverse conditions. To this end, difficulties with cognitive flexibility have frequently been associated with a wide range of NDs, including autism, ADHD, and dyslexia (e.g., Lage et al., 2024; Roshani et al., 2020; Cartwright et al., 2017), and have been proposed to be a shared weakness in co-occurring NDs, such as ADHD and dyslexia (e.g., Lonergan et al., 2019). Similarly, research has shown evidence of shared processing speed weaknesses in ADHD and dyslexia (McGrath et al., 2011). Additionally, it has been suggested that individual differences in processing speed are influenced by genes that also increase the risk for dyslexia, ADHD and their co-occurrence (Willcutt et al., 2010). Therefore, it is possible that patterns of change in the developmental trajectories of cognitive flexibility and processing speed abilities may increase susceptibility for the emergence of co-occurring neurodiverse conditions.

Moreover, given that Cluster 2 displayed the most cognitive difficulties, it is interesting that this data-driven group was not also characterised by the weakest mental health profile. Research has shown that difficulties associated with neurodiverse conditions increase the risk for mental health problems, such as internalising (Baraskewich & McMorris, 2019); thus, it would be expected that more severe cognitive profiles would co-occur with increased mental health difficulties. In fact, results of the ANOVA revealed high levels of emotional difficulties in Cluster 2 compared to Cluster 1, yet (as mentioned above), overall mental health did not effectively differentiate Cluster 1 from the other two data-driven groups. Therefore, these results suggest that individuals with cognitive flexibility and processing speed differences may display relatively average mental health profiles. This is surprising given that recent research has emphasised the role of cognitive flexibility as a management tool for self-regulation and mental wellbeing in adults (Goldin et al., 2025), implying that weakened cognitive flexibility may increase susceptibility to mental health problems. Similarly, processing speed has recently been suggested to play a ‘bridging’ role between cognition and emotion regulation strategies in adults (Castro et al., 2025), suggesting that weakened processing speed may have a significant impact on overall mental health, given the transdiagnostic role of emotion regulation strategies (England-Mason, 2020). Therefore, the patterns of difficulties observed in Cluster 2 did not necessarily align with broader literature.

The unanticipated profile of Cluster 2 may be associated with the high number of individuals with suspected NDs captured by this data-driven group. Given that those with suspected conditions are more likely to present with milder, subthreshold difficulties, it is possible that since over 60% of Cluster 2 comprised individuals without a diagnostic label, those with milder mental health difficulties may have overshadowed individuals with more severe mental health profiles. This may have driven the cluster to be better characterised by ‘moderate’ mental health, than a more severe profile. However, recent transdiagnostic literature has emphasised the crucial need for gaining a deeper understanding of the functioning of neurodivergent individuals with milder difficulties, since such difficulties are often overlooked in both research and clinical settings, despite having a significant impact on individual lived experience (Astle et al., 2022). Thus, it is important to not only focus on the relative strengths and weaknesses in transdiagnostic research, but also to pay attention to ‘middle ground’ processes. Such processes may be important in gaining a more comprehensive understanding of individual wellbeing.

Finally, Cluster 3 displayed the highest functioning cognitive profile, and was best defined by strong cognitive flexibility and processing speed skills, as well as strong language and working memory abilities. Individuals in this group also displayed the most emotional difficulties and hyperactive behaviour compared to the other clusters. Interestingly, this cluster captured the largest number of individuals with ADHD (making up 25% of the cluster), compared to the other data-driven groups. Despite ADHD being a neurodiverse condition associated with difficulties in all of the above cognitive domains (e.g., Kofler et al., 2010; Holmes et al., 2010; Leib et al., 2021; McInnes et al., 2003), recent evidence has suggested that ADHD traits (e.g., hyperactivity, impulsivity) correlate positively with self-reported strengths in cognitive domains, such as cognitive flexibility (Schippers et al., 2024). The cognitive and mental health profiles of Cluster 3 therefore provide further support for such evidence. However, since the broader literature generally points towards complex profiles of cognitive difficulties in adults with ADHD (Onandia-Hinchado et al., 2021), it is surprising that Cluster 3 included such a high number of participants with this diagnosis, given the strong cognitive profile of this data-driven group. Nonetheless, the significantly high levels of self-reported hyperactive behaviour in Cluster 3 reflect the high percentage of ADHD diagnoses within this group, providing good validation for the cluster structure.

Notwithstanding the inconsistencies with the broader literature, Cluster 3 may have uncovered a unique profile of high-functioning neurodivergent adults, despite experiencing significant

emotional and behavioural difficulties. This is important to consider in the context of future support strategies. For instance, cognitive control training has been shown to improve mental health outcomes (Koster et al., 2017), and many interventions, such as cognitive behavioural therapy, harness cognitive strategies to effectively treat a range of mental health problems (e.g., Tolin, 2010). Thus, the profile of Cluster 3 may have highlighted several cognitive areas in which neurodivergent individuals with mental health difficulties demonstrate specific strengths, which could provide potential targets for future support strategies. These findings also highlight the benefits of transdiagnostic dimensional research, as it provides the opportunity to assess strengths, not just weaknesses, that are relevant across a wide range of NDs (Astle et al., 2022). For instance, Cluster 3 not only comprised participants with ADHD, but also individuals with diagnoses of dyslexia and/or autism, as well as participants with suspected NDs. This suggests that the identified profile of cognitive strengths and mental health difficulties transcends diagnostic categories, and therefore any support strategies grounded in such transdiagnostic research have the capacity to benefit individuals with profiles that do not directly align with conventional diagnostic categories.

It should be noted that despite some alignment of the cluster profiles with the diagnostic criteria of their most prevalent diagnostic labels (e.g., dyslexia in Cluster 1, ADHD in Cluster 3), none of the data-driven clusters mapped directly onto traditional diagnostic categories. All of the clusters were transdiagnostic, in that they all captured a minimum of three diagnostic labels, with Cluster 2 being the most heterogeneous of all. This is in line with recent evidence from transdiagnostic studies showing that data-driven profiles of neurodevelopmental difficulties often fail to align with formal diagnostic labels in children (Mareva et al., 2023; Mareva et al., 2024). The current findings therefore build on previous quantitative research through providing empirical evidence of unique transdiagnostic cognitive and mental health profiles in neurodivergent adults.

4.5.1. Strengths and Limitations

There are several strengths to this chapter's methodology. First, cluster analysis is a completely data-driven technique, which does not rely on any predefined criteria for grouping individuals (Astle et al., 2022). In this way, clustering enabled the grouping of participants based on individual strengths and weaknesses in cognition and mental health, as opposed to grouping individuals based on arbitrary diagnostic categories. This has yielded three unique profiles of differing patterns of strengths and difficulties in various domains of functioning. Such findings, along with the specific composition of each cluster (in terms of diagnostic labels), have provided valuable insights into some of the mechanisms underpinning the complexity of NDs. For instance, the profile of Cluster 2 may have uncovered some of the cognitive mechanisms associated with the emergence of co-occurring NDs, given that this cluster was the most diverse in terms of diagnostic labels. Similarly, the contrast in the profiles of Cluster 1 and Cluster 3 could be seen as providing some insight into within-diagnosis heterogeneity, since individuals with formal diagnoses of ADHD, autism, and dyslexia were present in each cluster, yet best defined by different combinations of strengths and difficulties in cognition and mental health. Although further research is needed to confirm such preliminary insights, the findings provide strong evidence for the benefits of applying data-driven techniques to elucidate the processes underlying some of the complexity associated with neurodiverse conditions.

Second, the combination of PCA, cluster analysis and LDA has allowed for the identification of transdiagnostic clusters that reflect meaningful and interpretable profiles, driven by distinct cognitive and mental health dimensions, despite the multivariate nature of this complex dataset. This is a major advantage of dimensional research over traditional 'case-control' studies, which typically involve group-level statistical analyses, where significant between-group differences are used to make inferences about 'deficits' specific to the 'case' (i.e., clinical) group, without taking into consideration within-group variability (Astle & Fletcher-Watson, 2020). To this end, focusing on univariate and/or bivariate data (i.e., one specific variable or the relationship between two variables) omits the possibility to explore the heterogeneous nature of neurodiverse conditions, where the possibilities for complex interactions between various dimensions are many (Astle & Fletcher-Watson, 2020). Thus, the unique combination of various data-reduction techniques was well-suited for this transdiagnostic dataset, allowing for the identification of meaningful data-driven profiles of functioning, whilst also recognising the inherent heterogeneity of neurodiverse conditions.

Despite the advantages of data reduction techniques for exploring the underlying structure of complex datasets, some unexpected results emerged in this study. As mentioned above, the profile of Cluster 2 (highly heterogeneous, severe cognitive difficulties, yet average mental health) did not align well with the broader literature. Whilst this may be due to true observations within the dataset, it is important to consider how methodological limitations may have contributed to these results. First, information loss is a common limitation of dimensionality reduction techniques, such as PCA (Geiger & Kubin, 2012). Since PCA was used to reduce mental health variables into fewer components, it is possible that this procedure may have oversimplified the dataset, resulting in potential information loss. For instance, distinct patterns of difficulties (e.g., anxiety and depression) may have been merged into a single component (as can be seen in the Emotional Difficulties dimension). This process may have masked important individual variation within these mental health processes. Subsequently, this may have resulted in less extreme overall scores for individuals with more severe emotional difficulties, which could have been reflected in the moderate discriminant scores along Dimension 2 of the LDA for Cluster 2.

Second, it is possible that the clustering procedure itself may have contributed to the unexpected profile of Cluster 2. For example, *k*-means clustering can often be influenced by ‘dominant’ variables (i.e., those with larger variances), which can result in clusters being primarily determined by those specific features (e.g., Steinley & Brusco, 2008). Since a larger number of cognitive variables showing significant variability were entered into the analysis (in comparison to the mental health dimensions), the clustering algorithm may have underrepresented some of the mental health variables in the cluster solution. Additionally, *k*-means clustering assigns all participants to a group depending on which cluster centre the individual data points are nearest to (Tarpey, 2007), even if outliers are present. This may have resulted in the clustering of individuals with discrepant profiles. Therefore, Cluster 2 may well have included participants with complex profiles of severe cognitive and mental health difficulties – which would be more in line with the broader literature – yet this may have been overshadowed in the process of clustering.

Moreover, LDA identifies linear combinations of variables that best separate data-driven clusters, and assumes homogeneity of variance-covariance across groups (Zhao et al., 2024; Tharwat et al., 2017). If this assumption is violated (i.e., if the variances and covariances

between groups are significantly different), the LDA model may not accurately separate the data-driven groups. This could happen by, for example, giving more weight to cognitive variables over mental health dimensions, resulting in overexaggerated between-group differences in cognition, and potentially overriding more subtle, yet still important mental health differences. However, since Box's M test results suggested that the assumption was not violated, it is unlikely that this limitation could have contributed to the unexpected results, making the contribution of PCA or cluster analysis limitations more likely. Nonetheless, these methodological limitations should be considered when interpreting the results of this chapter.

Another noteworthy consideration is that the Social Skills dimension identified via PCA was not revealed to be significant in differentiating between clusters in neither the ANOVA, nor the LDA results. This is surprising given that social skills are one of the key functional outcomes shown to be affected in several neurodiverse conditions (e.g., Silveira-Zaldivar et al., 2021; Dellapiazza et al., 2021; McKay et al., 2023). This observation may also be the result of methodological limitations. For instance, given that the Social Skills component accounted for the smallest proportion of variance compared to the other components extracted from PCA, it is likely that this variable may have been omitted during the clustering process since cluster analysis tends to prioritise dominant variables with larger variances (e.g., Steinley & Brusco, 2008), as previously mentioned. Therefore, even though Social Skills formed a coherent construct, this dimension may not have displayed sufficient between-group variance to substantially influence the clustering process, or the discriminant functions identified by LDA. To this end, the combination of PCA, cluster analysis and LDA meant that each analysis built on the results of the previous one, and although the use of data reduction techniques is effective for simplifying and identifying the underlying structure of complex datasets, the combination of the techniques may have resulted in the exclusion of subtle (yet meaningful) features, which could not be recovered along the data reduction process.

Finally, some of the unexpected results of this chapter may have resulted from the use of a combination of self-report mental health measures, and direct assessments of cognition. In a recent study by Mareva and colleagues (2024), a *k*-means cluster analysis was applied to identify data-driven profiles of executive function in a transdiagnostic sample of neurodivergent children. This study included both performance-based tasks to directly assess executive function, as well as behaviour ratings (i.e., questionnaires) to capture the application of executive function skills in everyday contexts. The cluster analysis identified three data-

driven groups with differing profiles of executive function across the two measures. Importantly, there were significant differences in self-reported mental health outcomes across the three clusters (Mareva et al., 2024). These results suggest that there may be differences in the relationships between behavioural ratings and performance-based assessments of executive function with self-report measures of mental health. Thus, it is possible that the unexpected profile of Cluster 2 (i.e., severe cognitive difficulties in the absence of mental health or behavioural problems) may have resulted from the inconsistency between performance-based and self-report measures of cognition and mental health. This study may have therefore benefited from the inclusion of self-report measures of cognition, such as the Behaviour Rating Index of Executive Function (BRIEF; Gioia et al., 2000), in order to cross-compare and/or validate the unexpected patterns of results in the present dataset.

4.5.2. Conclusion and Future Directions

In conclusion, this chapter explored the underlying structure of the present transdiagnostic dataset through identifying naturally occurring data-driven groups. The results showed that the current sample of neurodivergent adults consisted of three transdiagnostic clusters, which were best characterised by specific combinations of strengths and difficulties along two dimensions relating to cognition and mental health. Importantly, the data-driven groups did not directly map onto traditional diagnostic categories. The results of this chapter have added to the emerging pool of data-driven accounts of neurodiversity, and highlighted several cognitive and mental health processes which may contribute to the complexity associated with NDs, thus providing further support for transdiagnostic dimensional accounts of neurodiverse conditions. These findings have the potential to inform future assessment and support of NDs in adults, but need further validation given their exploratory nature.

Future research should explore alternative dimensionality reduction approaches in order to enhance sensitivity to more subtle processes. For instance, hierarchical clustering provides the opportunity to give more weight to theoretically meaningful constructs that may contribute less to overall variance, allowing for the formation of more balanced, data-driven groupings (e.g., Everitt et al., 2011). It does so by hierarchically organising groups based on pairwise comparisons, making it better suited for detecting subtle patterns within the dataset, even if such data-driven groups reflect only small variations (Ran et al., 2023). Similarly, future studies could benefit from the use of network analysis for uncovering patterns of between-variable

interactions that may be overlooked in the process of clustering. Network analysis models direct between-variable connections within a given network, thus allowing for the identification of processes that may act as key mediators or ‘bridging’ mechanisms between different parts of the network (Borsboom et al., 2021; Mareva et al., 2019). In this way, the important role of more subtle processes can be explored, even if they do not necessarily explain large amounts of variance in a given dataset. The following chapter will therefore adopt an exploratory network analysis approach in order to gain a more comprehensive understanding of the subtle interactions between the cognitive and mental health processes in the current dataset.

Chapter 5: Network Analysis

This chapter will adopt an exploratory network analysis approach with the aim to gain a more comprehensive understanding of the interactions between the cognitive and mental health dimensions characterising the current dataset. The chapter will begin by providing a rationale for the application of network analysis in transdiagnostic research and the present study (Section 5.1.). This will be followed by a description of the methods used to estimate the network structure (Section 5.2.). Two network models with different regularisation parameters will then be compared in terms of their ability to balance sensitivity and specificity in detecting associations between variables (Section 5.3.). The results will present the estimated network structures, and highlight key nodes with high centrality and bridging roles. The chapter will then conclude with a discussion of the findings and their potential implications, including a consideration of methodological limitations and suggestions for future research (Section 5.4.).

5.1. Introduction

Understanding the complex interplay between cognitive and mental health processes requires a statistical approach with the capacity to capture interdependencies, rather than simply assuming linear relationships. In the previous chapter, a cluster analysis was performed to identify naturally occurring groups within the current transdiagnostic dataset. Since data-driven clusters are formed based on patterns of correlations between variables which typically reveal the linear component of their covariance (Pivodori et al., 2024), such statistical assumptions may not reflect the underlying nature of the complex interactions between cognitive and/or mental health processes. Network analysis offers an alternative method of visualising and quantifying the associations between such processes, allowing the identification of key variables and their structural roles within a given network (Borsboom et al., 2021). The more connections (i.e., correlations) a given variable has with other variables, the more variance it can explain between datasets, and the higher its predictability within the network model (Mareva et al., 2019).

Network analysis is increasingly being applied in the context of neurodiverse diagnoses (NDs), with a wide range of research providing evidence for the benefits of utilising a network approach for exploring complex between-variable interactions. For instance, Mareva and colleagues (2019) identified transdiagnostic associations across communication, cognitive and

behavioural difficulties, as well as potential ‘bridging symptoms’ that may contribute to the development of co-occurring conditions in a heterogeneous sample of struggling learners. Similarly, Wolff and colleagues (2023) adopted a network approach to assess individual-level risk and resilience factors associated with mental health in siblings of neurodivergent individuals, and found executive function (specifically cognitive inflexibility and hyperactivity/impulsivity) and emotion regulation difficulties to be the most influential risk factors for poorer functioning outcomes. Further, network analysis has also been applied to gain a deeper understanding of within-diagnosis heterogeneity. For instance, Liu and colleagues (2022) identified two traits of ADHD (“distracted” and “fidget”) to be the ‘central symptoms’, and suggested that a network-informed approach has the capacity to refine the ADHD phenotype and potentially reduce heterogeneity. A network approach has also been utilised to map reading profiles in dyslexia, with results supporting the notion that a single ‘core deficit’ cannot account for atypical reading development (Verwimp et al., 2023). Together, recent literature demonstrates the utility and applicability of network analysis in the context of neurodiversity research, particularly for characterising the underlying mechanisms that may contribute to individual variation or overlaps between conditions, which may not be captured by conventional statistical approaches or traditional diagnostic categories. Such data-driven evidence has the potential to identify individual areas of need, which can provide targets for more individualised support strategies.

Moreover, recent transdiagnostic literature has suggested that network modelling may also be used to validate clusters of characteristics against conventional diagnostic categories to confirm whether meaningful distinctions have been made between identified clusters, and to potentially aid the revision of diagnostic labels (Astle et al., 2022). The majority of research adopting a quantitative approach to exploring neurodiverse conditions has suggested that data-driven groups are most often transdiagnostic and thus do not directly align with traditional diagnostic categories, particularly at the cognitive level (e.g., Zhang et al., 2022; Mareva et al., 2024; Al-Saoud et al., 2025). However, evidence from research adopting a network analysis approach to exploring ADHD and autism symptoms in youth has also found individual ADHD and autism symptoms to be largely segregated in accordance with diagnostic boundaries, despite the high co-occurrence of the two conditions (Farhat et al., 2022; Rong et al., 2021). This emphasises the need for further quantitative accounts of NDs, not just in terms of understanding how individual profiles best fit into data-driven groups, but also in gaining a deeper understanding

of the dynamic interactions between various processes that may underpin the complexity of individual profiles.

5.1.1. Aims

The aim of this chapter is to adopt a network analysis approach to exploring the interplay between cognitive and mental health processes that characterise variability in the current transdiagnostic dataset. This is especially important given the results of the cluster analysis described in Chapter 4, where the combination of various data-reduction techniques enabled the characterisation of the underlying structure of the dataset, but likely resulted in the exclusion of subtle, yet meaningful between-variable interactions. The aim of this network analysis is therefore to a) estimate and visualise the network structure of the subtle interactions between cognitive and mental health dimensions, b) identify the most influential variables within the network, c) detect communities of variables, and d) determine which processes act as key connectors (or ‘bridges’) between such communities. Ultimately, this chapter provides the opportunity to further elucidate the mechanisms that may contribute to the complexity of NDs.

5.2. Methods

5.2.1. Data Preparation

The four principal components (Emotional Difficulty, Emotional Clarity, Social Skills and Hyperactive Behaviour) derived from mental health variables were entered into the analysis (see Chapter 4 for more detail). Principal components were used in the network analysis as highly correlated variables can inflate edge weights and consequently distort network structure, but principal component analysis (PCA) creates orthogonal components, which mitigates the issue of multicollinearity and enhances network stability (Chan et al., 2022). Cognitive variables were not reduced to principal components, since this would result in the loss of meaningful domain-specific differences (see Chapter 4). All raw data were converted to sample-referenced *z*-scores to ensure consistency in scaling. The Strengths and Difficulties Questionnaire (SDQ) and the Extended Strengths and Weaknesses Assessment of Normal Behaviour (E-SWAN) variables were rescaled such that higher scores indicated more adaptive functioning (or fewer difficulties) across all measures. While the network model was estimated

using regularised partial correlations (controlling for all other variables), Table 1 displays zero-order Pearson’s correlations to illustrate the direct bivariate relationships among variables. The partial correlations are visualised in the network graphs presented in the Results section. Note that for conciseness in the Results section, all cognitive variables will be referred to as acronyms derived from the names of specific tasks (see Table 1 legend). For reference, the cognitive domains represented by each task are described in Chapter 4 (Table 5).

5.2.2. Statistical Analyses

The analyses were conducted in the following steps: network estimation, network inference based on centrality measures, network stability, community detection, and exploratory bridge centrality analysis. All analyses were performed in *R* Studio (*R* Core Team, 2024), using the packages *qgraph* (Epskamp et al., 2012), *igraph* (Csardi & Nepusz, 2006), *psych* (Revelle, 2024), *bootnet* (Epskamp et al., 2018), *ggplot2* (Wickham & Sievert, 2009), *haven* (Wickham et al., 2025), and *networktools* (Jones, 2018).

5.2.3. Network Estimation and Visualisation

The network was estimated using the graphical variant of the least absolute shrinkage and selection operator (*glasso*) to avoid the inclusion of spurious connections. This approach estimates a Gaussian Graphical Model (GGM) by applying L1 regularisation, which imposes a penalty on the absolute values of the partial correlations between cognitive and mental health variables (Epskamp et al., 2018). This encourages weaker edges to shrink toward zero to promote a more sparse and interpretable network structure (Dito et al., 2019). The best fitting model was selected based on the Extended Bayesian Information Criterion (EBIC) with an initial tuning parameter of 0.5, in order to balance model parsimony and goodness of fit. This approach ensures good specificity (i.e., including only strong, meaningful connections), but could result in the loss of sensitivity to detect small edges (i.e., weaker connections) that are truly present within the network (Dziak et al., 2020). The network was visualised using a force-directed layout (Fruchterman-Reingold algorithm), which positions nodes with stronger connections closer together whilst pushing less connected nodes apart (Jones et al., 2018). This enhances the interpretability of network structure by naturally grouping related variables together. Each node in the network represents a cognitive or mental health dimension, and each edge represents a regularised partial correlation between two variables, indicating a direct

relationship after accounting for all other relationships. Thicker edges represent stronger connections, while weaker connections are reduced or removed entirely through regularisation.

Given the exploratory nature of this analysis, the initial $\gamma = 0.5$ model was compared with a denser, non-thresholded network (i.e., $\gamma = 0$), with the aim to avoid missing important (although weaker) connections with potential capacity to inform of underlying mechanisms. The two models were compared to explore the trade-off between specificity and sensitivity. The regularised model was more specific through capturing robust associations, but less sensitive to weaker connections. The non-thresholded network was more sensitive to weaker connections, but with a greater risk of including more spurious associations (thus demonstrating lower specificity).

Table 1. Pearson's correlation matrix for mental health components and cognitive variables ($n = 175$).

Variable	1	2	3	4	5	6	7	8	9	10
1. Emotional Difficulties	-									
2. Emotional Clarity	0.00	-								
3. Social Skills	0.00	-0.00	-							
4. Hyperactive Behaviour	0.00	0.00	0.00	-						
5. FICAT	0.19*	-0.03	-0.02	-0.08	-					
6. DCCST	0.10	0.01	-0.05	-0.06	0.69***	-				
7. PSMT	-0.12	0.11	-0.04	0.01	0.16*	0.30***	-			
8. LSWMT	-0.05	0.10	0.01	-0.14	0.25***	0.23**	0.29***	-		
9. PCPST	0.15*	0.06	0.04	-0.08	0.46***	0.56***	0.17*	0.13	-	
10. PVT	-0.18*	0.15*	0.09	-0.16*	0.30***	0.31***	0.22**	0.33***	0.16*	-
11. ORRT	-0.08	0.15	-0.09	-0.20**	0.13	0.10	0.09	0.31***	0.05	0.33***

Note. * $p < .05$, ** $p < .01$, *** $p < .001$. *FICAT* = Flanker Inhibitory Control and Attention Test. *DCCST* = Dimensional Change Card Sort Test. *PSMT* = Picture Sequence Memory Test. *LSWMT* = List Sorting Working Memory Test. *PCPST* = Pattern Comparison Processing Speed Test. *PVT* = Picture Vocabulary Test. *ORRT* = Oral Reading Recognition Test. The values represent Pearson's zero-order correlations between variables for descriptive purposes. The network model was estimated using a Gaussian Graphical Model (GGM) based on regularised partial correlations.

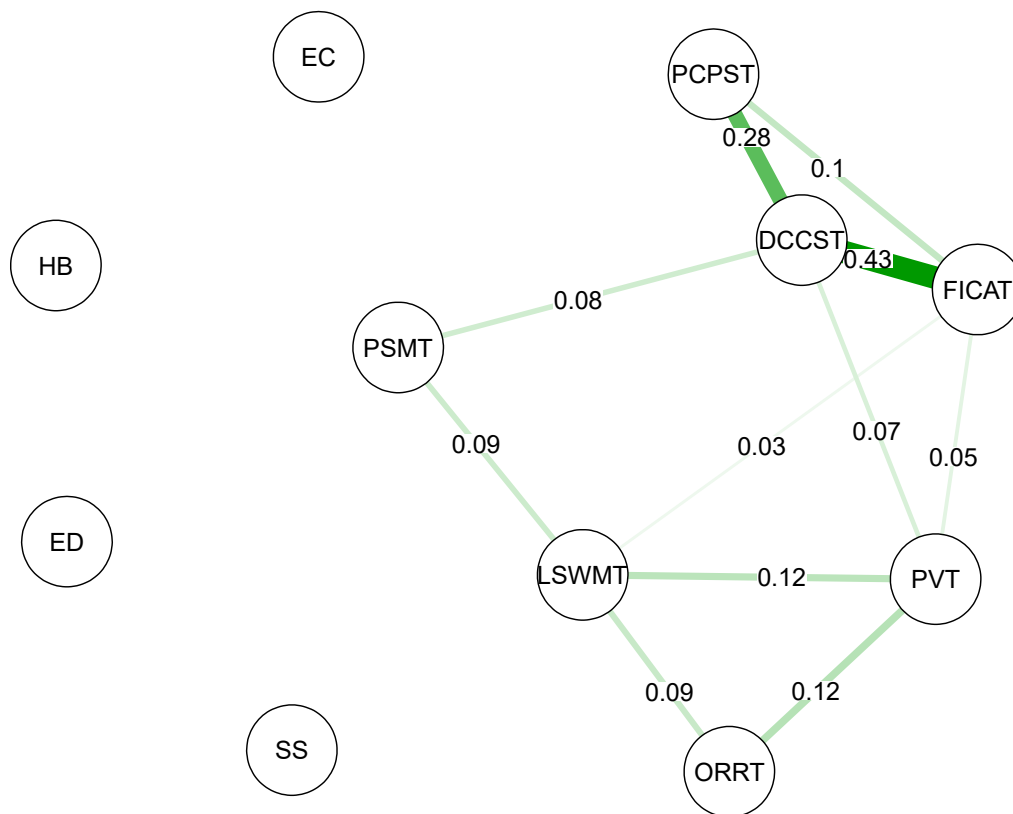
5.3. Results

5.3.1. Edge Weights

The initial network analysis revealed that the strongest connection was between the Flanker Inhibitory Control and Attention Test (FICAT) and the Dimensional Change Card Sort Test (DCCST; $r = 0.43$). A moderate association was also observed between the Pattern Comparison Processing Speed Test (PCPST) and DCCST ($r = 0.28$). Weaker relationships were observed between the List Sorting Working Memory Test (LSWMT) and the Picture Vocabulary Test (PVT; $r = 0.12$), the Oral Reading Recognition Test (ORRT) and PVT ($r = 0.12$), as well as PCPST and FICAT ($r = 0.10$). Other weaker, yet still relevant, connections are presented in Figure 1. The initial model ($\gamma = 0.5$) did not capture any connections relating to mental health dimensions.

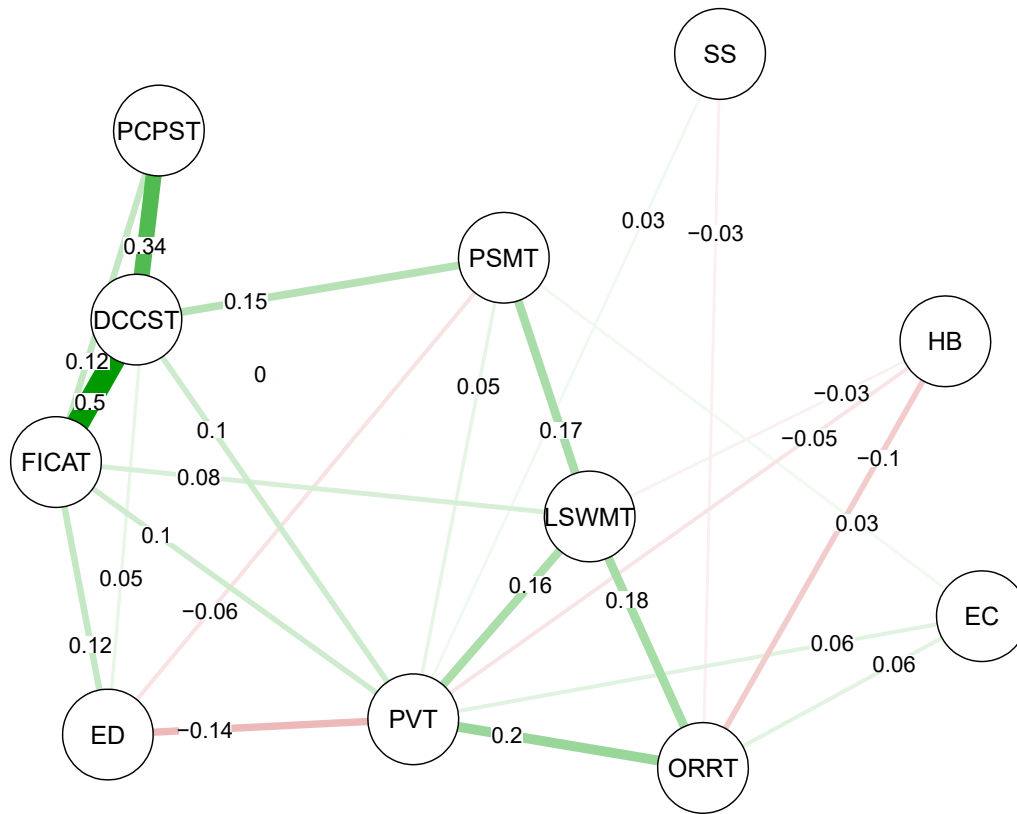
When applying no regularisation parameter ($\gamma = 0$), weaker connections across mental health dimensions were revealed (see Figure 2). The strongest associations between FICAT and DCCST ($r = 0.50$) and PCPST and DCCST ($r = 0.34$) were retained (and slightly strengthened). Similarly, the weaker relationships between PVT and LSWMT ($r = 0.16$), LSWMT and ORRT ($r = 0.18$), PVT and ORRT ($r = 0.20$) and PCPST and FICAT ($r = 0.12$) were maintained and strengthened. Increased, yet still relatively weak associations were also observed between the Picture Sequence Memory Test (PSMT) and DCCST ($r = 0.15$), as well as PSMT and LSWMT ($r = 0.17$), and FICAT and PVT ($r = 0.10$). Interestingly, the tuning = 0 model revealed a weaker, yet noteworthy relationship between Emotional Difficulties (ED) and FICAT ($r = 0.12$), which was not captured by the initial model. Several weaker (but notable) edges were also observed between Emotional Clarity (EC) and PVT ($r = 0.06$), and EC and ORRT ($r = 0.06$). Finally, the $\gamma = 0$ model revealed a weaker inverse relationship between ED and PVT ($r = -0.14$), as well as Hyperactive Behaviour (HB) and ORRT ($r = -0.10$). Relevant weak negative associations were also observed between ED and PSMT ($r = -0.06$), and HB and PVT ($r = -0.05$). Any non-significant edges < 0.05 were not considered, as these represent unstable connections that do not substantially contribute to the overall network structure (see Figure 2).

Figure 1. Estimated network model ($\gamma = 0.5$) showing cognitive and mental health dimensions.



Note. *EC* = Emotional Clarity. *HB* = Hyperactive Behaviour. *ED* = Emotional Difficulties. *SS* = Social Skills. *PCPST* = Pattern Comparison Processing Speed Test. *DCCST* = Dimensional Change Card Sort Test. *FICAT* = Flanker Inhibitory Control and Attention Test. *PSMT* = Picture Sequence Memory Test. *LSWMT* = List Sorting Working Memory Test. *PVT* = Picture Vocabulary Test. *ORRT* = Oral Reading Recognition Test. Each node represents a variable (i.e., a cognitive or mental health dimension), and each edge represents a regularised partial correlation between two nodes, controlling for all other variables. Stronger edges (indicated by thicker and more saturated lines) reflect stronger unique associations. Positive associations are shown in green, negative associations in red. The position of the nodes is determined by the strength of their connections, with more strongly connected nodes appearing closer together.

Figure 2. Estimated network model ($\gamma = 0$) showing cognitive and mental health dimensions.



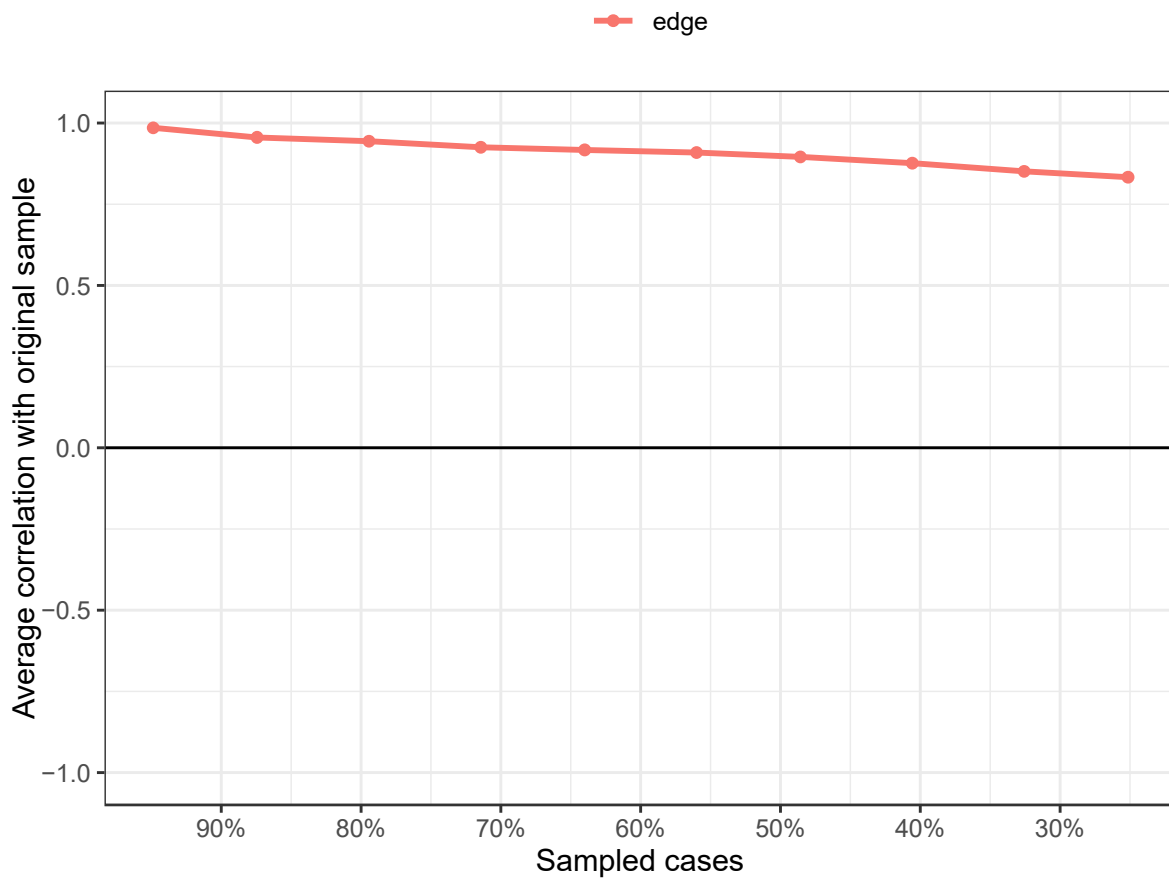
Note. *EC* = Emotional Clarity. *HB* = Hyperactive Behaviour. *ED* = Emotional Difficulties. *SS* = Social Skills. *PCPST* = Pattern Comparison Processing Speed Test. *DCCST* = Dimensional Change Card Sort Test. *FICAT* = Flanker Inhibitory Control and Attention Test. *PSMT* = Picture Sequence Memory Test. *LSWMT* = List Sorting Working Memory Test. *PVT* = Picture Vocabulary Test. *ORRT* = Oral Reading Recognition Test. Each node represents a variable (i.e., a cognitive or mental health dimension), and each edge represents a regularised partial correlation between two nodes, controlling for all other variables. Stronger edges (indicated by thicker and more saturated lines) reflect stronger unique associations. Positive associations are shown in green, negative associations in red. The position of the nodes is determined by the strength of their connections, with more strongly connected nodes appearing closer together.

5.3.2. Stability of Edge Weights

Bootstrapped stability tests were conducted to assess the robustness of the estimated edge weights in the initial network with a tuning parameter of 0.5. A series of sampling levels were tested, with drop percentages ranging from 5.1% to 74.9%, leading to varying sample sizes (from 44 to 115 participants). This was performed to determine the maximum drop proportions required to retain a correlation of 0.7 between nodes in at least 95% of the bootstrapped samples ($N = 1000$). The results indicated that the data drop percentage needed to be between 51.4% and 67.4%, corresponding to sample sizes ranging from 88 to 103 participants, in order for edges to remain stable. Additionally, a 0.59 correlation threshold was identified as the minimum required for edge stability, indicating that edges with lower correlations may become unstable as the sample size decreases.

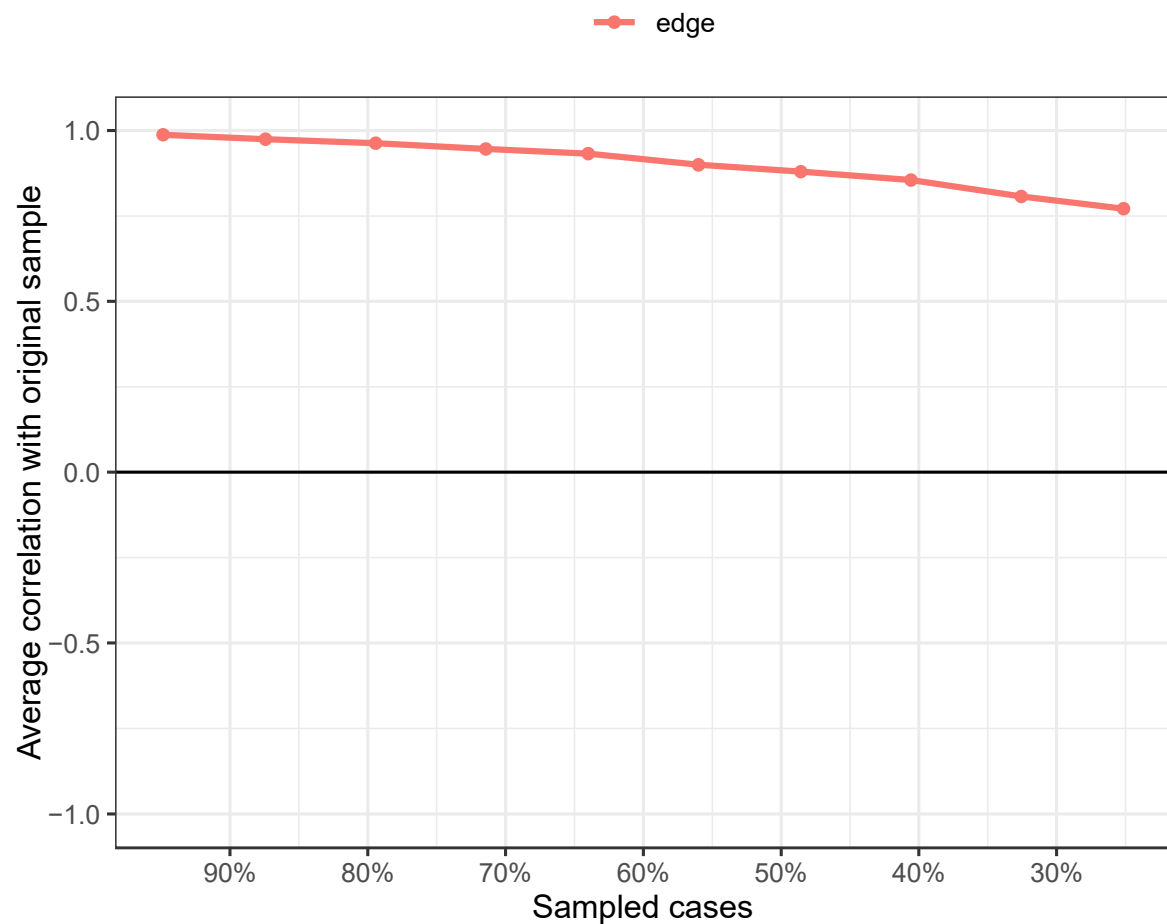
The stability of edge weights was also assessed for the second network model with a tuning parameter of 0. The model maintained a correlation of 0.7 between nodes in 95% of bootstrapped samples even with up to a 74.9% data drop, demonstrating greater overall stability. However, this model required a stronger correlation threshold of 0.67 for the edges to remain stable. These results suggest that the $\gamma = 0.5$ model demonstrated greater robustness in maintaining stable edge weights, as evidenced by a higher average correlation with the original sample across different data retention levels (see Figure 3). This demonstrates that the stronger regularisation applied in this model successfully removed weaker edges, whilst preserving only the most stable connections. Conversely, the $\gamma = 0$ model displayed greater variability in edge weights despite sample size reductions (see Figure 4), indicating that this model retained more connections (including those that were less stable). Ultimately, the results highlight a trade-off between sensitivity and specificity, with the $\gamma = 0.5$ model producing a more stable and sparser network (i.e., more specific, less sensitive), and the $\gamma = 0$ model being denser and more flexible (i.e., more sensitive, less specific), but at the cost of higher fluctuations in retained edges.

Figure 3. Edge weight stability plot for the $\gamma = 0.5$ network model.



Note. The y-axis represents the average correlation between edge weights in resampled networks and the original network. The x-axis indicates the proportion of sampled cases. Higher values reflect greater edge weight stability, with correlations approaching 1.0 indicating strong consistency across subsamples. Lower correlations suggest weaker or less stable connections. As the sample size decreases, variability in edge weight stability increases.

Figure 4. Edge weight stability plot for the $\gamma = 0$ network model.



Note. The y-axis represents the average correlation between edge weights in resampled networks and the original network. The x-axis indicates the proportion of sampled cases. Higher values reflect greater edge weight stability, with correlations approaching 1.0 indicating strong consistency across subsamples. Lower correlations suggest weaker or less stable connections. As the sample size decreases, variability in edge weight stability increases.

5.3.3. Centrality Measures

A series of centrality measures were calculated to assess the relative importance of nodes within each network. The indices include:

- a) Strength Centrality: the relative influence of each variable based on the weight of its connection.
- b) Degree Centrality: the number of direct connections a node has in terms of outgoing and incoming links.
- c) Closeness Centrality: how efficiently each variable connects to others in the network.
- d) Betweenness Centrality: how often a node lies on the shortest path between nodes.
- e) Expected Influence: the extent to which each variable exerts and receives influence within a network.

The full results are presented in Tables 2-3, and Figures 5-6 (for visual comparison between models).

Initial Network Model ($\gamma = 0.5$)

Given that the edge weights in the initial model ranged from 0.03 to 0.43, with most values clustering at the lower end, there was not enough variation to meaningfully differentiate between centrality measures. This resulted in identical values being observed across strength centrality, degree centrality and expected influence (see Table 2). Since the connections in this network model did not vary in importance, *degree centrality* alone could be used as a good proxy for influence. The results indicated that DCCST and FICAT displayed the highest degree centrality, whilst PCPST, PVT, LSWMT, ORRT, and PSMT displayed lower degree centrality, ranging from 0.38 to 0.17 (see Table 2). All mental health dimensions displayed a degree centrality of zero. These results indicate that cognitive dimensions were more central to the network, whilst there was a lack of connectivity between cognitive and mental health dimensions.

Further, DCCST demonstrated the highest *betweenness centrality* (see Table 2), indicating that this variable served as a key link between other variables within the network. PVT, PSMT and LSWMT displayed lower betweenness scores, suggesting that these variables also contributed to the network's overall connectivity, but were not as central. Finally, FICAT, ORRT and PCPST did not contribute to network connectivity, with betweenness scores of 0. Similarly,

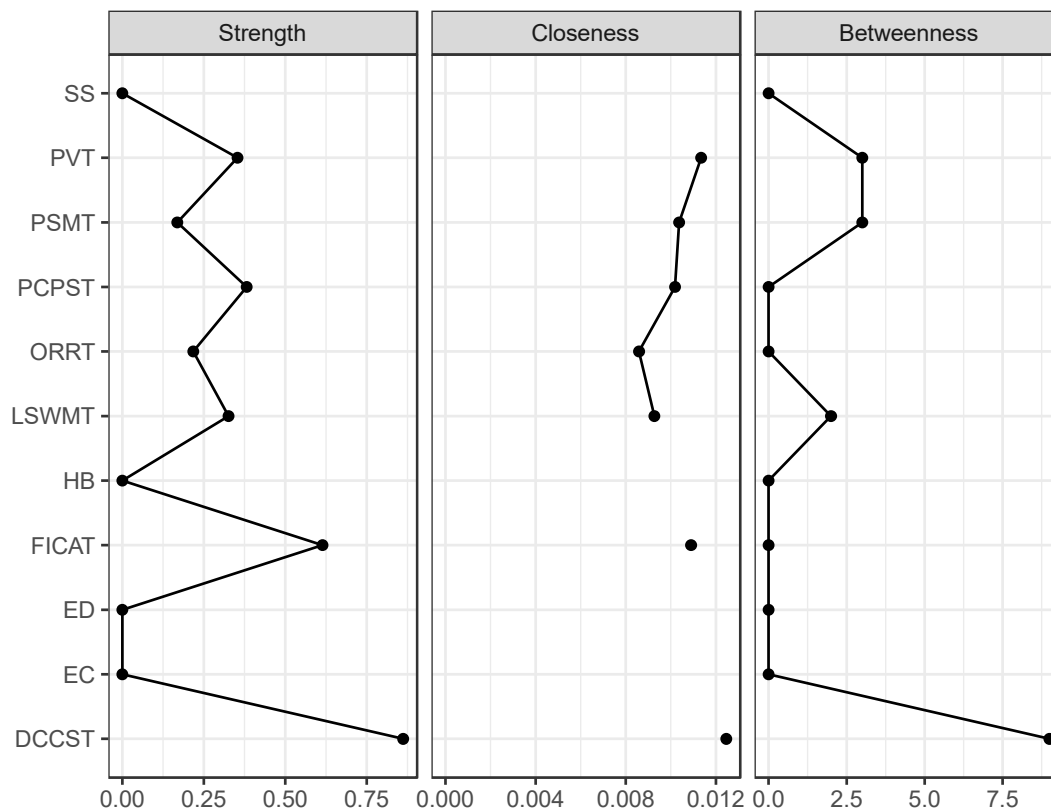
none of the mental health dimensions played an integrative role in the $\gamma = 0.5$ model (see Table 2).

Table 2. Centrality measures for the $\gamma = 0.5$ model.

Node	Strength Centrality	Degree Centrality	Closeness Centrality	Betweenness Centrality	Expected Influence
ED	0.000	0.000	0.000	0	0.000
EC	0.000	0.000	0.000	0	0.000
SS	0.000	0.000	0.000	0	0.000
HB	0.000	0.000	0.000	0	0.000
FICAT	0.615	0.615	0.000	0	0.615
DCCST	0.862	0.862	0.000	18	0.862
PSMT	0.169	0.169	0.000	6	0.169
LSWMT	0.326	0.326	0.000	4	0.326
PCPST	0.382	0.382	0.000	0	0.382
PVT	0.353	0.353	0.000	6	0.353
ORRT	0.218	0.218	0.000	0	0.218

Note. *ED* = Emotional Difficulties. *EC* = Emotional Clarity. *SS* = Social Skills. *HB* = Hyperactive Behaviour. *FICAT* = Flanker Inhibitory Control and Attention Test. *DCCST* = Dimensional Change Card Sort Test. *PSMT* = Picture Sequence Memory Test. *LSWMT* = List Sorting Working Memory Test. *PCPST* = Pattern Comparison Processing Speed Test. *PVT* = Picture Vocabulary Test. *ORRT* = Oral Reading Recognition Test.

Figure 5. Strength, closeness and betweenness centrality plots for the $\gamma = 0.5$ model.



Note. *ED* = Emotional Difficulties. *EC* = Emotional Clarity. *SS* = Social Skills. *HB* = Hyperactive Behaviour. *FICAT* = Flanker Inhibitory Control and Attention Test. *DCCST* = Dimensional Change Card Sort Test. *PSMT* = Picture Sequence Memory Test. *LSWMT* = List Sorting Working Memory Test. *PCPST* = Pattern Comparison Processing Speed Test. *PVT* = Picture Vocabulary Test. *ORRT* = Oral Reading Recognition Test.

Comparison Network Model ($\gamma = 0$)

The centrality measures for the $\gamma = 0$ model displayed relatively more variation compared to the initial ($\gamma = 0.5$) model. Here, *DCCST* and *FICAT* displayed the highest *strength centrality*, followed by *LSWMT*, *PCPST*, and *PVT* (see Table 3). The mental health dimensions displayed relatively low strength centrality values, indicating weak connections within the network. Further, *DCCST* and *FICAT* also demonstrated the highest *degree centrality*, followed by *PVT* and *LSWMT* (see Table 3). To this end, *ORRT*, *PCPST*, and *PSMT* displayed moderate degree centrality, whilst the mental health dimensions displayed the lowest degree centrality values. This is in line with the initial model, where cognitive variables were more central in the network structure, with mental health dimensions being less integrated.

Given that the $\gamma = 0$ network model captured more nuanced connections, *closeness centrality* indices were available. The cognitive dimensions exhibited the highest closeness centrality, with PVT and ORRT emerging as the most central nodes (see Table 3). LSWMT, DCCST, and FICAT also demonstrated relatively high closeness, followed by PSMT and PCPST. ED was the only mental health dimension with relatively high closeness, showing its importance within the network. In contrast, HB, EC, and SS displayed the lowest closeness values, showing that these dimensions were less integrated within the network.

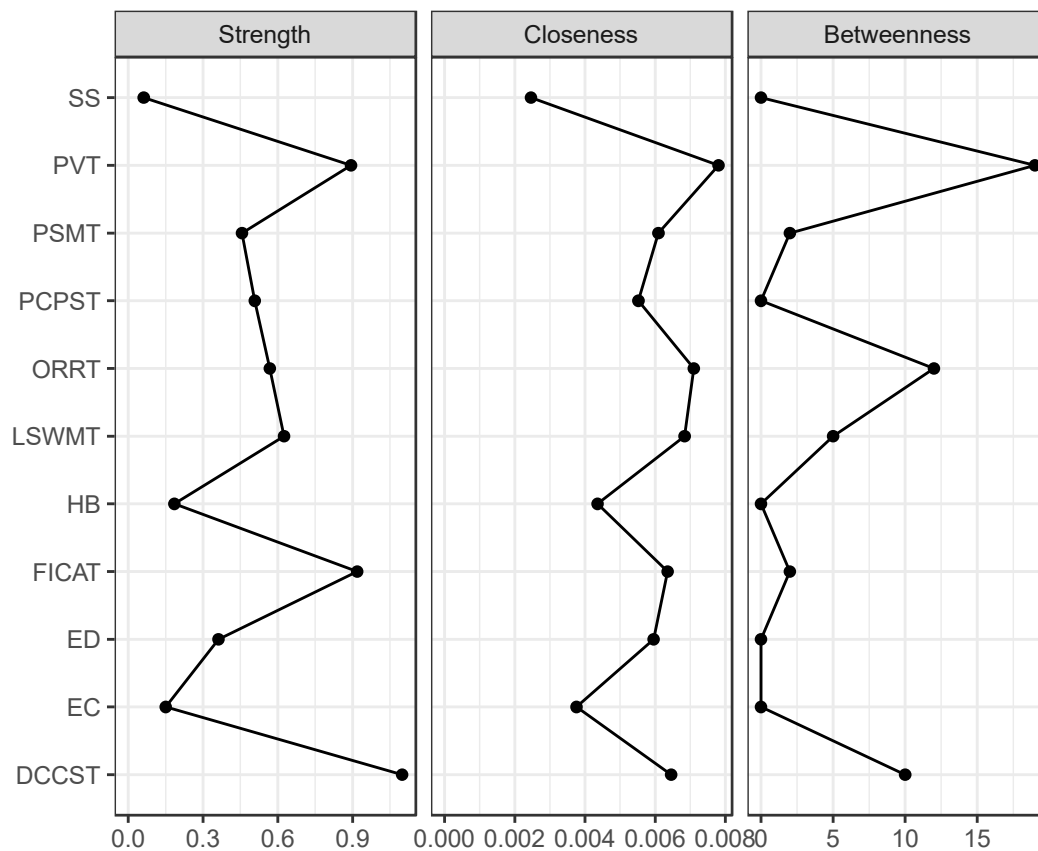
Further, *betweenness centrality* results indicated that PVT served as the ‘central hub’ for the connectivity of this network, followed by ORRT and DCCST (see Table 3). These variables played significant roles in facilitating interactions within the network. Further, LSWMT showed moderate betweenness, indicating that this variable contributed to some information flow, but was not as central as the others, whilst PSMT and FICAT both showed relatively low betweenness scores. PCPST along with the mental health dimensions (ED, EC, SS, HB) did not facilitate any indirect connections between other nodes. Finally, the *expected influence* results in the tuning = 0 network were identical to strength centrality (see Table 3). Thus, expected influence did not add any extra insight to the network structure.

Table 3. Centrality measures for the $\gamma = 0$ model.

Node	Strength Centrality	Degree Centrality	Closeness Centrality	Betweenness Centrality	Expected Influence
ED	-0.035	0.362	0.006	0	-0.035
EC	0.150	0.150	0.004	0	0.150
SS	-0.002	0.062	0.003	0	-0.002
HB	-0.185	0.185	0.004	0	-0.185
FICAT	0.919	0.919	0.006	4	0.919
DCCST	1.099	1.099	0.007	20	1.099
PSMT	0.340	0.457	0.006	4	0.340
LSWMT	0.567	0.625	0.007	10	0.567
PCPST	0.507	0.507	0.006	0	0.507
PVT	0.505	0.894	0.008	38	0.505
ORRT	0.301	0.568	0.007	24	0.301

Note. *ED* = Emotional Difficulties. *EC* = Emotional Clarity. *SS* = Social Skills. *HB* = Hyperactive Behaviour. *FICAT* = Flanker Inhibitory Control and Attention Test. *DCCST* = Dimensional Change Card Sort Test. *PSMT* = Picture Sequence Memory Test. *LSWMT* = List Sorting Working Memory Test. *PCPST* = Pattern Comparison Processing Speed Test. *PVT* = Picture Vocabulary Test. *ORRT* = Oral Reading Recognition Test.

Figure 6. Strength, closeness and betweenness centrality plots for the $\gamma = 0$ model.



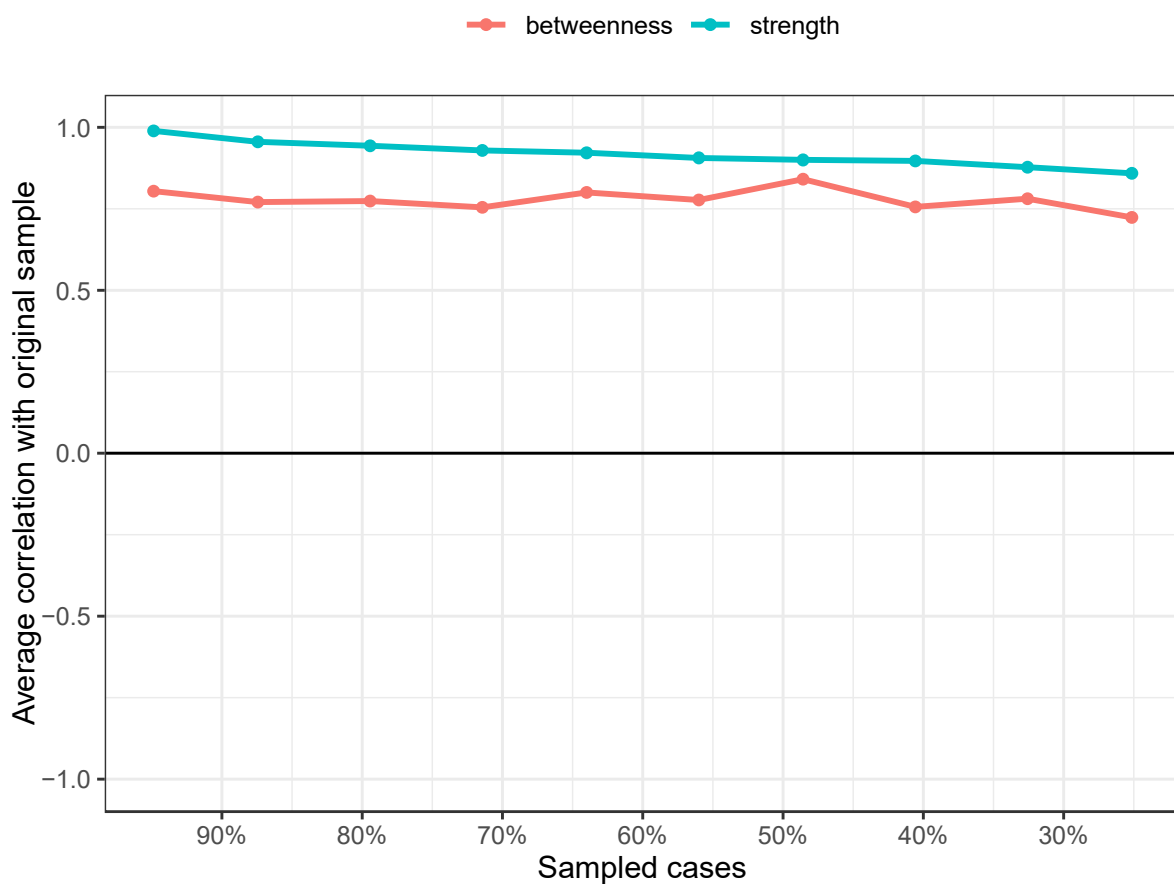
Note. *ED* = Emotional Difficulties. *EC* = Emotional Clarity. *SS* = Social Skills. *HB* = Hyperactive Behaviour. *FICAT* = Flanker Inhibitory Control and Attention Test. *DCCST* = Dimensional Change Card Sort Test. *PSMT* = Picture Sequence Memory Test. *LSWMT* = List Sorting Working Memory Test. *PCPST* = Pattern Comparison Processing Speed Test. *PVT* = Picture Vocabulary Test. *ORRT* = Oral Reading Recognition Test.

5.3.4. Stability of Centrality Measures

Bootstrapped stability tests ($N = 1000$) were performed to assess the robustness of the primary centrality measures (strength, closeness and betweenness) in both network models. In the $\gamma = 0.5$ model, the results indicated that both betweenness and closeness centrality were highly unstable with stability coefficients of zero, as their values fluctuated significantly with case removal. In contrast, strength centrality remained stable when up to 59.4% of the sample was removed, whilst maintaining a correlation of at least 0.7 in 95% of resamples. Since degree centrality and expected influence were identical to strength centrality, it can be inferred that

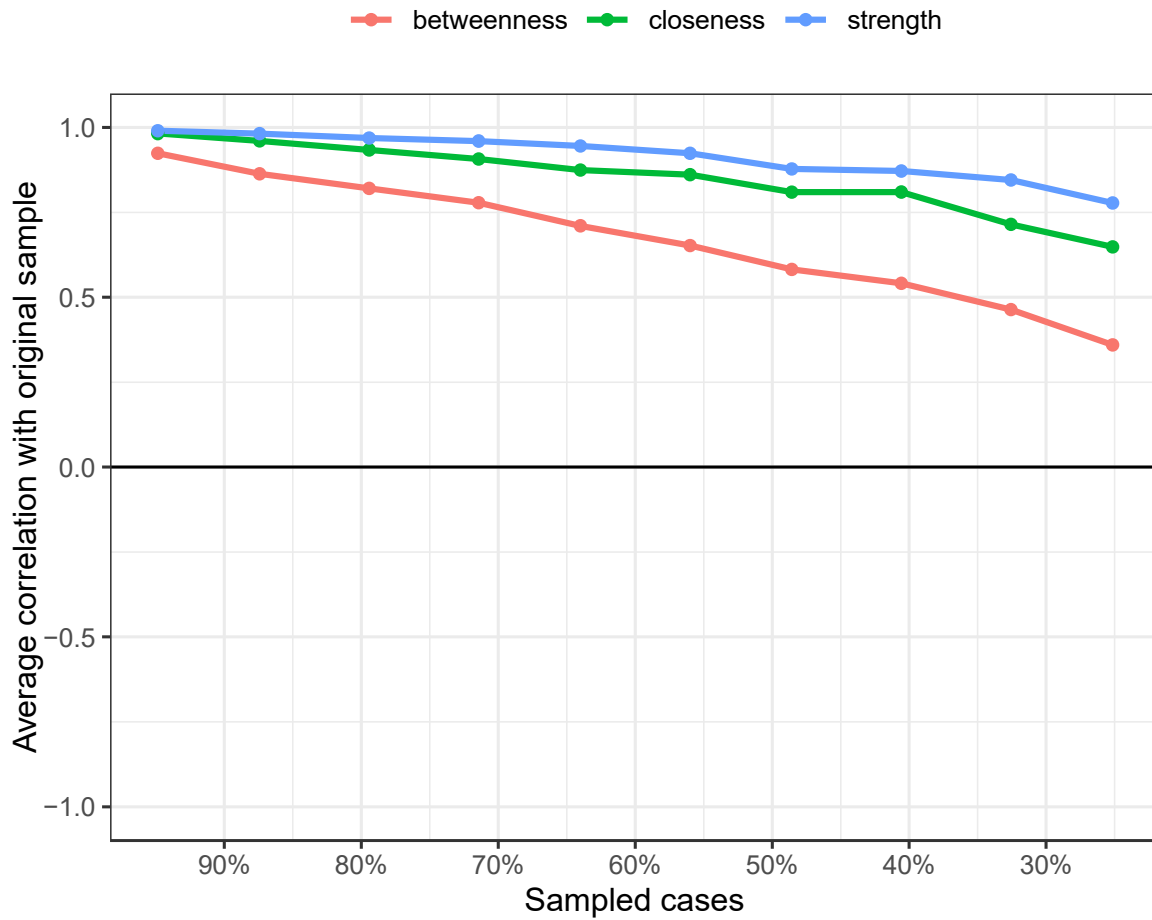
these measures demonstrated a similar level of stability in the $\gamma = 0.5$ model. Further, in the $\gamma = 0$ model, betweenness centrality exhibited a slightly higher stability coefficient of 0.051, suggesting that this measure remained stable with up to 5.1% data drop. Similarly to the initial model, closeness centrality displayed a stability coefficient of zero, suggesting high sensitivity to data variation. Again, strength centrality maintained a correlation of 0.7 in 95% of bootstrapped samples even with up to a 59.4% data drop, demonstrating greater overall stability (compared to betweenness and closeness centrality) in both models. The stability coefficients of the main centrality measures are presented in Figures 7 and 8.

Figure 7. Stability coefficients for betweenness and strength centrality for the $\gamma = 0.5$ model.



Note. Closeness centrality is not displayed because all nodes had a closeness centrality score of zero in this model. Strength centrality values were identical to degree centrality and expected influence.

Figure 8. Stability coefficients for betweenness, closeness and strength centrality ($\gamma = 0$).



Note. Strength centrality values were identical to expected influence.

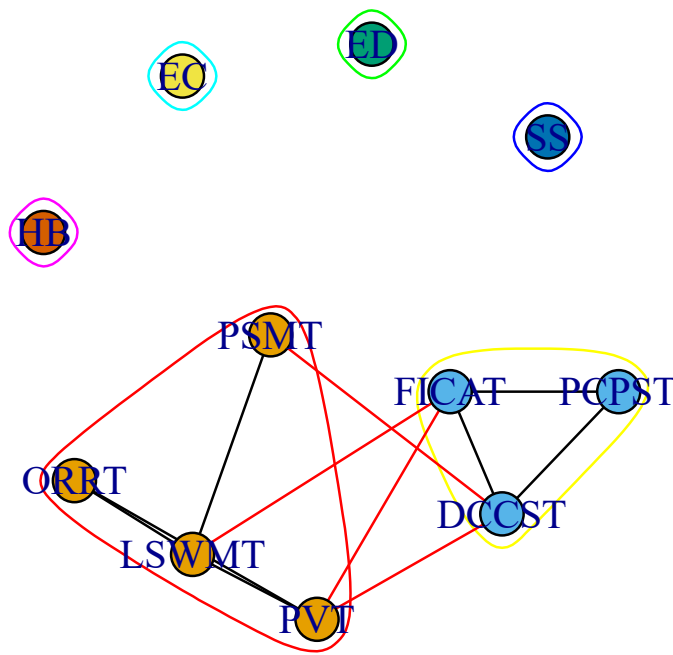
5.3.5. Community Detection

The Walktrap algorithm (Pons & Latapy, 2006) was applied to both network models to identify communities (i.e., clusters) of strongly connected nodes. The algorithm iteratively takes random walks between pairs of nodes to define clusters as closely connected parts of the network, with random walks getting trapped (Mareva et al., 2019). Any negative weights within the $\gamma = 0$ model were transformed such that they became compatible with the algorithm as it does not support negative values. Negative weights were shifted by adding a constant so that they became non-negative, whilst still preserving the relative relationship between edges.

In the initial $\gamma = 0.5$ model, six communities were detected (see Figure 9). Community 1 corresponded to language and memory functions and included LSWMT, PSMT, PVT and ORRT. Community 2 represented a group primarily associated with executive functions

(inhibition, cognitive flexibility) and processing speed, including FICAT, DCCST and PCPST. Interestingly, all mental health variables were dispersed across separate communities, with each constituting their own cluster. However, when the Walktrap algorithm was applied to the $\gamma = 0$ model, the group membership of the mental health variables shifted, such that EC, SS and HB became members of Community 1, whilst ED became a member of Community 2.

Figure 9. Communities identified using the Walktrap algorithm in the $\gamma = 0.5$ model.



Note. Community 1 = Red. Community 2 = Yellow. *EC* = Emotional Clarity. *HB* = Hyperactive Behaviour. *ED* = Emotional Difficulties. *SS* = Social Skills. *PCPST* = Pattern Comparison Processing Speed Test. *DCCST* = Dimensional Change Card Sort Test. *FICAT* = Flanker Inhibitory Control and Attention Test. *PSMT* = Picture Sequence Memory Test. *LSWMT* = List Sorting Working Memory Test. *PVT* = Picture Vocabulary Test. *ORRT* = Oral Reading Recognition Test.

5.3.6. Exploring Bridging Mechanisms

Given that the two main communities were interconnected and not completely distinct (see Figure 9), a bridge centrality analysis was performed to identify variables that may serve as key connectors or ‘bridging mechanisms’ between these groups in both models. The following bridge centrality indices were explored (also presented in Tables 4 and 5):

Bridge Strength reflects the extent to which a node serves as a direct connector between different communities. In the $\gamma = 0.5$ model, all mental health variables (ED, EC, SS and HB) had a bridge strength of zero, indicating that they did not serve as direct connectors within the initial network (see Table 4). Among the cognitive variables, DCCST and PVT demonstrated the highest bridge strength values, suggesting that vocabulary and cognitive flexibility abilities may serve as key connectors between clusters of strengths and weaknesses. Additionally, PSMT and FICAT displayed a weaker, but still present role as connectors between communities, followed by LSWMT. Interestingly, ORRT and PCPST did not serve as connectors, with bridge strengths of zero. In the $\gamma = 0$ model, the results indicated that DCCST, PVT, FICAT, PSMT and LSWMT demonstrated the highest bridge strength values, suggesting that these variables were the strongest direct connectors between the two communities (see Table 5). Notably, ED was the only mental health dimension with a non-zero bridge strength, indicating that it was a weak, yet notable connector. Similarly to the initial model, EC, SS, HB, ORRT and PCPST did not serve as direct connectors.

Bridge Betweenness reflects the extent to which a node lies on the shortest paths connecting different communities. In the $\gamma = 0.5$ model, mental health dimensions (ED, EC, SS, HB), FICAT, LSWMT, PCPST and ORRT exhibited a bridge betweenness of zero, indicating that these processes did not act as intermediaries between communities (see Table 4). In contrast, DCCST, PSMT, and PVT displayed non-zero bridge betweenness, revealing that cognitive flexibility, vocabulary and memory abilities may serve as important bridging mechanisms between communities. Results for the $\gamma = 0$ model indicated that mental health dimensions exhibited a bridge betweenness of zero, indicating that they did not serve as intermediaries. Among the cognitive variables, PVT, FICAT and DCCST displayed the highest bridge betweenness values (see Table 5), revealing that these nodes operated as the key connectors between the two communities. LSWMT was a weaker, but noteworthy, connector between communities.

Bridge Closeness measures how efficiently a node connects different communities. As expected in the $\gamma = 0.5$ model, the mental health dimensions had no bridging connections. Among the cognitive domains, PSMT, PVT, and DCCST demonstrated the highest bridge closeness, suggesting that these nodes were well-positioned to facilitate indirect connections between communities (see Table 4). FICAT, PCPST, ORRT, and LSWMT displayed slightly lower, yet still notable bridge closeness. In the $\gamma = 0$ model, cognitive dimensions generally

demonstrated higher bridge closeness values, with PVT, PSMT, and LSWMT being the most central in facilitating between-community interactions, followed by FICAT, DCCST, ORRT, and PCPST (see Table 5). Mental health dimensions displayed lower bridge closeness, with HB being the lowest, and EC showing the highest value among mental health dimensions.

Bridge Expected Influence assesses how strongly a node impacts other nodes in a different community. In the $\gamma = 0.5$ model, the direct pathways (i.e., 1-step expected influence) mirrored bridge strengths (see Table 4), due to all cross-community edge weights being non-negative values. The 2-step expected influence, which represents indirect effects, further supported the key roles of DCCST, PVT, and PSMT as influential bridging nodes across communities. To this end, FICAT also displayed a high bridge expected influence value, indicating that despite a lack of direct paths across communities, this node still exerted notable influence as a result of its connections with other more influential nodes (e.g., DCCST). Further, LSWMT, PCPST, and ORRT displayed lower expected influence across communities. In the $\gamma = 0$ model, the 1-step expected influence also mirrored bridge strength (see Table 5). Further, DCCST, FICAT, and PVT showed the strongest indirect connections, indicating the highest potential for broader influence across the two communities. Additionally, LSWMT, PSMT, PCPST, and ORRT demonstrated some weaker, but noteworthy influence. Finally, among the mental health dimensions, ED displayed the highest bridge expected influence (although still relatively weak compared to the cognitive variables), followed by EC, SS, and HB.

Table 4. Bridge centrality results for the $\gamma = 0.5$ model.

Node	Bridge Strength	Bridge Betweenness	Bridge Closeness	Direct EI	Indirect EI
ED	0.000	0	0.000	0.000	0.000
EC	0.000	0	0.000	0.000	0.000
SS	0.000	0	0.000	0.000	0.000
HB	0.000	0	0.000	0.000	0.000
FICAT	0.077	0	0.048	0.077	0.161
DCCST	0.148	8	0.054	0.148	0.204
PSMT	0.082	3	0.070	0.082	0.142
LSWMT	0.029	0	0.039	0.029	0.065
PCPST	0.000	0	0.045	0.000	0.049
PVT	0.114	3	0.058	0.114	0.190
ORRT	0.000	0	0.040	0.000	0.017

Note. *ED* = Emotional Difficulties. *EC* = Emotional Clarity. *SS* = Social Skills. *HB* = Hyperactive Behaviour. *FICAT* = Flanker Inhibitory Control and Attention Test. *DCCST* = Dimensional Change Card Sort Test. *PSMT* = Picture Sequence Memory Test. *LSWMT* = List Sorting Working Memory Test. *PCPST* = Pattern Comparison Processing Speed Test. *PVT* = Picture Vocabulary Test. *ORRT* = Oral Reading Recognition Test. *EI* = Expected Influence.

Table 5. Bridge centrality results for the $\gamma = 0$ model.

Node	Bridge Strength	Bridge Betweenness	Bridge Closeness	Direct EI	Indirect EI
ED	0.082	0	0.087	0.082	0.255
EC	0.000	0	0.094	0.000	0.159
SS	0.000	0	0.087	0.000	0.081
HB	0.000	0	0.063	0.000	0.081
FICAT	0.459	11	0.130	0.459	1.450
DCCST	0.671	9	0.127	0.671	1.619
PSMT	0.369	0	0.174	0.369	0.934
LSWMT	0.366	4	0.148	0.366	1.046
PCPST	0.000	0	0.101	0.000	0.458
PVT	0.476	12	0.176	0.476	1.202
ORRT	0.000	0	0.116	0.000	0.278

Note. *ED* = Emotional Difficulties. *EC* = Emotional Clarity. *SS* = Social Skills. *HB* = Hyperactive Behaviour. *FICAT* = Flanker Inhibitory Control and Attention Test. *DCCST* = Dimensional Change Card Sort Test. *PSMT* = Picture Sequence Memory Test. *LSWMT* = List Sorting Working Memory Test. *PCPST* = Pattern Comparison Processing Speed Test. *PVT* = Picture Vocabulary Test. *ORRT* = Oral Reading Recognition Test. *EI* = Expected Influence.

5.3.7. Stability of Bridging Mechanisms

To assess the stability of the bridging mechanisms in both models, the standard deviation (SD) of each node's bridge centrality was calculated across multiple bootstrapped samples ($N = 1000$). In the $\gamma = 0.5$ model, the results indicated that the mental health dimensions (ED, EC, SS and HB) displayed zero variation in bridge centrality ($SD = 0$), suggesting that these nodes remained consistently disconnected across resampling. Additionally, FICAT ($SD = 0.029$), DCCST ($SD = 0.051$), LSWMT ($SD = 0.035$), PCPST ($SD = 0.011$), PSMT ($SD = 0.046$), PVT ($SD = 0.047$) and ORRT ($SD = 0.015$) all displayed low standard deviations, indicating that bridge centrality for all cognitive dimensions showed minimal variability and remained consistent across multiple bootstrap resamples, suggesting high stability.

Moreover, in the $\gamma = 0$ model, ED ($SD = 0.026$), EC ($SD = 0.024$) and SS ($SD = 0.007$) demonstrated low variability, indicating high stability in their bridge centrality values. Additionally, HB had a standard deviation of zero, implying complete robustness in its bridge centrality across resamples. While cognitive dimensions, such as DCCST ($SD = 0.088$), PVT ($SD = 0.081$), FICAT ($SD = 0.077$) and PSMT ($SD = 0.075$) showed somewhat higher variability, the standard deviations remained relatively low, suggesting adequate stability in their bridge roles. To this end, LSWMT showed a slightly lower standard deviation of 0.069, whilst PCPST and ORRT displayed the lowest variability ($SD = 0.042$ and 0.035 , respectively). Overall, the stability analysis suggested that bridge centrality values remained consistent for both mental health and cognitive dimensions across multiple bootstrapped samples in the $\gamma = 0$ model.

5.4. Discussion

This chapter adopted an exploratory network approach to characterising the presumed complex interactions between cognitive and mental health processes in a transdiagnostic sample of neurodivergent adults. Two network models were compared. The initial model applied a regularisation parameter of 0.5 in order to ensure good specificity through including only strong connections. However, this model was ultimately not sensitive enough to detecting weaker (yet still meaningful) connections with mental health dimensions. Therefore, a denser, non-thresholded network model was explored to capture any subtle between-variable relationships.

5.4.1. Key Associations (Edges)

The results revealed that the strongest connections in both network models were between inhibition and cognitive flexibility ($r = 0.43$), as well as cognitive flexibility and processing speed ($r = 0.28$). These results are as expected, given the high level of interdependence between executive functions, and the overlap in cognitive domains assessed by the majority of cognitive tasks, including those in the NIH Toolbox Cognition Battery (Miyake et al., 2000; Zelazo et al., 2014). Similarly, research suggests that variance in task-switching abilities (or cognitive flexibility) can be accounted for by individual differences in processing speed (e.g., Moretti et al., 2018), which indicates a strong relationship between the two processes, and is in line with the current results.

Further, the $\gamma = 0$ model revealed a weaker, yet noteworthy relationship between emotional difficulties and inhibition ($r = 0.12$). This is in line with previous literature suggesting a relationship between response inhibition and emotional difficulties, such as emotion dysregulation (King, 2020), and also supports broader literature suggesting that cognitive difficulties associated with neurodiverse conditions may increase the risk for mental health problems, such as internalising (Baraskewich & McMorris, 2019). The $\gamma = 0$ model also revealed a weaker inverse relationship between emotional difficulties and vocabulary ($r = -0.14$). Since higher scores on the emotional difficulties dimension represent fewer difficulties, the inverse correlation suggests that higher emotional wellbeing may be associated with weaker vocabulary skills. Although the moderate relationship is in line with literature suggesting an association between vocabulary and emotional wellbeing, the results are not as expected, since

increased emotional difficulties have typically been associated with weaker vocabulary (Westrupp et al., 2020; Garwood & McKenna, 2020). Given the relatively small magnitude of this effect, these results should be interpreted with caution.

Additionally, the $\gamma = 0$ model revealed a notable weaker inverse relationship between hyperactive behaviour and reading ($r = -0.10$). Since high scores on the hyperactive behaviour dimension represent fewer difficulties, the inverse relationship indicates that hyperactive behaviour may be associated with stronger reading skills. This is surprising, given the high co-occurrence rates of ADHD and reading difficulties (DuPaul et al., 2013), and not in line with broader literature suggesting that ADHD traits (such as hyperactivity) may increase the risk of academic difficulties in many areas, including reading (e.g., Sunde et al., 2022). Nonetheless, these results should also be interpreted with caution, given that the relatively small magnitude of effect suggests a minimal inverse relationship between the two dimensions.

Although the non-thresholded network model enabled for the identification of more subtle connections due to increased sensitivity, it required a correlation threshold of 0.67 for the edges to remain stable. Since the abovementioned relationships were much weaker, the stability analysis suggests that such unexpected results would not hold with a reduction in sample size. Similarly, the $\gamma = 0.5$ network model required a correlation threshold of 0.59 for its edges to remain stable. Although this threshold was lower than that required for the $\gamma = 0$ model, none of the edges approached this correlation, and thus this network model also demonstrated relative instability. These results suggest that the current sample size may be too small, and therefore there may not be enough consistent structure within the dataset. Consequently, individual edge interpretations should be treated with caution in both network models, regardless of the magnitude of effect, whilst node centrality indices may be used to provide a more robust interpretation of the overall network structure (see below).

5.4.2. Key Processes (Nodes)

Results of the centrality measures revealed that the $\gamma = 0.5$ model was structurally simple, as centrality rankings were driven purely by the number of connections between nodes, rather than the strength of these connections. Such findings imply that the $\gamma = 0.5$ model lacked central ‘hubs’, where certain nodes could exert significantly more influence due to high connection strength. Therefore, degree centrality alone was used as a proxy for influence in this model, as

it reflects the number of direct connections a node has in terms of outgoing and incoming links (Evans & Chen, 2022). To this end, cognitive flexibility and inhibitory control emerged as the most connected nodes, whilst cognitive flexibility also demonstrated the highest betweenness centrality, therefore acting as a key intermediary node within this network. Similar results were observed for the $\gamma = 0$ model, where the central roles of cognitive flexibility and inhibitory control were confirmed by strength centrality indices, suggesting that these dimensions were the most influential in the current dataset. Interestingly, vocabulary also served as the ‘central hub’ for the connectivity of the $\gamma = 0$ network model, based on betweenness and closeness centrality indices. Conversely, mental health dimensions were either very weakly connected (as seen in the $\gamma = 0$ model), or completely disconnected ($\gamma = 0.5$ model) from each other and/or from other nodes within the network.

The findings suggest that cognitive flexibility and inhibitory control are highly influential processes in the cognitive functioning of neurodivergent adults. This is in line with a large body of evidence suggesting that executive function differences underpin a broad range of neurodiverse conditions, and often contribute to individual variability and/or between-diagnosis overlaps (Sadozai et al., 2024). For instance, individual differences in inhibitory functioning have been shown to underpin some of the variability in ADHD presentations (e.g., Adams et al., 2008). Since the current results suggest that differences in inhibitory control are likely to be associated with changes in other dimensions within the network, this study provides support for the notion that difficulties with inhibition may have cascading effects across multiple domains of functioning, and thus act as a potential mechanism underpinning the heterogeneous profiles of neurodivergent adults.

Further, cognitive flexibility was found to be the key ‘mediator’ in the initial network model, suggesting it is not only the most influential dimension, but may also play a central role in facilitating communication between other cognitive processes. Similarly, cognitive flexibility was one of the top three ‘central hubs’ for the connectivity of the $\gamma = 0$ network model. These findings are in line with research proposing cognitive flexibility to be the ‘pinnacle’ of cognitive control, exerting overarching influence across all other cognitive processes (Braem & Egner, 2019). The key intermediary role of cognitive flexibility is especially important in the context of the $\gamma = 0$ model, where connections with mental health were evident (although subtle), since cognitive flexibility has been considered as a transdiagnostic risk factor for the development of co-occurring internalising and externalising problems in neurodivergent

individuals (Lei et al., 2022). This suggests that the current study provides further empirical evidence for the importance of targeting cognitive flexibility difficulties in neurodivergent individuals, as it may have wider beneficial effects not only for improving other cognitive processes, but also for targeting co-occurring mental health difficulties and/or decreasing susceptibility to their co-development.

Additionally, given that the working memory is also a key component of executive function (Miyake et al., 2000), it is surprising that it was not identified as an influential node in the initial $\gamma = 0.5$ model. However, it was the third most influential node in the $\gamma = 0$ network, based on strength centrality indices. This is in line with research suggesting that working memory difficulties may underpin a wide range of cognitive problems in children with suspected special educational needs, including difficulties in other executive functions, such as sustained attention (Holmes et al., 2010). The current study therefore builds on such findings, providing evidence for the influential role of working memory on other cognitive processes in a broad sample of neurodivergent adults. Together, results of the centrality measures provide a strong case for the importance of executive functions in overall cognitive and/or mental health functioning of neurodivergent adults. This supports previous data-driven findings from transdiagnostic studies with neurodivergent children, where different executive function profiles have been shown to be related to differences in academic achievement (e.g., reading) and mental health (e.g., internalising) outcomes (Mareva et al., 2024).

Although the connections between mental health and cognitive dimensions were relatively weak and unstable (even when applying no regularisation parameter), the aim of this network analysis was to identify subtle interactions, which may have been overlooked in previous analyses (presented in Chapter 4). To this end, the emotional difficulties node was the most noteworthy, demonstrating the highest connectivity based on degree and closeness centrality measures. These results suggest that differences in emotion regulation were most often associated with differences in cognitive processing, among the mental health dimensions entered into the analysis. This is in line with research suggesting a direct association between emotion regulation and cognitive processes, such as cognitive control or cognitive flexibility (Rónai et al., 2024; Ghosh & Halder, 2020). Since emotion regulation has been proposed to be a transdiagnostic mechanism underpinning the co-occurrence of neurodiverse conditions (England-Mason, 2020), the current results provide further support for the notion that emotion regulation strategies could be a potential target for future transdiagnostic interventions. This is

especially important since emotional difficulties have also been found to mediate the relationship between cognition and mental health problems (specifically internalising and externalising) in neurodivergent populations (Tajik-Parvinchi et al., 2021), suggesting that, similarly to cognitive flexibility (see above), targeting emotion regulation processes may have beneficial effects for a wide range of difficulties associated with neurodiverse conditions, including co-occurring mental health problems.

Finally, vocabulary appeared to play a key role in facilitating between-variable interactions in the non-thresholded network model, followed by reading and cognitive flexibility. The central intermediary role of language processes was unanticipated, but may be a reflection of the sample demographics. A potential explanation for the unexpected results is that the role of language processes may have been exacerbated in a sample predominantly consisting of neurodivergent university students (particularly those with diagnosed or suspected reading difficulties). Research has shown a relationship between the adoption of compensatory strategies supported by general cognitive skills and the improvement of reading performance in dyslexic university students (Faisca et al., 2023). This suggests that language processes may well have played a central role in facilitating connections between other cognitive processes within this network. Further, a large proportion of the sample were bilingual (see Chapter 3). To this end, research has repeatedly provided evidence for a ‘bilingual advantage’ in non-linguistic cognitive processes, such as cognitive flexibility, resulting from the frequent switching between languages (e.g., Bialystok, 2017). This could suggest that a large proportion of the current sample may have relied on relative strengths in language processes to account for weaknesses in other, non-linguistic cognitive domains, potentially shedding light on the prominent role of vocabulary and reading skills within this network.

Nonetheless, the above findings should be interpreted with caution, given that the stability of the betweenness and closeness centrality indices in the $\gamma = 0$ model was highly sensitive to data variation. Conversely, strength centrality results were relatively stable in both network models. These findings indicate that, among all centrality metrics, the strength-based associations involving executive functions (particularly cognitive flexibility and inhibitory control) were the most robust, reinforcing their central role in the cognitive and mental health network of the current transdiagnostic dataset. However, whilst strength centrality highlights the most strongly connected nodes, it doesn’t offer a deeper understanding of the mechanisms through which executive functions may contribute to the overall network dynamics. Further insights into the

network structure were gained through community detection and bridge centrality analysis to allow for the identification of functionally distinct communities within the network and the key nodes that link them (see below).

5.4.3. Communities and Key Bridging Mechanisms

Results of the Walktrap community detection revealed two distinct communities. The first community corresponded to language and memory processes, whilst the second community consisted of executive function and processing speed abilities. In the initial model, each mental health dimension constituted its own community. In the $\gamma = 0$ network model, emotional clarity, social skills and hyperactive behaviour became members of Community 1 (language and memory), whilst the emotional difficulties dimension joined Community 2 (executive function and processing speed). Interestingly, the communities identified in the network analysis corresponded closely to the discriminant dimensions that best differentiated between data-driven clusters in Chapter 4. Dimension 1 was primarily defined by a combination of cognitive flexibility and processing speed abilities (aligning with Community 2), whilst Dimension 2 was best defined by a combination of language and memory skills (aligning with Community 1), with emotional difficulties and hyperactive behaviour also significantly contributing to this dimension. These findings suggest that the underlying structure of the associations among variables, as captured by network communities, reflected very similar latent dimensions that distinguished between naturally occurring groups within the sample. This provides converging evidence for the robustness of the cognitive and mental health patterns across different analytical approaches.

The above findings indicate a clear separation between language/memory and executive function/processing speed domains. This is interesting, since the results demonstrate that Community 1 was a combination of both fluid and crystallised cognitive abilities, whilst Community 2 consisted of purely fluid cognition processes. In the NIH Toolbox Cognition Battery, the crystallised cognition composite reflects scores from tasks measuring language skills (i.e., vocabulary and reading decoding), whilst the fluid cognition composite reflects scores from tasks assessing executive function (i.e., inhibition, cognitive flexibility and working memory), episodic memory and processing speed (Weintraub et al., 2014). The current results therefore indicate that this organisation of cognitive functions did not appear to hold in the current dataset. This suggests that neurodivergent adults may display unique cognitive

profiles, in which specific fluid and crystallised cognitive abilities are interdependent. For instance, individuals may rely heavily on fluid cognitive skills (e.g., episodic or working memory) for the completion of reading or vocabulary tasks, resulting in stronger associations between crystallised and fluid cognitive domains within the network. This is in line with research evidence suggesting that neurodivergent adults may develop strategies to compensate for their weaknesses through leveraging areas of strength (e.g., Canela et al., 2017; Livingston et al., 2020). In particular, dyslexic individuals have been shown to harness strengths in declarative memory to compensate for difficulties with phonological decoding (Ullman & Pullman, 2015). Since a large proportion of the current sample have been referred for suspected difficulties with reading (see Chapter 3), this could potentially explain the observed interactions between language and memory processes in this dataset.

Nonetheless, the strong associations between language and memory abilities are not surprising, given that their relationship has been well-established in previous literature. For instance, working memory capacity has previously been linked with reading skills in school-aged children (Gathercole et al., 2003). Similarly, a large body of evidence has demonstrated the implication of working memory in early reading acquisition and later reading performance (Peng et al., 2018). Working memory training has also proved to be an effective strategy in improving reading skills in children (Karchach et al., 2014). Therefore, the structure of Community 1 is in line with broader literature, and suggests that the strong relationship between memory and language skills is not only observable in child populations, but may also hold in adult samples. This may have implications for future support strategies aimed at improving language (e.g., reading, vocabulary) skills in neurodivergent adults.

Further, Community 2 consisted of purely fluid cognitive abilities (cognitive flexibility, inhibitory control, and processing speed). It is not surprising that these domains were tightly interconnected, since research has shown strong associations between executive function and processing speed (e.g., McAuley & White, 2011). This community structure indicates that slowed processing speed may be closely related to difficulties in executive function, supporting previous research suggesting that processing speed often accounts for performance variability in executive function tasks (Rose et al., 2011). Interestingly, working memory was not a member of this community, despite being a core component of executive function (Miyake et al., 2000). This dissociation between different aspects of executive function may be unique to neurodivergent adults, with research suggesting that differences in executive function may

contribute to heterogeneity across a wide range of neurodiverse conditions (Sonuga-Barke et al., 2010; Geurts et al., 2014; Doyle et al., 2018). The current results therefore provide further data-driven insights into the heterogeneous nature of executive function in neurodivergent adults, through identifying distinct patterns of between-variable interactions.

Interestingly, emotional difficulties were closely associated with language and memory skills in Chapter 4, yet more closely related to executive function and processing speed through Walktrap community detection (in the $\gamma = 0$ model). Since emotion regulation has been proposed as a transdiagnostic mechanism underpinning the co-occurrence of neurodiverse conditions (England-Mason, 2020), it is important to consider its role in connecting the two communities through bridge centrality measures. To this end, emotional difficulties were the only mental health dimension with a non-zero bridge strength, highlighting its role in the overall network structure as a potential ‘bridging mechanism’. This suggests that emotion regulation may mediate the relationship between language/memory and executive function/processing speed abilities. For instance, if an individual is characterised by a profile of difficulties in executive function and processing speed, they may be more susceptible to developing further weaknesses in language and/or memory functioning, if they also experience co-occurring emotional difficulties. These findings emphasise the importance of targeting mental health processes in improving individual wellbeing in neurodivergent adults, as processes such as emotion regulation may be relevant to a wide range of outcomes across conditions.

Finally, cognitive flexibility and vocabulary were identified as the most influential cognitive ‘bridging mechanisms’, demonstrating the highest bridge strength across both network models. This further emphasises their central roles within the network as described in Section 5.4.2. above. Together, these results provide a strong case for the notion that utilising cognitive flexibility and vocabulary as targets for intervention strategies or individualised support may have widespread benefits for multiple domains of cognitive functioning. This is in line with extant research utilising cognitive flexibility training to improve a number of functional outcomes in autistic children, including social interaction and communication skills (Varanda & Fernandes, 2017). Thus, the current findings suggest that similar methods could be applicable across a range of neurodiverse conditions in adults. Further, interventions targeting vocabulary skills have typically been used in the context of language and reading difficulties in children (e.g., Steele & Mills, 2011; Marulis et al., 2010). The current results could suggest

that the role of vocabulary skills may be more widespread in neurodivergent adults, exerting influence across broader, non-linguistic cognitive domains. Such interactions could be associated with the well-established benefits of language learning on overall cognitive functioning in adults, resulting from strengthened neural connections or enhanced neuroplasticity (Ware et al., 2021). However, although stability analyses suggested that bridge centrality results remained consistent and robust across both models, the current results should be interpreted with caution, given the exploratory nature of this network analysis.

5.4.4. Strengths and Limitations

This chapter has several strengths. First, the use of network analysis has enabled the exploration of more subtle between-variable interactions, addressing some of the limitations of Chapter 4. Whilst cluster analysis allowed for the identification of naturally occurring groups within the present dataset, and demonstrated that individual profiles did not directly map onto traditional diagnostic categories, the network analysis provided further valuable insights into the processes that may have contributed to the formation of such transdiagnostic clusters. For instance, cognitive flexibility, inhibitory control, and vocabulary were found to exert the most influence across the network and/or facilitate communication between cognitive and mental health dimensions. This suggested that variability in these processes may play a key role in shaping individual profiles of functioning across a wide range of NDs, or potentially contribute to the co-occurrence of difficulties. Moreover, this chapter has highlighted the meaningful role of emotion regulation as a potential bridging mechanism that may underpin the development of co-occurring mental health difficulties in neurodivergent adults. Thus, this chapter has built on the results presented in Chapter 4, and allowed for a more nuanced understanding of the processes that may contribute to the complexity of NDs in adults. Ultimately, the findings of this chapter have the potential to inform future support strategies, which may be relevant across a broad range of NDs.

Additionally, this chapter contributes meaningfully to the emerging pool of data-driven accounts of NDs, building on existing applications of network analysis within the field of neurodiversity research. Specifically, the present analysis examined both cognitive and mental health variables in a transdiagnostic sample of neurodivergent adults. Such an integrative approach has been applied in previous work, but was typically focused on child samples. For instance, Mareva and colleagues (2024) used self-organising maps and *k*-means clustering to

identify data-driven profiles of executive function in neurodivergent children, and explored their associations with internalising symptoms. Similarly, Zhou and colleagues (2023) used network analysis to investigate the relationship between ADHD symptoms, mood difficulties, and cognitive functioning in children. These studies demonstrate a growing interest in mapping the interplay between cognition and mental health in neurodiverse populations. However, to our knowledge, the present study is the first to apply a network approach to both domains in a transdiagnostic sample of neurodivergent adults. This represents a key strength of the chapter, as it advances the existing literature in an unexplored area.

However, this chapter is not without limitations. First, it is important to consider that network models are constructed based on between-variable associations, and therefore causality cannot be inferred from such networks (Epskamp & Fried, 2018). For instance, a strong edge may represent a meaningful relationship between two processes, but the interpretability of this association is limited. This is especially important to consider in the context of identifying targets for future support, as understanding which difficulty may precede and/or influence the other may be crucial for guiding effective support strategies (e.g., Masten & Cicchetti, 2010). Nonetheless, network analysis offers valuable insights by identifying the most influential or central processes that may activate difficulties in other areas (Mareva et al., 2019). Targeting these key processes may lead to cascading beneficial effects across a wide range of difficulties (e.g., Astle et al., 2022), making network analysis a useful tool for highlighting potential targets for support strategies, even when causal directionality cannot be established.

Further, network models require relatively large samples for stable and interpretable results (Epskamp et al., 2018). Given that this study's sample size was established to ensure sufficient power for meaningful subgrouping in the initial cluster analysis (Dalmaijer et al., 2022), the present sample may not have been adequate for the network analysis. It has recently been suggested that standard psychological sample sizes are often insufficient for accurately estimating complex network structures, particularly those with many nodes (Epskamp et al., 2018). This is especially important to consider in the context of non-regularised network models, which are likely to overfit or yield unstable connections when estimated from a small sample size (Epskamp et al., 2017). This limitation has in fact been reflected in the relative instability of edge weights and main centrality indices in the present network models, particularly in the $\gamma = 0$ model. This suggests that the robustness and generalisability of the

present findings may have been compromised as a result of a small sample size relative to the number of variables entered into the analysis.

In order to increase the stability and reliability of the estimated networks using the current dataset, a network model without the inclusion of mental health dimensions could have been explored. This would have decreased the sample size-to-variable ratio, potentially enhancing the robustness of the estimated edges. Additionally, a more focused cognitive network may have allowed for a clearer interpretation of within-domain relationships (e.g., among executive functions), which may have been weakened in the full network due to cross-domain interactions between cognitive and mental health variables. Nonetheless, the inclusion of both cognitive and mental health dimensions allowed for an exploration of the complex interplay between these processes. While potentially reducing model sparsity, this broader approach aligned with this chapter's aims to explore more subtle associations between cognition and mental health, thus providing valuable (although preliminary) insights, beyond that offered by the initial cluster analysis in Chapter 4.

Finally, given the cross-sectional nature of this dataset, the present network analysis allowed for an understanding of the associations between cognitive and mental health dimensions within a static network. However, it did not have the capacity to capture the ways in which such between-variable relationships may change over time. These insights would have been particularly useful for tracking individual wellbeing in the context of neurodiverse conditions, which have been shown to present differently across different developmental stages (e.g., Antolini & Colizzi, 2023). To this end, dynamic network modelling on longitudinal data could allow for an exploration of the changing patterns and structures that emerge through between-variable interactions over time; for instance, by demonstrating changes in the values of nodes, or the strength of the connections (i.e., edges) between these nodes (Den Hartigh et al., 2016). This could offer insights into the developmental trajectories of cognitive and mental health functioning across the lifespan. Such insights could potentially help to identify early indicators of change or risk, and/or inform the development of early support strategies to promote individual wellbeing from childhood to adulthood.

5.4.5. Conclusions and Future Directions

In conclusion, this chapter has provided valuable insights into the complex interactions between cognitive and mental health dimensions characterising the current sample of neurodivergent adults, and highlighted several influential processes which may be relevant to a wide range of NDs, and/or serve as potential targets for future transdiagnostic support strategies. However, given the exploratory nature of the results, further research is needed to validate these preliminary insights and establish their generalisability beyond the current sample. Future research should focus on recruiting larger transdiagnostic samples of adults to ensure sufficient power for detecting more robust patterns, thus increasing the reliability of findings. Future research may also focus on collecting longitudinal data to capture shifts in between-variable relationships over time through dynamic network modelling, offering the opportunity to track individual wellbeing across the lifespan. Such an approach may also shed light on the temporal relationships between patterns of difficulties, which could be beneficial for providing targets for early support strategies.

Chapter 6: General Discussion

This chapter will provide an overview and discussion of the findings presented in this thesis. First, the primary aims of the thesis and each empirical chapter will be summarised (Section 6.1.), followed by a summary and discussion of key findings, including their theoretical and practical implications (Sections 6.2. to 6.3.). Next, the methodological strengths and limitations of the systematic review and transdiagnostic study will be discussed (Section 6.4.). Finally, recommendations for future research will be presented (Section 6.5.), and an overall conclusion will be drawn (Section 6.6.).

6.1. Thesis Aims

The primary aim of this thesis was to gain a deeper understanding of the complexity associated with neurodiverse diagnoses (NDs) in adults. First, a systematic review (Chapter 2) was conducted in order to explore whether the prevalence rates and cognitive manifestations of co-occurring Attention Deficit Hyperactivity Disorder (ADHD) and Reading Difficulties (RD) change from childhood to adulthood. This chapter provided additional rationale for the need for further research into the cognitive mechanisms that may contribute to the co-occurrence of neurodiverse conditions, specifically in adults. Second, a transdiagnostic study protocol (described in Chapter 3) was developed in order to address some of the shortcomings of categorical research. The aim of this study was to yield a more comprehensive data-driven account of the cognitive and mental health functioning of neurodivergent adults. To this end, two complementary data analysis techniques were applied. First, a cluster analysis (Chapter 4) was performed with the aim to explore naturally occurring groups within the transdiagnostic dataset, the ways in which these groups may differ on key cognitive and mental health dimensions, and whether they directly map onto traditional diagnostic categories. Second, a network analysis (Chapter 5) was conducted with the aim to further explore the complex interactions between cognitive and mental health dimensions, and to identify the most influential processes, as well as communities of functions and the mechanisms that may facilitate communication between them. Together, these analyses enabled for the exploration of potential cognitive and mental health mechanisms that may help to characterise the complexity of NDs in adults (in terms of individual variation, between-diagnosis overlaps, and/or co-occurrence of conditions). The key findings of each chapter are summarised and discussed in Sections 6.2. and 6.3.

6.2. Summary of Key Chapter Findings

6.2.1. Systematic Review and Meta-Analysis

Chapter 2 consisted of a systematic review, which aimed to explore the prevalence rates and cognitive manifestations of co-occurring ADHD+RD across development. The results of this chapter showed that the co-occurrence of ADHD+RD did not differ between children and adults, with both age groups displaying co-occurrence rates of approximately 23%. The overall co-occurrence estimates were significantly lower in studies employing more inclusive recruitment methods (e.g., broad sampling from schools/communities), and significantly higher in studies where ‘purposive’ sampling (e.g., recruitment based on clinical referrals) was employed. The model used to define RD (e.g., ‘discrepancy’ vs. ‘low achievement’) did not have a significant effect on overall co-occurrence estimates. Further, the results showed that both children and adults with co-occurring ADHD+RD demonstrated significant difficulties in overall cognitive function (as assessed by measures of attention, cognitive flexibility, inhibition, planning, problem solving, processing speed, vocabulary, working memory and general memory), when compared with typically developing controls. There were no significant differences between child and adult cognitive function in any of the above domains.

6.2.2. Transdiagnostic Study: Cluster Analysis

Chapter 4 comprised a cluster analysis, which aimed to explore the underlying structure of the transdiagnostic dataset through identifying naturally occurring data-driven groups. The results of this chapter showed that the current sample of neurodivergent adults consisted of three transdiagnostic groups, which were characterised by specific combinations of strengths and difficulties along the following dimensions: Dimension 1 (defined by cognitive flexibility and processing speed abilities), and Dimension 2 (defined by language, memory, emotional difficulties and hyperactive behaviour). Cluster 1 was best defined by Dimension 2, and characterised by weak language and working memory abilities, with few emotional difficulties/low hyperactive behaviour. Cluster 2 was best defined by Dimension 1, displaying the most ‘severe’ cognitive profile, with weak cognitive flexibility and processing speed. Cluster 3 was best defined by both dimensions, displaying the strongest cognitive profile (strong cognitive flexibility, processing speed, language and working memory abilities), and a

weak mental health profile (with high emotional difficulties and hyperactive behaviour) relative to the other clusters. Together, the data-driven groups did not directly map onto traditional diagnostic categories, since all groups encompassed a combination of formal diagnostic labels, individuals with mild difficulties and/or suspected diagnoses, as well as a small proportion of typically developing adults.

6.2.3. Transdiagnostic Study: Network Analysis

Chapter 5 aimed to build on the analyses presented in Chapter 4, through adopting a network analysis approach in order to gain further insight into the complex interactions between the cognitive and mental health processes characterising the current dataset. The results of this chapter highlighted cognitive flexibility, inhibitory control and vocabulary skills as the most central processes in facilitating communication between cognitive and mental health dimensions within the network. Further, two distinct communities of functions were identified, with Community 1 primarily corresponding to language and memory processes (along with emotional clarity, social skills, and hyperactive behaviour), and Community 2 primarily corresponding to executive function and processing speed abilities (along with emotional difficulties). To this end, cognitive flexibility and vocabulary were identified as key ‘bridging mechanisms’, emphasising their important role in facilitating information flow between the two communities and the overall network structure. Finally, emotion regulation was highlighted as the most noteworthy mental health dimension within the network, with its subtle yet meaningful role in bridging the two identified communities.

6.3. Discussion of Findings

The overarching aim of this thesis was to gain a more comprehensive understanding of the complexity associated with the scope and nature of neurodiverse conditions in adults. The present results support recent transdiagnostic literature suggesting that current diagnostic systems do not adequately capture the underlying nature of NDs (Astle et al., 2022), and build on the emerging quantitative accounts of neurodiversity (e.g., Holmes et al., 2021; Bathelt et al., 2018; Astle et al., 2019), through providing a data-driven exploration of the broad cognitive and mental health dimensions that may be relevant across a wide range of NDs. Overall, this thesis contributes to a broader understanding of the cognitive and mental health functioning of neurodivergent adults, offering valuable insights with the capacity to address current challenges

in characterising and identifying neurodiverse conditions in adulthood (e.g., Sadusky et al., 2021; Lipinski et al., 2022; Goodman et al., 2024). The following section provides a discussion of how the current findings a) support the transdiagnostic dimensional approach, b) challenge the traditional categorical structure of NDs, and c) contribute to novel data-driven accounts of cognitive and mental health functioning in neurodivergent adults.

6.3.1. Transdiagnostic Patterns and Heterogeneity

The findings from this thesis support the notion that the complex, heterogeneous nature of neurodiverse diagnoses may be better understood through a transdiagnostic lens (Astle et al., 2022). First, the systematic review in Chapter 2 revealed that the co-occurrence of ADHD with RD remained high across the lifespan, suggesting that the two conditions may not be entirely distinct as proposed by traditional diagnostic categories. This implies that there may be shared (i.e., transdiagnostic) mechanisms or overlapping developmental pathways contributing to the frequent co-development of ADHD+RD that persists across the lifespan. Further, findings from Chapter 2 also showed that both children and adults with ADHD+RD displayed significantly weaker cognitive functioning when compared with ‘neurotypical’ controls, yet with substantially high levels of heterogeneity in cognitive scores observed across studies. Although it was not feasible to investigate whether this heterogeneity could be attributable to methodological differences between studies, the findings suggest that the cognitive profiles of children and adults with co-occurring ADHD+RD are highly variable. Since both ADHD and RD have been associated with complex and heterogeneous cognitive profiles (Vos et al., 2022; Chalmpé & Vlachos, 2025), the results suggest that the co-occurrence of ADHD and RD may reflect an even greater degree of cognitive variability, reinforcing the notion that fixed diagnostic categories may not adequately capture the breadth of individual differences across neurodiverse conditions.

Second, results from Chapter 4 (cluster analysis) showed that the current dataset of neurodivergent adults was best classified into three data-driven clusters. Importantly, all clusters were transdiagnostic, in that they all comprised at least three diagnostic labels, as well as individuals with suspected neurodiverse conditions, and a small proportion of neurotypical participants. Thus, in line with previous transdiagnostic research (e.g., Mareva et al., 2024), the present data-driven groups did not directly map onto traditional diagnostic categories. These findings suggest that the profiles of neurodivergent adults may be better characterised by

patterns of processes that cut across diagnostic boundaries, rather than a strict set of criteria associated with a given diagnosis. Additionally, since the majority of primary diagnostic labels were dispersed across the three data-driven groups, with each of these groups being characterised by their own combination of strengths and weaknesses in cognition and mental health, the results from Chapter 4 also provided further evidence for the heterogeneous nature of NDs, and specifically the high levels of individual variation in current diagnostic categories, and potential overlap between them.

Further, findings from Chapter 5 (network analysis) provided additional evidence for the role of transdiagnostic mechanisms in characterising the complexity and heterogeneity associated with neurodiverse conditions. In this chapter, several cognitive functions emerged as potential shared mechanisms through which NDs may co-occur, whilst the results also highlighted some influential processes that may contribute to individual variation. For instance, cognitive flexibility, inhibitory control and vocabulary skills were identified as the ‘central hubs’ of the transdiagnostic network, suggesting that variability in these processes may play a key role in shaping individual profiles, potentially driving individual variation in cognitive and/or mental health functioning in neurodivergent adults. Similarly, cognitive flexibility and vocabulary were highlighted as key ‘bridging mechanisms’, facilitating communication between two communities of more closely related functions (corresponding to executive function/processing speed, and language/memory), highlighting these processes as potential mechanisms underpinning the co-occurrence of difficulties in the current transdiagnostic sample. These findings build on recent data-driven, transdiagnostic accounts of neurodiversity, where similar cognitive domains (e.g., executive functions and language skills) have been proposed to aid in the characterisation of individual variation and potential origins of co-occurring conditions in children with learning difficulties (Holmes et al., 2021; Mareva et al., 2019).

Taken together, the findings from Chapters 2, 4 and 5 underscore the importance of adopting a transdiagnostic approach to exploring the heterogeneous profiles of neurodivergent adults. Chapter 2 highlighted that the co-occurrence of NDs (specifically ADHD+RD) and the heterogeneity of associated cognitive profiles remain consistently high across the lifespan, suggesting that diagnostic categories may not be as distinct and as adequately explanatory as previously suggested. Chapter 4 provided data-driven evidence to suggest that current diagnostic categories show both variability and overlaps, and that individual profiles of functioning may be better characterised by transdiagnostic mechanisms than formal diagnostic

labels. Chapter 5 further supported the above findings, providing additional data-driven evidence for the role of specific processes in contributing to individual variation and/or the co-occurrence of difficulties in the present transdiagnostic sample. Overall, this thesis provides substantial empirical evidence for the benefits of adopting a transdiagnostic approach to exploring the heterogeneous nature of neurodiverse conditions.

6.3.2. Dimensional vs. Categorical Approaches to Assessment and Support

The reconceptualisation of neurodiverse conditions as a multidimensional space through broader assessment batteries is a crucial aspect of adopting a transdiagnostic approach. It promotes a shift away from reinforcing the assumed categorical structure of NDs, and offers the opportunity for the development of more adequate support (Astle et al., 2022; Astle & Fletcher-Watson, 2020). The present transdiagnostic sample was assessed through a series of broad and continuous measures, which yielded empirical evidence to support a dimensional (rather than categorical) structure of NDs. As previously mentioned, Chapter 4 (cluster analysis) indicated that the current sample was best characterised by a set of strengths and weaknesses along two dimensions. This two-dimensional structure was further confirmed by the network analysis in Chapter 5. The results showed that processes which may primarily be associated with distinct diagnostic categories (e.g., working memory difficulties in ADHD, reading difficulties in dyslexia; Kofler et al., 2010; Lyon et al., 2003) can be highly interconnected and co-occur within the same dimension or community. These findings suggest that NDs likely lie on a spectrum of shared and interacting processes, and may therefore be better characterised by a set of overarching continuous dimensions, rather than discrete categories resulting from ‘core’ difficulties.

The present results are in line with the emerging dimensional research frameworks and classification systems, such as Research Domain Criteria (RDoC) and the Hierarchical Taxonomy of Psychopathology (HiTOP). The frameworks advocate for the alternative characterisation and classification of mental health conditions based on underlying dimensions (e.g., cognitive or emotional) as opposed to traditional diagnostic categories (Insel et al., 2010; Kotov et al., 2017). Such dimensions cut across diagnostic categories, thus naturally addressing the issue of heterogeneity and co-occurrence, and can provide more comprehensive accounts of individual functioning (Ruggero et al., 2019; Kozak & Cuthbert, 2016). The broad and continuous nature of dimensions also captures the full range of abilities (i.e., both strengths and

weaknesses), making this approach well-suited for meaningfully distinguishing between individuals in subclinical parts of the population (Alexander et al., 2020). This is especially valuable for the assessment and support of neurodiverse conditions, where the profiles of many individuals do not align with traditional diagnostic categories, as evidenced in the present thesis.

Through the identification of dimensions relevant to adults with both clinical and subthreshold presentations of various NDs, the present findings not only provide further evidence for understanding neurodiversity as a multidimensional construct, but also highlight potential areas for improving assessment and support of NDs. For instance, the identified dimensions may be used to enhance diagnostic sensitivity to individual profiles that neither align with traditional diagnostic categories, nor meet current diagnostic criteria. They may also be utilised as targets for future interventions based on individual needs, rather than diagnostic labels. This approach to assessment and support has the capacity to increase access to more adequate personalised support, both for individuals with complex profiles (e.g., those with co-occurring conditions), as well as those with milder difficulties (Astle et al., 2022). Importantly, since the dimensions identified in the present study also reflect relative strengths in functioning, they may be even more informative for intervention development. The leveraging of individual strengths to support areas of need has proved useful in neurodivergent children (e.g., Naples & Tuckwiller, 2021; Scarcella et al., 2023), and has the potential to be effective in neurodivergent adults, who often develop compensatory strategies over time (e.g., Ullman & Pullman, 2015; van der Putten et al., 2024). Such practical implications of the present findings underscore the value of administering dimensional assessments, as gold standard measures typically focus on assessing ‘core deficits’ associated with specific diagnostic labels, thus limiting the opportunity for a more comprehensive understanding of individual strengths *and* weaknesses (Astle & Fletcher-Watson, 2020). Overall, the findings from Chapters 4-5 highlight the utility of considering NDs as continuous dimensions as opposed to distinct categories, particularly for evaluating and supporting neurodiverse conditions in adults.

6.3.3. Cognition and Mental Health in Neurodivergent Adults

Cognition and mental health are both important for understanding individual wellbeing in neurodiverse populations, and the interplay between them may be key to uncovering some of the complexity associated with NDs (as described in Chapter 1). This thesis has provided a

data-driven account of the interactions between several cognitive and mental health processes, which may help to characterise a wide range of neurodivergent profiles. First, Chapter 4 showed that data-driven transdiagnostic groups could be defined by a constellation of strengths and weaknesses in cognition (language and memory) and mental health (emotional difficulties and externalising problems). Second, Chapter 5 highlighted emotion regulation (i.e., emotional difficulties) as a noteworthy mental health mechanism which may mediate the relationship between more closely related cognitive processes (i.e., language/memory and executive function/processing speed). Together, Chapters 4 and 5 demonstrated that although the interplay between cognitive and mental health processes may be subtle, it contributes meaningfully to data-driven grouping, supporting the notion that cognition and mental health are not completely independent constructs in the context of NDs. These findings suggest that integrating both domains of functioning in the assessment of neurodivergent adults can provide a more comprehensive understanding of individual profiles, beyond that offered by cognitive or mental health assessments alone, thus providing further support for dimensional accounts of neurodiverse conditions.

The abovementioned findings are in line with a recent review proposing that neurodiverse diagnoses should be incorporated into a single continuous transdiagnostic model, along with mental health conditions, such as internalising or externalising (Michellini et al., 2024). Such a framework has the capacity to overcome some of the barriers associated with the identification and support of complex neurodiverse profiles, in terms of characterising the developmental continuity and co-occurrence between NDs and mental health conditions (Michellini et al., 2024). This theoretical model builds on the transdiagnostic dimensional research frameworks mentioned in the previous section (i.e., RDoC and HiTOP), and highlights the utility of the present findings in providing empirical evidence for the ways in which mental health and/or emotional processes may interact with cognition in neurodivergent adults.

To this end, Chapters 4 and 5 have revealed some overarching dimensions which may be relevant across multiple neurodiverse and mental health conditions, and thus suggested potential mechanisms that may contribute to their co-development/co-occurrence. Such evidence is especially valuable in the context of NDs in adulthood, since research suggests that there is a limited understanding of how to effectively support mental health in neurodivergent adults (e.g., Asherson et al., 2022). This challenge is further exacerbated by the uncertainty around adult presentations of NDs, which has been shown to have an adverse impact on their

identification in clinical contexts (Lipinski et al., 2022; Sadusky et al., 2021; Goodman et al., 2024), suggesting that the identification of the co-occurrence of NDs with mental health conditions may be a challenge in itself. Thus, this thesis provides preliminary data-driven insights into cognition and mental health in neurodivergent adults, which extend on existing dimensional accounts of NDs, and underscore the value of integrative approaches for improving the assessment and support of neurodivergent adults in both clinical and research contexts.

6.4. Strengths and Limitations

6.4.1. Strengths

This thesis has several methodological strengths. First, the systematic review aimed to synthesise data across a broad range of cognitive domains in both children and adults with co-occurring ADHD+RD. Thus, it was one of the first systematic reviews to attempt to adopt a transdiagnostic dimensional *and* developmental approach to neurodiverse conditions. The implementation of a lifespan approach addressed the current lack of longitudinal studies assessing developmental changes in the prevalence and/or cognitive manifestations of ADHD+RD. The review also addressed the limited availability of epidemiological/prevalence evidence across different stages of development, through quantifying the co-occurrence of ADHD+RD by synthesising study-level proportions. Similarly, the lack of research into the co-occurrence of conditions in neurodivergent adults was highlighted throughout the review, providing a target group for the present empirical study. Further, the methodology of the systematic review enabled for the exploration of the effect of broader versus more restrictive sampling on the identification of co-occurring ADHD+RD across studies. The review therefore yielded quantitative evidence for the impact of categorical study designs on neurodiversity research, and provided additional rationale for the employment of more inclusive recruitment methods in the present study. More generally, the systematic review was largely methodologically robust. For instance, sensitivity and publication bias analyses were conducted to evaluate the robustness of the evidence base, whilst transparency in reporting was consistently maintained throughout the review process. Additionally, there was a high level of agreement between reviewers across multiple stages of the review, enhancing the reliability of the findings by reducing reviewer bias. Overall, the overarching strength of the systematic review is that it extended on the narrative literature review in Chapter 1, through providing a

comprehensive synthesis of available evidence on a specific example of the complexity associated with NDs, thus offering a more refined rationale for the present study.

The present study also has ample strengths. The transdiagnostic dimensional design of the study directly addressed several shortcomings of categorical research, and built on the findings of the systematic review. First, through the employment of a more relaxed approach to recruitment, the present study addressed the limitations of ‘case-control’ study designs, which have been shown to lead to a loss of important information regarding the complexity associated with NDs (Astle et al., 2022). To this end, the study’s inclusive sample captured a broad spectrum of abilities, from typical to atypical functioning, making it highly representative of the heterogeneous neurodiverse population, and allowing for the exploration of mechanisms that may contribute to such heterogeneity. Additionally, through focusing on neurodivergent adults specifically, the study addressed the current lack of research into the complexity of NDs in adulthood (as underscored via the systematic and narrative reviews).

Second, the combination of dimensional cognitive and mental health assessments complemented the transdiagnostic nature of the study, and addressed some of the limitations associated with ‘core deficit’ accounts of diagnostic categories (Astle & Fletcher-Watson, 2020). This approach allowed for the exploration of strengths and weaknesses across several domains of functioning, therefore providing more valuable information than that offered by ‘gold standard’ assessments, which typically focus on the atypical end of the spectrum and may not be suitable for differentiating between individuals with milder difficulties (Alexander et al., 2020). Another strength of this broader approach to assessment is that it highlighted interactions between both cognitive and mental health processes that may reflect areas of need relevant across a wide range of neurodiverse conditions, providing an even more comprehensive understanding than cognition or mental health alone (see Section 6.3.3.).

Third, the data-driven analytical approach employed was well-suited for this study’s transdiagnostic sample and the dimensional assessments administered, and enabled for the uncovering of valuable insights from such complex, multivariate data. The combination of cluster and network analyses allowed for a more comprehensive understanding of the underlying structure of the dataset and the nuanced between-variable interactions contributing to this structure. This multi-method approach enhanced the interpretability of the findings, through enabling the identification of meaningful patterns, which may be overlooked when

adopting more traditional univariate or bivariate approaches that often accompany categorical or ‘case-control’ study designs (Astle et al., 2022). Importantly, the employment of data-driven methods offered the opportunity to explore the present dataset in a hypothesis-generating rather than hypothesis-confirming manner, which is particularly valuable in the context of neurodiversity research, where novel data-driven insights are crucial for advancing theoretical models. This allowed for the reconceptualisation of neurodiversity as a multidimensional space, in which naturally occurring transdiagnostic groups were identified, without confinement to predefined diagnostic groups, allowing for a shift away from reinforcing the categorical structure of NDs. Thus, the overarching strength of the present study was that it built on the emerging quantitative accounts of neurodiversity, and yielded empirical evidence with the capacity to better characterise neurodiversity in adults, providing a foundation for more comprehensive assessments and/or more adequate support of NDs in adulthood.

The present study also has more general strengths. First, the sample was not only representative of the neurodiverse population, but also ethnically diverse, as the participant pool consisted of multiple ethnicities, including Caucasian, Asian, African/Caribbean and Mixed Heritage individuals. Second, the sample age ranged from 18 to 51 years, making it more representative of the adult population compared to other studies within this field, which typically focus on specific age groups (e.g., university students, or older adults). Further, the present sample was also representative of individuals with different levels of education (including secondary school, sixth form/college, undergraduate and postgraduate degrees), which further emphasises its appropriateness for capturing the full spectrum of functioning (e.g., in terms of cognitive ability and educational attainment). Another strength of the sample is that it not only comprised individuals with co-occurring NDs, but also those with clinically diagnosed co-occurring mental health conditions, further highlighting the transdiagnostic relevance of this study.

Moreover, to our knowledge, this study was the first to employ the NIH Cognition Toolbox with a neurodivergent adult sample in the United Kingdom, with the majority of research utilising this measure being conducted in the United States. The current dataset could potentially be used to contribute to the extant NIH database, or to aid in the validation of the NIH Toolbox for the neurodivergent adult population in the United Kingdom. Further, the systematic review has been pre-registered on PROSPERO and conducted in accordance with PRISMA guidelines (Page et al., 2021), thus promoting methodological transparency and

reproducibility. This thesis therefore aligns with the principles of the open science framework, and has the potential for broader research impact beyond the current project.

6.4.2. Limitations

This thesis is not without limitations. As discussed in Chapter 2, the results of the systematic review were constrained by significantly high levels of heterogeneity. Whilst it is likely that a substantial proportion of the identified between-study heterogeneity may have been a reflection of the high variability associated with the diverse cognitive profiles of ADHD and RD (Vos et al., 2022; Chalmpe & Vlachos, 2025), the findings suggest that there was systematic variation across studies, which may have resulted from methodological differences not accounted for in the review. This raises questions around the certainty in the body of evidence included, particularly relating to the strength or meaning of the pooled effect sizes across studies. Similarly, there was evidence of significant publication bias among child studies. It is possible that this may have resulted from a lack of dimensional assessments employed in the broader literature, with the majority of studies failing to assess relative strengths in cognitive function, or simply choosing not to report non-significant results. This raises further questions around the precision of effect estimates due to missing evidence, potentially suggesting that the full picture was not captured. Thus, although sensitivity analyses indicated that the results of the syntheses were statistically robust and stable, the presence of publication bias and substantial heterogeneity suggest that the validity and generalisability of the overall systematic review may be limited.

These findings highlight the need to consider how missing or varied evidence may influence apparent statistical robustness, and suggest that the present review may have benefitted from the inclusion of ‘grey’ literature or further subgroup analyses. To this end, the systematic review was constrained by the inclusion of published journal articles only, suggesting that some relevant studies may have been missed. For instance, the inclusion of theses, research reports, or conference posters may have mitigated the issue of high publication bias, as such literature may be more likely to report non-significant results, or apply novel (i.e., dimensional) approaches to cognitive assessment. Further, although efforts were made to investigate the impact of sampling methods and model used to define RD, the high heterogeneity in co-occurrence rates could have been further explored by considering whether the methods used to confirm ADHD accounted for any variability across studies. Similarly, the substantial

heterogeneity observed across studies addressing the cognitive manifestations of ADHD+RD may have been further explored through investigating whether differences in measures used to assess specific cognitive domains accounted for any variability in effect size. Overall, these mitigations could have enhanced the ability to draw firm conclusions about the prevalence and cognitive manifestations of co-occurring ADHD and RD, thus increasing the validity and generalisability of the systematic review.

The challenge of generalisability may also apply to the present transdiagnostic study. Although the sample may have been highly representative of neurodiverse conditions, educational levels, ethnicities, and age groups, the vast majority of participants were recruited from Aston University. Thus, the majority of participants were undergraduate or postgraduate students, which limits the external validity of the study. This is because neurodivergent individuals who attend university may be more able to compensate for their difficulties. For instance, students with dyslexia have been shown to achieve at a normal rate of study through developing compensatory strategies for their reading problems (Olofsson et al., 2015). This may not be the case for neurodivergent individuals who choose not to attend university as a result of more pronounced difficulties. Although efforts were made to recruit participants external to the university (e.g., via charities and healthcare practitioners), only a small proportion of the sample were non-university students, and therefore the present study may lack generalisability beyond an academic setting. This limitation could have been mitigated by employing more targeted recruitment strategies aimed at community settings or neurodiversity advocacy groups, or by delegating aspects of recruitment to individuals with increased access to such stakeholders.

Similarly, given the in-person nature of the study and relative time constraints, participant recruitment was restricted to the Birmingham area. Although there were several participants who were not local to Birmingham (e.g., relocated for university or travelled to participate), the study may lack generalisability beyond this specific location. This limitation is directly related to the nature of the NIH Toolbox, which must be administered on an iPad with an active subscription. Thus, offering remote participation options may have increased accessibility, and further broadened the geographic and/or educational diversity of the sample. Whilst online administration is currently not an option for the NIH Toolbox, future studies may opt for other dimensional cognitive assessment batteries, such as the Cambridge Neuropsychological Test Automated Battery (CANTAB; Robbins et al., 1994), which offer web-based testing.

The cognitive assessment was further constrained by the requirement for a trained examiner to be present. Specifically, the List Sorting Working Memory Test (LSWMT) and the Oral Reading Recognition Test (ORRT) were administered by the examiner, who was required to indicate whether participant answers were correct or incorrect. First, the LSWMT did not require any training as an answer sheet was provided in the NIH Toolbox Administrator's Manual, but the requirement for examiner-administered scoring may have introduced bias. Specifically, it is possible that the examiner may have unintentionally influenced participant performance through facial expressions, tone of voice, or body language, particularly if they were motivated by a desire for participants to perform well. Second, the ORRT required substantial training regarding the correct pronunciation of low frequency words (e.g., "diamantiferous"). This training was provided via the NIH Toolbox's training resources (i.e., voice recordings), but was limited to the correct American English pronunciation of words, which did not always correspond to the British English pronunciation. This may have led to inaccurate scoring or unintended penalisation of participants using correct but non-American pronunciations. Similarly, the uncertainty regarding the British pronunciation of particular words may have resulted in incorrect answers being marked as correct. Individual variation in pronunciation as a result of varying British accents may have further contributed to this uncertainty. Thus, the reliance on examiner scoring in both the LSWMT and ORRT may have introduced the potential for bias, highlighting the challenge of maintaining objectivity in examiner-administered assessments. This may have implications for the consistency and reliability of task administration and scoring, as well as the validity of the data yielded from the LSWMT and the ORRT.

Whilst this limitation could, in principle, have been mitigated by assessing inter-rater reliability (e.g., having a second trained examiner independently score a subset of responses), this was not feasible within the constraints of the NIH Toolbox. This is because responses for the LSWMT and ORRT were entered directly into the iPad during testing, with the scores being automatically recorded by the system without the option for manual adjustment. Therefore, it was not possible to conduct independent verification of scoring accuracy or consistency across examiners. Future studies utilising the NIH Toolbox may therefore consider incorporating additional procedures to reduce potential for examiner bias. For instance, video or audio recordings of test sessions could be used to allow for retrospective review. Additionally, it may be beneficial for the developers of the NIH Toolbox to consider incorporating an option for

manual scoring or post-hoc score verification within the software, which would be particularly valuable for measures that rely on subjective judgment or verbal responses, allowing for the assessment of inter-rater reliability where possible. This function would enhance the transparency and replicability of research using the NIH Toolbox.

The mental health assessment was also not without its limitations, given that all selected measures were self-report. This may have impacted the objectivity of the data as self-report measures are subject to introspective ability and response bias, which may be even more of a challenge in neurodiverse populations. For instance, autistic individuals have been shown to demonstrate reduced introspection and mentalising abilities, and thus poorer knowledge of the self and others (Robinson et al., 2017). This may have, for instance, posed a challenge when answering questions, such as “compared to other people of your age, how well do you...” in the Extended Strengths and Weaknesses Assessment of Normal Behaviour (E-SWAN). Although the self-report measures were selected based on their neutrally worded questions and non-leading multiple-choice answer options, low introspective ability may have increased the risk of response bias. For example, individuals who lack awareness of their own emotional or behavioural patterns may have attempted to select the ‘expected’ or socially desirable answer. This may have reduced the validity of the mental health dataset. To address this limitation, future studies could include brief measures of introspective ability, such as the Perth Alexithymia Questionnaire-Short Form (PAQ-S; Preece et al., 2023), in order to identify participants whose self-report responses should be interpreted with caution. Additionally, future studies may benefit from involving neurodivergent individuals in the design of self-report mental health measures, in order to ensure that items are better tailored to how they understand and/or express their experiences.

Another noteworthy limitation is that the ‘Suspected ND’ group was determined based on self-report responses to a demographic questionnaire, where individuals were asked whether they experience and/or have ever been referred for any difficulties associated with neurodiverse conditions (e.g., memory or attention problem). This group therefore included individuals with a wide range of suspected difficulties, which were not further subcategorised. While the present study adopted a transdiagnostic approach, the inability to further explore the nature of suspected difficulties within this group restricted interpretation of how their profiles related to the data-driven clusters identified in Chapter 4. Future research could incorporate more detailed

screening tools to assess suspected difficulties, allowing for a deeper understanding of the composition of transdiagnostic clusters, even among individuals without formal diagnoses.

A final limitation to consider is that the target sample size of 160 to 240 participants was established based on research suggesting that studies should aim for 20 to 30 observations per expected subgroup when conducting cluster analysis, as this provides sufficient power for detecting number of clusters and individual cluster membership (Dalmaijer et al., 2022). The present study initially aimed to explore a total of 8 core variables on which individuals could differ. This included 5 cognitive variables (attention, executive function, memory, processing speed, and language), based on the NIH Toolbox literature suggesting that the seven tasks included in the Cognition Battery tap these overarching cognitive domains (Weintraub et al., 2013). It also included 3 primary mental health variables (internalising problems, externalising problems, and emotion regulation). These were derived from the literature review in Chapter 1, with the mental health measures being specifically selected to reflect these key domains. However, the final analyses were conducted with a total of 11 variables. This is because the 7 cognitive variables (as reflected by each task administered) were retained in order to make finer distinctions between cognitive domains. For instance, inhibitory control and cognitive flexibility were regarded as separate functions, rather than collapsing them into the overarching domain of executive function, as this allowed for the exploration of more nuanced between-variable interactions. Additionally, a principal component analysis (PCA) of the mental health variables suggested a 4-component solution (emotional difficulties, emotional clarity, social skills, and hyperactive behaviour), which did not directly correspond to the abovementioned mental health domains.

Thus, the inclusion of 11 variables in the final analyses, along with a total sample size of 175, may have reduced power for identifying meaningful patterns across the cluster and network analyses (Chapters 4-5). For instance, since cluster analysis can be highly sensitive to individual cases (e.g., Gao et al., 2023), which suggests that removing or adding one case can significantly affect the course of the analysis, the small sample size (relative to the number of variables) may have affected the stability of the cluster solution. Likewise, a small sample may not have provided enough information to accurately represent the underlying structure of the dataset, which could have reduced the generalisability of the data-driven clusters (Dalmaijer et al., 2022). Additionally, since network edges (i.e., connections between variables) are estimated based on pairwise relationships, a smaller sample size may have resulted in such edges

becoming less reliable. This is because when there are many variables with few observations, there's a greater risk of model overfitting, where identified edges reflect statistical noise rather than true associations (e.g., Ying, 2019). This can lead to the model performing poorly on new unseen data, resulting in reduced generalisability. Smaller samples may also destabilise the network structure, as seen in the stability analyses results in Chapter 5, where most centrality indices were shown to be highly sensitive to data variation across bootstrapped samples. Thus, the sample size-to-variable ratio may have affected the interpretability and/or reliability of the cluster and network analyses results. In order to validate the current preliminary insights and establish their generalisability beyond the present sample, future studies should recruit larger samples to ensure sufficient power for detecting more robust and/or meaningful patterns.

6.5. Future Directions

In light of the present thesis, future research should focus on yielding further data-driven accounts of neurodiverse conditions to build on the emerging transdiagnostic dimensional approach to neurodiversity. While such work is needed across the lifespan, it is particularly warranted in the neurodivergent adult population, where challenges related to assessment, diagnosis and access to appropriate support remain especially pronounced. Further, there is a need for longitudinal research to explore developmental trajectories and track individual wellbeing over time through a transdiagnostic dimensional lens, particularly during the transition from childhood to adulthood. For instance, dynamic network modelling could be used to track how between-variable relationships evolve over time (e.g., Den Hartigh et al., 2016). Such research would enable for a deeper understanding of the dynamic interactions between various domains of functioning that contribute to change across developmental stages, and potentially inform the development of more targeted and timely interventions across the lifespan. Importantly, future transdiagnostic studies should also adopt an integrative approach to dimensional assessment, through assessing both cognition and mental health, in order to gain a more comprehensive understanding of individual wellbeing, and potentially validate and/or extend the preliminary findings of this thesis.

Similarly, future systematic reviews may benefit from considering synthesising evidence across both mental health and cognition in children and adults with co-occurring ADHD+RD. Such an approach could highlight mental health processes relevant to the co-occurrence of ADHD and RD, or those that are currently under researched within this population, potentially

providing a basis for future dimensional assessments. Future systematic reviews may also consider further exploring the impact of methodology on between-study variability (e.g., measures used to assess cognition, or methods used to confirm diagnosis), and/or broadening their inclusion criteria to include more relevant studies in order to address publication bias. These considerations may increase certainty in drawing firm conclusions from the body of evidence, thus enhancing the overall generalisability of the findings. The abovementioned suggestions could also be applied to systematic reviews of different populations, for instance, children and adults with co-occurring ADHD and autism. Such quantitative syntheses across different patterns of co-occurring conditions could provide a valuable evidence base for future transdiagnostic studies, and/or targets for future support strategies relevant to a wide range of NDs in both children and adults.

Moreover, future transdiagnostic studies should ensure appropriate sampling as this has implications for both the applicability and the robustness of findings. Efforts should be made to recruit participants from a variety of sources, including different communities and locations, in order to increase the representativeness of the sample beyond convenience-based populations, and to ensure that the findings are generalisable to the broader neurodiverse community. Additionally, future studies employing cluster or network analyses should ensure sufficiently large sample sizes for detecting meaningful patterns and improving the stability of results. Such larger-scale research may help to validate the data-driven groupings and between-variable interactions identified in the present thesis. This is particularly important given that the associations between cognitive and mental health processes were evident, yet relatively weak and unstable. Increasing sample size could enhance the reliability of such findings, and provide stronger empirical support for the theoretical and practical implications proposed in this thesis.

Furthermore, future studies may opt for remote testing. This may not only encourage participation, thus increasing sample size and representativeness, but also overcome some of the challenges associated with examiner-administered cognitive assessments (e.g., examiner bias). It may also help to reduce social desirability bias in self-report mental health measures through limiting in-person interaction with the researcher, thus increasing feelings of anonymity. Likewise, remote testing may be more appropriate and/or accessible for neurodivergent individuals who experience increased levels of social anxiety, which may in some cases deter prospective participants from taking part in face-to-face research due to

avoidance of social interactions (e.g., Montaser et al., 2023; Jakobsson Støre et al., 2024). Overall, online-based cognitive and mental health assessments may be more suitable for studies where large sample sizes, accessibility and inclusivity are crucial. However, potential limitations of remote testing should be considered, including technological barriers, lack of controlled testing environments, and/or reduced opportunities for clarifying instructions, which may be especially relevant in neurodivergent populations. Therefore, balancing the benefits and drawbacks of remote versus in-person testing is essential for developing future transdiagnostic studies.

Finally, future transdiagnostic studies may focus on exploring a different combination of data reduction techniques to uncover data-driven insights from larger datasets. While the present study employed a combination of PCA, cluster analysis, and linear discriminant analysis (LDA) to investigate the underlying structure of the dataset, exploratory factor analysis (EFA) and multinomial logistic regression were also explored. These techniques were not deemed appropriate for the current dataset due to reduced interpretability compared to the selected methods (see Appendix B, Supplementary Materials 3), but may be worth exploring with larger datasets. To this end, multinomial logistic regression may be more robust than LDA when dealing with non-normally distributed data (which is often the case with mental health questionnaires), thus providing more reliable insights into the structure of the dataset, but may require a larger sample size for the model to perform well (Antonogeorgos et al., 2009; Bono et al., 2017; Pate et al., 2023). Similarly, EFA can provide valuable information about latent traits that characterise the dataset beyond simply reducing the dimensionality of the data, but may also require a larger sample size for a more reliable estimation of between-variable relationships (Alavi et al., 2020; Rouquette & Falissard, 2011). Thus, incorporating these techniques in future studies with larger, well-powered samples could provide further insights into the complex interplay between cognition and mental health in neurodiverse conditions.

6.6. Conclusion

This thesis has built on the emerging transdiagnostic dimensional accounts of neurodiversity through providing data-driven insights into the cognitive and mental health profiles of neurodivergent adults. First, the findings of the systematic review suggested that the co-occurrence of ADHD+RD remains consistently high across the lifespan, and that both children and adults experience comparable levels of difficulty across several cognitive domains. The review provided a foundation for the empirical study, through highlighting that complex profiles of co-occurring neurodiverse conditions persist into adulthood, and that adults are substantially underrepresented in the literature. Second, the findings of the present study revealed that a transdiagnostic sample of neurodivergent adults was best characterised by three data-driven groups, which did not directly map onto traditional diagnostic categories. These transdiagnostic groups were defined by a combination of strengths and weaknesses along two dimensions relating to cognition and mental health, supporting a dimensional rather than categorical structure of neurodiverse conditions. Additionally, the findings highlighted several cognitive and mental health processes which may contribute to some of the complexity associated with neurodiverse diagnoses (in terms of individual variation, or the co-occurrence of conditions).

Overall, this thesis has provided a significant contribution to the field of neurodiversity research through a) adopting both a transdiagnostic dimensional and a developmental approach to data synthesis in the systematic review, b) developing a novel transdiagnostic dimensional protocol to explore neurodiversity in adults, thus directly addressing a current gap in the literature, c) adopting an integrative approach to dimensional assessment via exploring the interactions between cognitive and mental health processes to gain a more comprehensive understanding of individual wellbeing, and d) employing data-driven methods to generate novel accounts of neurodiverse conditions, rather than attempting to confirm existing diagnostic categories and their associated ‘core’ difficulties. Together, the findings of this thesis may be used to inform more adequate assessment and/or support of NDs in adults, based on individual needs rather than diagnostic labels. However, further research yielding data-driven accounts of neurodiverse conditions is required to build on the present findings, and to further extend current transdiagnostic dimensional frameworks.

References

Note. *** Indicates that a reference was included in the systematic review.

- Aaron, P. G., Joshi, R. M., Palmer, H., Smith, N., & Kirby, E. (2002). Separating genuine cases of reading disability from reading deficits caused by predominantly inattentive ADHD behavior. *Journal of Learning Disabilities*, 35(5), 425–447. <https://doi.org/10.1177/00222194020350050301>. ***
- Adams, J. W., & Snowling, M. J. (2001). Executive function and reading impairments in children reported by their teachers as “hyperactive”. *British Journal of Developmental Psychology*, 19(2), 293–306. <https://doi.org/10.1348/026151001166083>. ***
- Adams, J. W., Snowling, M. J., Hennessy, S. M., & Kind, P. (1999). Problems of behaviour, reading and arithmetic: Assessments of comorbidity using the Strengths and Difficulties Questionnaire. *The British Journal of Educational Psychology*, 69(4), 571–585. <https://doi.org/10.1348/000709999157905>. ***
- Adams, Z. W., Derefinko, K. J., Milich, R., & Fillmore, M. T. (2008). Inhibitory functioning across ADHD subtypes: Recent findings, clinical implications, and future directions. *Developmental Disabilities Research Reviews*, 14(4), 268–275. <https://doi.org/10.1002/ddrr.37>
- Adwas, A. A., Jbireal, J. M., & Azab, A. E. (2019). Anxiety: Insights into signs, symptoms, etiology, pathophysiology, and treatment. *East African Scholars Journal of Medical Sciences*, 2(10), 580–591.
- Aitken, M., Martinussen, R., Childs, R., & Tannock, R. (2020). Profiles of co-occurring difficulties identified through school-based screening. *Journal of Attention Disorders*, 24(9), 1355–1365. <https://doi.org/10.1177/1087054716684377>. ***
- Akyurek, G., & Bumin, G. (2019). An investigation of executive function in children with dyslexia. *Psychiatry and Behavioral Sciences*, 9(1). <https://doi.org/10.5455/PBS.20181128092027>
- Al Dahhan, N. Z., Halverson, K., Peek, C. P., Wilmot, D., D’Mello, A., Romeo, R. R., Meegoda, O., Imhof, A., Wade, K., Sridhar, A., Falke, E., Centanni, T. M., Gabrieli, J. D. E., & Christodoulou, J. A. (2022). Dissociating executive function and ADHD influences on reading ability in children with dyslexia. *Cortex; a Journal Devoted to the Study of the Nervous System and Behavior*, 153, 126–142. <https://doi.org/10.1016/j.cortex.2022.03.025>. ***
- Al-Saoud, S., Nichols, E. S., Brossard-Racine, M., Wild, C. J., Norton, L., & Duerden, E. G. (2025). A transdiagnostic examination of cognitive heterogeneity in children and adolescents with neurodevelopmental disorders. *Child Neuropsychology: A Journal on Normal and Abnormal Development in Childhood and Adolescence*, 31(2), 293–311. <https://doi.org/10.1080/09297049.2024.2364957>

- Alavi, M., Visentin, D. C., Thapa, D. K., Hunt, G. E., Watson, R., & Cleary, M. (2020). Exploratory factor analysis and principal component analysis in clinical studies: Which one should you use? *Journal of Advanced Nursing*, 76(8), 1886–1889. <https://doi.org/10.1111/jan.14377>
- Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review*, 30(2), 217–237. <https://doi.org/10.1016/j.cpr.2009.11.004>
- Aldenderfer, M. S., & Blashfield, R. K. (1984). *Cluster analysis*. Sage Publications. <https://doi.org/10.4135/9781412983648>
- Alderson, R. M., Rapport, M. D., Hudec, K. L., Sarver, D. E., & Kofler, M. J. (2010). Competing core processes in attention-deficit/hyperactivity disorder (ADHD): Do working memory deficiencies underlie behavioral inhibition deficits? *Journal of Abnormal Child Psychology*, 38(4), 497–507. <https://doi.org/10.1007/s10802-010-9387-0>
- Alexander, L. M., Salum, G. A., Swanson, J. M., & Milham, M. P. (2020). Measuring strengths and weaknesses in dimensional psychiatry. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 61(1), 40–50. <https://doi.org/10.1111/jcpp.13104>
- Alexander, R. T., Langdon, P. E., O'Hara, J., Howell, A., Lane, T., Tharian, R., & Shankar, R. (2021). Psychiatry and neurodevelopmental disorders: Experts by experience, clinical care and research. *The British Journal of Psychiatry*, 218(1), 1–3. <https://doi.org/10.1192/bjp.2020.237>
- Altay, M. A., & Görker, I. (2018). Assessment of psychiatric comorbidity and WISC-R profiles in cases diagnosed with specific learning disorder according to DSM-5 criteria. *Archives of Neurology and Psychiatry*, 55(2), 127–134. <https://doi.org/10.5152/npa.2017.18123>. ***
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). American Psychiatric Association. <https://doi.org/10.1176/appi.books.9780890425596>
- American Psychiatric Association. (2022). *Diagnostic and statistical manual of mental disorders* (5th ed., text rev.). American Psychiatric Association Publishing. <https://doi.org/10.1176/appi.books.9780890425787>
- Antolini, G., & Colizzi, M. (2023). Where do neurodevelopmental disorders go? Casting the eye away from childhood towards adulthood. *Healthcare*, 11(7), 1015. <https://doi.org/10.3390/healthcare11071015>
- Antonogeorgos, G., Panagiotakos, D. B., Priftis, K. N., & Tzonou, A. (2009). Logistic regression and linear discriminant analyses in evaluating factors associated with asthma prevalence among 10- to 12-years-old children: Divergence and similarity of the two statistical methods. *International Journal of Pediatrics*, 2009(1), Article 952042. <https://doi.org/10.1155/2009/952042>

- Areces, D., García, T., González-Castro, P., Alvarez-García, D., & Rodríguez, C. (2018). Naming speed as a predictive diagnostic measure in reading and attentional problems. *Child Neuropsychology: A Journal on Normal and Abnormal Development in Childhood and Adolescence*, 24(8), 1115–1128. <https://doi.org/10.1080/09297049.2017.1391191>. ***
- Aro, T., Eklund, K., Eloranta, A.-K., Ahonen, T., & Rescorla, L. (2022). Learning disabilities elevate children's risk for behavioral-emotional problems: Differences between LD types, genders, and contexts. *Journal of Learning Disabilities*, 55(6), 465–481. <https://doi.org/10.1177/00222194211056297> ***
- Åsberg, J., Kopp, S., Berg-Kelly, K., & Gillberg, C. (2010). Reading comprehension, word decoding and spelling in girls with autism spectrum disorders (ASD) or attention-deficit/hyperactivity disorder (AD/HD): Performance and predictors. *International Journal of Language and Communication Disorders*, 45(1), 61–71. <https://doi.org/10.3109/13682820902745438>. ***
- Asherson, P., & Agnew-Blais, J. (2019). Annual Research Review: Does late-onset attention-deficit/hyperactivity disorder exist? *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 60(4), 333–352. <https://doi.org/10.1111/jcpp.13020>
- Asherson, P., Leaver, L., Adamou, M., Arif, M., Askey, G., Butler, M., Cubbin, S., Newlove-Delgado, T., Kustow, J., Lanham-Cook, J., Findlay, J., Maxwell, J., Mason, P., Read, H., van Rensburg, K., Müller-Sedgwick, U., Sedgwick-Müller, J., & Skirrow, C. (2022). Mainstreaming adult ADHD into primary care in the UK: Guidance, practice, and best practice recommendations. *BMC Psychiatry*, 22(1), 640. <https://doi.org/10.1186/s12888-022-04290-7>
- Astle, D. E., & Fletcher-Watson, S. (2020). Beyond the core-deficit hypothesis in developmental disorders. *Current Directions in Psychological Science*, 29(5), 431–437. <https://doi.org/10.1177/0963721420925518>
- Astle, D. E., Bathelt, J., CALM Team, & Holmes, J. (2019). Remapping the cognitive and neural profiles of children who struggle at school. *Developmental Science*, 22(1), Article e12747. <https://doi.org/10.1111/desc.12747>
- Astle, D. E., Holmes, J., Kievit, R., & Gathercole, S. E. (2022). Annual Research Review: The transdiagnostic revolution in neurodevelopmental disorders. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 63(4), 397–417. <https://doi.org/10.1111/jcpp.13481>
- Baddeley, A. (2007). Working memory. *Thought and Action*. Oxford University Press. <https://doi.org/10.1093/acprof:oso/9780198528012.001.0001>
- Balduzzi, S., Rücker, G., & Schwarzer, G. (2019). How to perform a meta-analysis with R: A practical tutorial. *Evidence-Based Mental Health*, 22(4), 153–160. <https://doi.org/10.1136/ebmental-2019-300117>

- Balshem, H., Helfand, M., Schünemann, H. J., Oxman, A. D., Kunz, R., Brozek, J., Vist, G. E., Falck-Ytter, Y., Meerpohl, J., Norris, S., & Guyatt, G. H. (2011). GRADE guidelines: 3. Rating the quality of evidence. *Journal of Clinical Epidemiology*, 64(4), 401–406. <https://doi.org/10.1016/j.jclinepi.2010.07.015>
- Banerjee, M., & Wellner, J. A. (2005). Score statistics for current status data: Comparisons with likelihood ratio and Wald statistics. *The International Journal of Biostatistics*, 1(1). <https://doi.org/10.2202/1557-4679.1001>
- Baraskewich, J., & McMorris, C. A. (2019). Internalizing mental health issues in individuals with neurodevelopmental disorders: Implications for practitioners. *Current Developmental Disorders Reports*, 6(1), 1–8. <https://doi.org/10.1007/s40474-019-0154-9>
- Barker, T. H., Migliavaca, C. B., Stein, C., Colpani, V., Falavigna, M., Aromataris, E., & Munn, Z. (2021). Conducting proportional meta-analysis in different types of systematic reviews: A guide for synthesisers of evidence. *BMC Medical Research Methodology*, 21(1), 189. <https://doi.org/10.1186/s12874-021-01381-z>
- Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin*, 121(1), 65–94. <https://doi.org/10.1037/0033-2909.121.1.65>
- Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). Does the autistic child have a “theory of mind”? *Cognition*, 21(1), 37–46. [https://doi.org/10.1016/0010-0277\(85\)90022-8](https://doi.org/10.1016/0010-0277(85)90022-8)
- Bathelt, J., Holmes, J., Astle, D. E., & Centre for Attention Learning and Memory (CALM) Team. (2018). Data-driven subtyping of executive function-related behavioral problems in children. *Journal of the American Academy of Child and Adolescent Psychiatry*, 57(4), 252–262.e4. <https://doi.org/10.1016/j.jaac.2018.01.014>
- Battistutta, L., Schiltz, C., & Steffgen, G. (2021). The Mediating Role of ADHD Symptoms and Emotion Regulation in the Association between Executive Functions and Internalizing Symptoms: A Study among Youths with and without ADHD and/or Dyslexia. *Advances in Neurodevelopmental Disorders*, 5(4), 396–412. <https://doi.org/10.1007/s41252-021-00212-y>
- Bauer, P. J., Dikmen, S. S., Heaton, R. K., Mungas, D., Slotkin, J., & Beaumont, J. L. (2013). CHAPTERS 3. NIH Toolbox Cognition Battery (CB): Measuring episodic memory. *Monographs of the Society for Research in Child Development*, 78(4), 34–48. <https://doi.org/10.1111/mono.12033>
- Beaumont, J. L., Havlik, R., Cook, K. F., Hays, R. D., Wallner-Allen, K., Korper, S. P., Lai, J.-S., Nord, C., Zill, N., Choi, S., Yost, K. J., Ustsinovich, V., Brouwers, P., Hoffman, H. J., & Gershon, R. (2013). Norming plans for the NIH Toolbox. *Neurology*, 80(11)(Suppl. 3), S87–S92. <https://doi.org/10.1212/WNL.0b013e3182872e70>

- Becker, A., Daseking, M., Kerner auch Koerner, J. (2021). Cognitive profiles in the WISC-V of children with ADHD and specific learning disorders. *Sustainability*, 13(17), 9948. <https://doi.org/10.3390/su13179948>. ***
- Ben-Hur, A., & Guyon, I. (2003). Detecting stable clusters using principal component analysis. *Methods in Molecular Biology (Clifton, NJ)*, 224, 159–182. <https://doi.org/10.1385/1-59259-364-X:159>
- Bental, B., & Tirosh, E. (2007). The relationship between attention, executive functions and reading domain abilities in attention deficit hyperactivity disorder and reading disorder: A comparative study. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 48(5), 455–463. <https://doi.org/10.1111/j.1469-7610.2006.01710.x> ***
- Berenguer, C., Roselló, B., Colomer, C., Baixauli, I., & Miranda, A. (2018). Children with autism and attention deficit hyperactivity disorder. Relationships between symptoms and executive function, theory of mind, and behavioral problems. *Research in Developmental Disabilities*, 83, 260–269. <https://doi.org/10.1016/j.ridd.2018.10.001>
- Berger, P., & Buttelmann, D. (2022). A meta-analytic approach to the association between inhibitory control and parent-reported behavioral adjustment in typically-developing children: Differentiating externalizing and internalizing behavior problems. *Developmental Science*, 25(1), Article e13141. <https://doi.org/10.1111/desc.13141>
- Berking, M., & Znoj, H. (2008). Entwicklung und Validierung eines Fragebogens zur standardisierten Selbsteinschätzung emotionaler Kompetenzen (SEK-27). *Zeitschrift für Psychiatrie Psychologie und Psychotherapie*, 56(2), 141–153. <https://doi.org/10.1024/1661-4747.56.2.141>
- Bialystok, E. (2017). The bilingual adaptation: How minds accommodate experience. *Psychological Bulletin*, 143(3), 233–262. <https://doi.org/10.1037/bul0000099>
- Biederman, J., Mick, E., & Faraone, S. V. (2000). Age-dependent decline of symptoms of attention deficit hyperactivity disorder: Impact of remission definition and symptom type. *The American Journal of Psychiatry*, 157(5), 816–818. <https://doi.org/10.1176/appi.ajp.157.5.816>
- Biederman, J., Mick, E., Faraone, S. V., Braaten, E., Doyle, A., Spencer, T., Wilens, T. E., Frazier, E., & Johnson, M. A. (2002). Influence of gender on attention deficit hyperactivity disorder in children referred to a psychiatric clinic. *The American Journal of Psychiatry*, 159(1), 36–42. <https://doi.org/10.1176/appi.ajp.159.1.36>. ***
- Biederman, J., Petty, C. R., Doyle, A. E., Spencer, T., Henderson, C. S., Marion, B., Fried, R., & Faraone, S. V. (2008). Stability of executive function deficits in girls with ADHD: A prospective longitudinal followup study into adolescence. *Developmental Neuropsychology*, 33(1), 44–61. <https://doi.org/10.1080/87565640701729755>
- Blanken, L. M. E., White, T., Mous, S. E., Basten, M., Muetzel, R. L., Jaddoe, V. W. V., Wals, M., van der Ende, J., Verhulst, F. C., & Tiemeier, H. (2017). Cognitive functioning

- in children with internalising, externalising and dysregulation problems: A population-based study. *European Child and Adolescent Psychiatry*, 26(4), 445–456.
<https://doi.org/10.1007/s00787-016-0903-9>
- Bonafina, M. A., Newcorn, J. H., McKay, K. E., Koda, V. H., & Halperin, J. M. (2000). ADHD and reading disabilities: A cluster analytic approach for distinguishing subgroups. *Journal of Learning Disabilities*, 33(3), 297–307.
<https://doi.org/10.1177/002221940003300307>. ***
- Bono, R., Blanca, M. J., Arnau, J., & Gómez-Benito, J. (2017). Non-normal distributions commonly used in health, education, and social sciences: A systematic review. *Frontiers in Psychology*, 8, 1602. <https://doi.org/10.3389/fpsyg.2017.01602>
- Bonti, E., Zerva, I. K., Koundourou, C., & Sofologi, M. (2024). The high rates of comorbidity among neurodevelopmental disorders: Reconsidering the clinical utility of distinct diagnostic categories. *Journal of Personalized Medicine*, 14(3), 300.
<https://doi.org/10.3390/jpm14030300>
- Booth, J. N., Boyle, J. M. E., & Kelly, S. W. (2010). Do tasks make a difference? Accounting for heterogeneity of performance of children with reading difficulties on tasks of executive function: Findings from a meta-analysis. *The British Journal of Developmental Psychology*, 28(1), 133–176. <https://doi.org/10.1348/026151009x485432>
- Borenstein, M. (2023). How to understand and report heterogeneity in a meta-analysis: The difference between I-squared and prediction intervals. *Integrative Medicine Research*, 12(4), Article 101014. <https://doi.org/10.1016/j.imr.2023.101014>
- Borenstein, M., Hedges, L. V., Higgins, J. P. T., & Rothstein, H. R. (2009). *Introduction to meta-analysis*. John Wiley & Sons. <https://doi.org/10.1002/9780470743386>
- Borsboom, D., Deserno, M. K., Rhemtulla, M., Epskamp, S., Fried, E. I., McNally, R. J., Robinaugh, D. J., Perugini, M., Dalege, J., Costantini, G., Isvoranu, A.-M., Wysocki, A. C., van Borkulo, C. D., van Bork, R., & Waldorp, L. J. (2021). Network analysis of multivariate data in psychological science. *Nature Reviews Methods Primers*, 1(1), 58.
<https://doi.org/10.1038/s43586-021-00055-w>
- Boucher, J., Mayes, A., & Bigham, S. (2012). Memory in autistic spectrum disorder. *Psychological Bulletin*, 138(3), 458–496. <https://doi.org/10.1037/a0026869>
- Boulton, K. A., Coghill, D., Silove, N., Pellicano, E., Whitehouse, A. J. O., Bellgrove, M. A., Rinehart, N. J., Lah, S., Redoblado-Hodge, M.-A., Badawi, N., Heussler, H., Rogerson, N., Burns, J., Farrar, M. A., Nanan, R., Novak, I., Goldwater, M. B., Munro, N., Togher, L., . . . & Guastella, A. J. (2021). A national harmonised data collection network for neurodevelopmental disorders: A transdiagnostic assessment protocol for neurodevelopment, mental health, functioning and well-being. *JCPP Advances*, 1(4), Article e12048. <https://doi.org/10.1002/jcv2.12048>

- Bowler, D. M., Gaigg, S. B., & Lind, S. (2011). Memory in autism: Binding, self and brain. In I. Roth & P. Rezaie (Eds.), *Researching the autism spectrum: Contemporary perspectives* (pp. 316–346). Cambridge University Press.
<https://doi.org/10.1017/CBO9780511973918.013>
- Boyes, M. E., Leitão, S., Claessen, M., Badcock, N. A., & Nayton, M. (2020). Correlates of externalising and internalising problems in children with dyslexia: An analysis of data from clinical casefiles. *Australian Psychologist*, 55(1), 62–72.
<https://doi.org/10.1111/ap.12409>
- Braem, S., & Egner, T. (2018). Getting a grip on cognitive flexibility. *Current Directions in Psychological Science*, 27(6), 470–476. <https://doi.org/10.1177/0963721418787475>
- Brandenburg, J., Huschka, S. S., Visser, L., & Hasselhorn, M. (2021). Are different types of learning disorder associated with distinct cognitive functioning profiles? *Frontiers in Psychology*, 12, Article 725374. <https://doi.org/10.3389/fpsyg.2021.725374>. ***
- Brimo, K., Dinkler, L., Gillberg, C., Lichtenstein, P., Lundström, S., & Åsberg Johnels, J. (2021). The co-occurrence of neurodevelopmental problems in dyslexia. *Dyslexia*, 27(3), 277–293. <https://doi.org/10.1002/dys.1681>. ***
- Brown Waesche, J. S., Schatschneider, C., Maner, J. K., Ahmed, Y., & Wagner, R. K. (2011). Examining agreement and longitudinal stability among traditional and RTI-based definitions of reading disability using the affected-status agreement statistic. *Journal of Learning Disabilities*, 44(3), 296–307. <https://doi.org/10.1177/0022219410392048>
- Bryant, A., Guy, J., CALM Team, & Holmes, J. (2020). The strengths and difficulties questionnaire predicts concurrent mental health difficulties in a transdiagnostic sample of struggling learners. *Frontiers in Psychology*, 11, Article 587821.
<https://doi.org/10.3389/fpsyg.2020.587821>
- Cai, R. Y., Hardan, A. Y., Phillips, J. M., Frazier, T. W., & Uljarević, M. (2021). Brief report: Emotion regulation influences on internalizing and externalizing symptoms across the normative-clinical continuum. *Frontiers in Psychiatry*, 12, Article 693570.
<https://doi.org/10.3389/fpsyg.2021.693570>
- Cai, R. Y., Richdale, A. L., Uljarević, M., Dissanayake, C., & Samson, A. C. (2018). Emotion regulation in autism spectrum disorder: Where we are and where we need to go. *Autism Research: Official Journal of the International Society for Autism Research*, 11(7), 962–978. <https://doi.org/10.1002/aur.1968>
- Caldani, S., Acquaviva, E., Moscoso, A., Peyre, H., Delorme, R., & Bucci, M. P. (2022). Reading performance in children with ADHD: An eye-tracking study. *Annals of Dyslexia*, 72(3), 552–565. <https://doi.org/10.1007/s11881-022-00269-x>. ***
- Callard, F., Rose, D., & Wykes, T. (2012). Close to the bench as well as at the bedside: Involving service users in all phases of translational research. *Health Expectations: An*

International Journal of Public Participation in Health Care and Health Policy, 15(4), 389–400. <https://doi.org/10.1111/j.1369-7625.2011.00681.x>

- Camm-Crosbie, L., Bradley, L., Shaw, R., Baron-Cohen, S., & Cassidy, S. (2019). ‘People like me don’t get support’: Autistic adults’ experiences of support and treatment for mental health difficulties, self-injury and suicidality. *Autism: The International Journal of Research and Practice*, 23(6), 1431–1441. <https://doi.org/10.1177/1362361318816053>
- Canela, C., Buadze, A., Dube, A., Eich, D., & Liebreinz, M. (2017). Skills and compensation strategies in adult ADHD—A qualitative study. *PLOS One*, 12(9), Article e0184964. <https://doi.org/10.1371/journal.pone.0184964>
- Capano, L., Minden, D., Chen, S. X., Schacher, R. J., & Ickowicz, A. (2008). Mathematical learning disorder in school-age children with attention-deficit hyperactivity disorder. *Canadian Journal of Psychiatry. Revue Canadienne de Psychiatrie*, 53(6), 392–399. <https://doi.org/10.1177/070674370805300609>. ***
- Capri, T., Martino, G., Giannatiempo, S., Semino, M., & Fabio, R. A. (2019). Attention, problem solving and decision making in adult subjects with ADHD. *Journal of Clinical and Developmental Psychology*, 1(1).
- Carawan, L. W., Nalavany, B. A., & Jenkins, C. (2016). Emotional experience with dyslexia and self-esteem: The protective role of perceived family support in late adulthood. *Aging and Mental Health*, 20(3), 284–294. <https://doi.org/10.1080/13607863.2015.1008984>
- Carlozzi, N. E., Tulskey, D. S., Chiaravalloti, N. D., Beaumont, J. L., Weintraub, S., Conway, K., & Gershon, R. C. (2014). NIH toolbox cognitive battery (NIHTB-CB): The NIHTB pattern comparison processing speed test. *Journal of the International Neuropsychological Society*, 20(6), 630–641. <https://doi.org/10.1017/S1355617714000319>
- Carmine Pastura, G. M. C., Mattos, P., & Campos Araújo, A. P. Q. C. (2009). Academic performance in ADHD when controlled for comorbid learning disorders, family income, and parental education in Brazil. *Journal of Attention Disorders*, 12(5), 469–473. <https://doi.org/10.1177/1087054708320401>. ***
- Carroll, J. M., Maughan, B., Goodman, R., & Meltzer, H. (2005). Literacy difficulties and psychiatric disorders: Evidence for comorbidity. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 46(5), 524–532. <https://doi.org/10.1111/j.1469-7610.2004.00366.x>. ***
- Cartwright, K. B., Coppage, E. A., Lane, A. B., Singleton, T., Marshall, T. R., & Bentivegna, C. (2017). Cognitive flexibility deficits in children with specific reading comprehension difficulties. *Contemporary Educational Psychology*, 50, 33–44. <https://doi.org/10.1016/j.cedpsych.2016.01.003>
- Casaleto, K. B., Umlauf, A., Beaumont, J., Gershon, R., Slotkin, J., Akshoomoff, N., & Heaton, R. K. (2015). Demographically corrected normative standards for the English

- version of the NIH toolbox cognition battery. *Journal of the International Neuropsychological Society: JINS*, 21(5), 378–391. <https://doi.org/10.1017/S1355617715000351>
- Casey, M. B., Cohen, M., Schuerholz, L. J., Singer, H. S., & Denckla, M. B. (2000). Language-based cognitive functioning in parents of offspring with ADHD comorbid for Tourette syndrome or learning disabilities. *Developmental Neuropsychology*, 17(1), 85–110. https://doi.org/10.1207/S15326942DN1701_06. ***
- Caspi, A., Houts, R. M., Ambler, A., Danese, A., Elliott, M. L., Hariri, A., Harrington, H., Hogan, S., Poulton, R., Ramrakha, S., Rasmussen, L. J. H., Reuben, A., Richmond-Rakerd, L., Sugden, K., Wertz, J., Williams, B. S., & Moffitt, T. E. (2020). Longitudinal assessment of mental health disorders and comorbidities across 4 decades among participants in the Dunedin birth cohort study. *JAMA Network Open*, 3(4), Article e203221. <https://doi.org/10.1001/jamanetworkopen.2020.3221>
- Castro, D., Lopes, P., Araújo, A. S., Ferreira, F., Rodrigues, A. R., Cardoso, J., Ferreira-Santos, F., & Ferreira, T. B. (2025). The differential impact of processing speed and cognitive flexibility on cognitive emotion regulation strategies and depression. *Journal of Affective Disorders*, 379, 567–575. <https://doi.org/10.1016/j.jad.2025.02.106>
- Caye, A., Rocha, T. B.-M., Anselmi, L., Murray, J., Menezes, A. M. B., Barros, F. C., Gonçalves, H., Wehrmeister, F., Jensen, C. M., Steinhausen, H.-C., Swanson, J. M., Kieling, C., & Rohde, L. A. (2016). Attention-deficit/hyperactivity disorder trajectories from childhood to young adulthood: Evidence from a birth cohort supporting a late-onset syndrome. *JAMA Psychiatry*, 73(7), 705–712. <https://doi.org/10.1001/jamapsychiatry.2016.0383>
- Chadwick, O., Taylor, E., Taylor, A., Heptinstall, E., & Danckaerts, M. (1999). Hyperactivity and reading disability: A longitudinal study of the nature of the association. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 40(7), 1039–1050. <https://doi.org/10.1111/1469-7610.00522>. ***
- Chalmpé, M., & Vlachos, F. (2024). Are there distinct subtypes of developmental dyslexia? *Frontiers in Behavioral Neuroscience*, 18, Article 1512892. <https://doi.org/10.3389/fnbeh.2024.1512892>
- Chan, J. Y.-L., Leow, S. M. H., Bea, K. T., Cheng, W. K., Phoong, S. W., Hong, Z.-W., & Chen, Y.-L. (2022). Mitigating the multicollinearity problem and its machine learning approach: A review. *Mathematics*, 10(8), 1283. <https://doi.org/10.3390/math10081283>
- Chan, W. S. R., Hung, S. F., Liu, S. N., & Lee, C. K. K. (2008). Cognitive profiling in Chinese developmental dyslexia with attention-deficit/hyperactivity disorders. *Reading and Writing*, 21(6), 661–674. <https://doi.org/10.1007/s11145-007-9084-8>. ***
- Chapman, R. (2021). Neurodiversity and the social ecology of mental functions. *Perspectives on Psychological Science*, 16(6), 1360–1372. <https://doi.org/10.1177/1745691620959833>

- Cheema, H. A., Shahid, A., Ehsan, M., & Ayyan, M. (2022). The misuse of funnel plots in meta-analyses of proportions: Are they really useful? *Clinical Kidney Journal*, 15(6), 1209–1210. <https://doi.org/10.1093/ckj/sfac035>
- Cheung, C. H. M., Rijdsdijk, F., McLoughlin, G., Faraone, S. V., Asherson, P., & Kuntsi, J. (2015). Childhood predictors of adolescent and young adult outcome in ADHD. *Journal of Psychiatric Research*, 62, 92–100. <https://doi.org/10.1016/j.jpsychires.2015.01.011>
- Claesdotter, E., Cervin, M., Åkerlund, S., Råstam, M., & Lindvall, M. (2018). The effects of ADHD on cognitive performance. *Nordic Journal of Psychiatry*, 72(3), 158–163. <https://doi.org/10.1080/08039488.2017.1402951>
- Clopper, C. J., & Pearson, E. S. (1934). The use of confidence or fiducial limits illustrated in the case of the binomial. *Biometrika*, 26(4), 404–413. <https://doi.org/10.1093/biomet/26.4.404>
- Cochran, W. G. (1954). Some methods for strengthening the common χ^2 tests. *Biometrics*, 10(4), 417–451. <https://doi.org/10.2307/3001616>
- Condo, J. S., Chan, E. S. M., & Kofler, M. J. (2022). Examining the effects of ADHD symptoms and parental involvement on children's academic achievement. *Research in Developmental Disabilities*, 122, Article 104156. <https://doi.org/10.1016/j.ridd.2021.104156>. ***
- Conners, C. K., Pitkanen, J., & Rzepa, S. R. (2011). Conners comprehensive behavior rating Scale. In J. S. Kreutzer, J. DeLuca, & B. Caplan (Eds.), *Encyclopedia of clinical neuropsychology* (pp. 678–680). Springer. https://doi.org/10.1007/978-0-387-79948-3_1536
- Conway, C. C., Forbes, M. K., South, S. C., & HiTOP Consortium. (2022). A Hierarchical Taxonomy of Psychopathology (HiTOP) primer for mental health researchers. *Clinical Psychological Science*, 10(2), 236–258. <https://doi.org/10.1177/21677026211017834>
- Costello, E. J., Mustillo, S., Erkanli, A., Keeler, G., & Angold, A. (2003). Prevalence and development of psychiatric disorders in childhood and adolescence. *Archives of General Psychiatry*, 60(8), 837–844. <https://doi.org/10.1001/archpsyc.60.8.837>
- Crawford, S. G., Kaplan, B. J., & Dewey, D. (2006). Effects of coexisting disorders on cognition and behavior in children with ADHD. *Journal of Attention Disorders*, 10(2), 192–199. <https://doi.org/10.1177/1087054706289924>. ***
- Crippa, A., Marzocchi, G. M., Piroddi, C., Besana, D., Giribone, S., Vio, C., Maschietto, D., Fornaro, E., Repossi, S., & Sora, M. L. (2015). An integrated model of executive functioning is helpful for understanding ADHD and associated disorders. *Journal of Attention Disorders*, 19(6), 455–467. <https://doi.org/10.1177/1087054714542000>. ***
- Csardi, G., & Nepusz, T. (2006). The igraph software. *Complex Systems*, 1695, 1–9.

- Cuthbert, B. N. (2014). The RDoC framework: Facilitating transition from ICD/DSM to dimensional approaches that integrate neuroscience and psychopathology. *World Psychiatry*, 13(1), 28–35. <https://doi.org/10.1002/wps.20087>
- Cuthbert, B. N. (2022). Research domain criteria (RDoC): Progress and potential. *Current Directions in Psychological Science*, 31(2), 107–114. <https://doi.org/10.1177/09637214211051363>
- Cuthbert, B. N., & Kozak, M. J. (2013). Constructing constructs for psychopathology: The NIMH research domain criteria. *Journal of Abnormal Psychology*, 122(3), 928–937. <https://doi.org/10.1037/a0034028>
- Czamara, D., Tiesler, C. M. T., Kohlböck, G., Berdel, D., Hoffmann, B., Bauer, C.-P., Koletzko, S., Schaaf, B., Lehmann, I., Herbarth, O., von Berg, A., Müller-Myhsok, B., Schulte-Körne, G., & Heinrich, J. (2013). Children with ADHD symptoms have a higher risk for reading, spelling and math difficulties in the GINIplus and LISApplus cohort studies. *PLOS One*, 8(5), Article e63859. <https://doi.org/10.1371/journal.pone.0063859>.

- D'Agati, E., Curatolo, P., & Mazzone, L. (2019). Comorbidity between ADHD and anxiety disorders across the lifespan. *International Journal of Psychiatry in Clinical Practice*, 23(4), 238–244. <https://doi.org/10.1080/13651501.2019.1628277>
- Da Silveira, L. T. Y. D., Ferreira, J. C., & Patino, C. M. (2023). Mixed-effects model: A useful statistical tool for longitudinal and cluster studies. *Jornal Brasileiro de Pneumologia: Publicacao Oficial da Sociedade Brasileira de Pneumologia e Tisiologia*, 49(2), Article e20230137. <https://doi.org/10.36416/1806-3756/e20230137>
- Dalglish, T., Black, M., Johnston, D., & Bevan, A. (2020). Transdiagnostic approaches to mental health problems: Current status and future directions. *Journal of Consulting and Clinical Psychology*, 88(3), 179–195. <https://doi.org/10.1037/ccp0000482>
- Dalmajer, E. S., Nord, C. L., & Astle, D. E. (2022). Statistical power for cluster analysis. *BMC Bioinformatics*, 23(1), 205. <https://doi.org/10.1186/s12859-022-04675-1>
- Darweesh, A. M., Elserogy, Y. M., Khalifa, H., Gabra, R. H., & El-Ghafour, M. A. (2020). Psychiatric comorbidity among children and adolescents with dyslexia. *Middle East Current Psychiatry*, 27, 1–9. <https://doi.org/10.1186/s43045-020-00035-y> ***
- Davidson, F., Cherry, K., & Corkum, P. (2016). Validating the behavior rating inventory of executive functioning for children with ADHD and their typically developing peers. *Applied Neuropsychology. Child*, 5(2), 127–137. <https://doi.org/10.1080/21622965.2015.1021957>
- De Groot, B. J. A., Van den Bos, K. P., Van der Meulen, B. F., & Minnaert, A. E. M. G. (2017). Rapid naming and phonemic awareness in children with or without reading disabilities and/or ADHD. *Journal of Learning Disabilities*, 50(2), 168–179. <https://doi.org/10.1177/0022219415609186>. ***

- de Jong, C. G. W., Licht, R., Sergeant, J. A., & Oosterlaan, J. (2012). RD, ADHD, and their comorbidity from a dual route perspective. *Child Neuropsychology: A Journal on Normal and Abnormal Development in Childhood and Adolescence*, 18(5), 467–486. <https://doi.org/10.1080/09297049.2011.625354>. ***
- de Jong, C. G. W., Van De Voorde, S., Roeyers, H., Raymaekers, R., Oosterlaan, J., & Sergeant, J. A. (2009). How distinctive are ADHD and RD? Results of a double dissociation study. *Journal of Abnormal Child Psychology*, 37(7), 1007–1017. <https://doi.org/10.1007/s10802-009-9328-y>. ***
- de Oliveira, D. G., da Silva, P. B., Dias, N. M., Seabra, A. G., & Macedo, E. C. (2014). Reading component skills in dyslexia: Word recognition, comprehension and processing speed. *Frontiers in Psychology*, 5, 1339. <https://doi.org/10.3389/fpsyg.2014.01339>
- Deeks, J. J., Higgins, J. P., Altman, D. G., & Cochrane Statistical Methods Group. (2019). Analysing data and undertaking meta-analyses. In J. P. T. Higgins, J. Thomas, J. Chandler, M. Cumpston, T. Li, M. J. Page, V. A. Welch (Eds.), *Cochrane handbook for systematic reviews of interventions* (pp. 241–284). John Wiley & Sons. <https://doi.org/10.1002/9781119536604.ch10>
- Del’Homme, M., Kim, T. S., Loo, S. K., Yang, M. H., & Smalley, S. L. (2007). Familial association and frequency of learning disabilities in ADHD sibling pair families. *Journal of Abnormal Child Psychology*, 35(1), 55–62. <https://doi.org/10.1007/s10802-006-9080-5>. ***
- Dellapiazza, F., Audras-Torrent, L., Michelon, C., & Baghdadli, A. (2021). Clinical characteristics of children with ASD and comorbid ADHD: Association with social impairment and externalizing and internalizing behaviours. *Research in Developmental Disabilities*, 113, Article 103930. <https://doi.org/10.1016/j.ridd.2021.103930>
- Den Hartigh, R. J. R., Van Dijk, M. W. G., Steenbeek, H. W., & Van Geert, P. L. C. (2016). A dynamic network model to explain the development of excellent human performance. *Frontiers in Psychology*, 7, 532. <https://doi.org/10.3389/fpsyg.2016.00532>
- Dewey, D., Kaplan, B. J., Crawford, S. G., & Fisher, G. C. (2001). Predictive accuracy of the Wide Range Assessment of Memory and Learning in children with attention deficit hyperactivity disorder and reading difficulties. *Developmental Neuropsychology*, 19(2), 173–189. https://doi.org/10.1207/S15326942DN1902_3. ***
- Diamond, A. (2013). Executive functions. *Annual Review of Psychology*, 64(1), 135–168. <https://doi.org/10.1146/annurev-psych-113011-143750>
- Dikmen, S. S., Bauer, P. J., Weintraub, S., Mungas, D., Slotkin, J., Beaumont, J. L., Gershon, R., Temkin, N. R., & Heaton, R. K. (2014). Measuring episodic memory across the lifespan: NIH toolbox picture sequence memory test. *Journal of the International Neuropsychological Society*, 20(6), 611–619. <https://doi.org/10.1017/S1355617714000460>

- Ding, C., & He, X. (2004, July). K-means clustering via principal component analysis. In *Proceedings of the Twenty-First International Conference on Machine Learning* (p. 29). ACM Press. <https://doi.org/10.1145/1015330.1015408>
- Dito, G. A., Safitri, A., Afendi, F. M., Anisa, R., Salim, A., & Sartono, B. (2019, July). Graphical user interface (GUI) for the least absolute shrinkage and selection operator (LASSO) regression. In *IOP Conference Series: Earth and Environmental Science*, 299(1). <https://doi.org/10.1088/1755-1315/299/1/012031>
- Doyle, C., Smeaton, A. F., Roche, R. A. P., & Boran, L. (2018). Inhibition and updating, but not switching, predict developmental dyslexia and individual variation in reading ability. *Frontiers in Psychology*, 9, 795. <https://doi.org/10.3389/fpsyg.2018.00795>
- Dubois, M., Kyllingsbaek, S., Prado, C., Musca, S. C., Peiffer, E., Lassus-Sangosse, D., & Valdois, S. (2010). Fractionating the multi-character processing deficit in developmental dyslexia: Evidence from two case studies. *Cortex; a Journal Devoted to the Study of the Nervous System and Behavior*, 46(6), 717–738. <https://doi.org/10.1016/j.cortex.2009.11.002>
- Duff, D. M., Hendricks, A. E., Fitton, L., & Adlof, S. M. (2023). Reading and math achievement in children with dyslexia, developmental language disorder, or typical development: Achievement gaps persist from second through fourth grades. *Journal of Learning Disabilities*, 56(5), 371–391. <https://doi.org/10.1177/00222194221105515>
- DuPaul, G. J., Gormley, M. J., & Laracy, S. D. (2013). Comorbidity of LD and ADHD: Implications of DSM-5 for assessment and treatment. *Journal of Learning Disabilities*, 46(1), 43–51. <https://doi.org/10.1177/0022219412464351>
- Duranović, M., Begić, L., Avdić, Z., & Klebić, J. (2023). Impulsivity in children with dyslexia. *Research in Education and Rehabilitation*, 6(2), 175–190. <https://doi.org/10.51558/2744-1555.2023.6.2.175>
- Duval, S., & Tweedie, R. (2000). Trim and fill: A simple funnel-plot-based method of testing and adjusting for publication bias in meta-analysis. *Biometrics*, 56(2), 455–463. <https://doi.org/10.1111/j.0006-341x.2000.00455.x>
- Dwyer, P. (2022). The neurodiversity approach(es): What are they and what do they mean for researchers? *Human Development*, 66(2), 73–92. <https://doi.org/10.1159/000523723>
- Dykman, R. A., & Ackerman, P. T. (1991). Attention deficit disorder and specific reading disability: Separate but often overlapping disorders. *Journal of Learning Disabilities*, 24(2), 96–103. <https://doi.org/10.1177/002221949102400206>. ***
- Dziak, J. J., Coffman, D. L., Lanza, S. T., Li, R., & Jermini, L. S. (2020). Sensitivity and specificity of information criteria. *Briefings in Bioinformatics*, 21(2), 553–565. <https://doi.org/10.1093/bib/bbz016>

- Efron, D., Sciberras, E., Anderson, V., Hazell, P., Ukoumunne, O. C., Jongeling, B., Schilpzand, E. J., Bisset, M., & Nicholson, J. M. (2014). Functional status in children with ADHD at age 6–8: A controlled community study. *Pediatrics*, 134(4), e992–e1000. <https://doi.org/10.1542/peds.2014-1027-e1000>. ***
- Egger, M., Davey Smith, G., Schneider, M., & Minder, C. (1997). Bias in meta-analysis detected by a simple, graphical test. *BMJ*, 315(7109), 629–634. <https://doi.org/10.1136/bmj.315.7109.629>
- Eide, B. L., & Eide, F. F. (2011). *The Dyslexic Advantage: Unlocking the hidden potential of the dyslexic brain*. Hudson Street, New York, Press.
- El Sheikh, M. M., El Missiry, M. A., Hatata, H. A., Sabry, W. M., El Fiky, A. A. A., & Essawi, H. I. (2016). Frequency of occurrence of specific reading disorder and associated psychiatric comorbidity in a sample of Egyptian primary school students. *Child and Adolescent Mental Health*, 21(4), 209–216. <https://doi.org/10.1111/camh.12174>. ***
- Elgendi, M. M., Stewart, S. H., MacKay, E. J., & Deacon, S. H. (2021). Two aspects of psychological functioning in undergraduates with a history of reading difficulties: Anxiety and self-efficacy. *Annals of Dyslexia*, 71(1), 84–102. <https://doi.org/10.1007/s11881-021-00223-3>
- Ellis Weismer, S., Kaushanskaya, M., Larson, C., Mathée, J., & Bolt, D. (2018). Executive function skills in school-age children with autism spectrum disorder: Association with language abilities. *Journal of Speech, Language, and Hearing Research: JSLHR*, 61(11), 2641–2658. https://doi.org/10.1044/2018_JSLHR-L-RSAUT-18-0026
- EndNote Team. (2013). EndNote (Version X9.3.3) [Computer software]. Clarivate.
- England-Mason, G. (2020). Emotion regulation as a transdiagnostic feature in children with neurodevelopmental disorders. *Current Developmental Disorders Reports*, 7(3), 130–138. <https://doi.org/10.1007/s40474-020-00200-2>
- Epskamp, S., & Fried, E. I. (2018). A tutorial on regularized partial correlation networks. *Psychological Methods*, 23(4), 617–634. <https://doi.org/10.1037/met0000167>
- Epskamp, S., Borsboom, D., & Fried, E. I. (2018). Estimating psychological networks and their accuracy: A tutorial paper. *Behavior Research Methods*, 50(1), 195–212. <https://doi.org/10.3758/s13428-017-0862-1>
- Epskamp, S., Cramer, A. O. J., Waldorp, L. J., Schmittmann, V. D., & Borsboom, D. (2012). qgraph: Network visualizations of relationships in psychometric data. *Journal of Statistical Software*, 48(4), 1–18. <https://doi.org/10.18637/jss.v048.i04>
- Epskamp, S., Kruis, J., & Marsman, M. (2017). Estimating psychopathological networks: Be careful what you wish for. *PLOS One*, 12(6), Article e0179891. <https://doi.org/10.1371/journal.pone.0179891>

- Epskamp, S., Waldorp, L. J., Möttus, R., & Borsboom, D. (2018). The Gaussian graphical model in cross-sectional and time-series data. *Multivariate Behavioral Research*, 53(4), 453–480. <https://doi.org/10.1080/00273171.2018.1454823>
- Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. *Perception and Psychophysics*, 16(1), 143–149. <https://doi.org/10.3758/BF03203267>
- Español-Martín, G., Pagerols, M., Prat, R., Rivas, C., Ramos-Quiroga, J. A., Casas, M., & Bosch, R. (2023). The impact of attention-deficit/hyperactivity disorder and specific learning disorders on academic performance in Spanish children from a low-middle- and a high-income population. *Frontiers in Psychiatry*, 14, Article 1136994. <https://doi.org/10.3389/fpsyt.2023.1136994>. ***
- Evans, T. S., & Chen, B. (2022). Linking the network centrality measures closeness and degree. *Communications Physics*, 5(1), 172. <https://doi.org/10.1038/s42005-022-00949-5>
- Everitt, B. S., Landau, S., Leese, M., & Stahl, D. (2011). *Cluster analysis* (5th ed.). John Wiley & Sons. <https://doi.org/10.1002/9780470977811>
- Fáisca, L., Reis, A., & Araújo, S. (2023). Cognitive subtyping of university students with dyslexia in a semi-transparent orthography: What can weaknesses and strengths tell us about compensation? *Journal of Cultural Cognitive Science*, 7(2), 121–136. <https://doi.org/10.1007/s41809-023-00124-4>
- Fan, L., & Wang, Y. (2023). The relationship between executive functioning and attention deficit hyperactivity disorder in young children: A cross-lagged study. *Current Psychology*, 42(25), 21375–21383. <https://doi.org/10.1007/s12144-022-03233-5>
- Farah, R., Ionta, S., & Horowitz-Kraus, T. (2021). Neuro-behavioral correlates of executive dysfunctions in dyslexia over development from childhood to adulthood. *Frontiers in Psychology*, 12, Article 708863. <https://doi.org/10.3389/fpsyg.2021.708863>
- Faraone, S. V., & Biederman, J. (2016). Can attention-deficit/hyperactivity disorder onset occur in adulthood? *JAMA Psychiatry*, 73(7), 655–656. <https://doi.org/10.1001/jamapsychiatry.2016.0400>
- Faraone, S. V., Biederman, J., & Mick, E. (2006). The age-dependent decline of attention deficit hyperactivity disorder: A meta-analysis of follow-up studies. *Psychological Medicine*, 36(2), 159–165. <https://doi.org/10.1017/S003329170500471X>
- Faraone, S. V., Biederman, J., Lehman, B. K., Spencer, T., Norman, D., Seidman, L. J., Kraus, I., Perrin, J., Chen, W. J., & Tsuang, M. T. (1993). Intellectual performance and school failure in children with attention deficit hyperactivity disorder and in their siblings. *Journal of Abnormal Psychology*, 102(4), 616–623. <https://doi.org/10.1037/0021-843X.102.4.616>. ***

- Faraone, S. V., Rostain, A. L., Blader, J., Busch, B., Childress, A. C., Connor, D. F., & Newcorn, J. H. (2019). Practitioner Review: Emotional dysregulation in attention-deficit/hyperactivity disorder—implications for clinical recognition and intervention. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 60(2), 133–150. <https://doi.org/10.1111/jcpp.12899>
- Farhat, L. C., Brentani, H., de Toledo, V. H. C., Shephard, E., Mattos, P., Baron-Cohen, S., Thapar, A., Casella, E., & Polanczyk, G. V. (2022). ADHD and autism symptoms in youth: A network analysis. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 63(2), 143–151. <https://doi.org/10.1111/jcpp.13436>
- Fernández-Andrés, M. I., Tejero, P., & Vélez-Calvo, X. (2021). Visual attention, orthographic word recognition, and executive functioning in children with ADHD, dyslexia, or ADHD + dyslexia. *Journal of Attention Disorders*, 25(7), 942–953. <https://doi.org/10.1177/1087054719864637> ***
- Finlay-Jones, A., Varcin, K., Leonard, H., Bosco, A., Alvares, G., & Downs, J. (2019). Very early identification and intervention for infants at risk of neurodevelopmental disorders: A transdiagnostic approach. *Child Development Perspectives*, 13(2), 97–103. <https://doi.org/10.1111/cdep.12319>
- Fleiss, J. L., Levin, B., & Paik, M. C. (2003). *Statistical methods for rates and proportions*. John Wiley & Sons. <https://doi.org/10.1002/0471445428>
- Fletcher-Watson, S., Brook, K., Hallett, S., Murray, F., & Crompton, C. J. (2021). Inclusive practices for neurodevelopmental research. *Current Developmental Disorders Reports*, 8(2), 88–97. <https://doi.org/10.1007/s40474-021-00227-z>
- Fletcher-Watson, S. (2022). Transdiagnostic research and the neurodiversity paradigm: Commentary on the transdiagnostic revolution in neurodevelopmental disorders by Astle et al. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 63(4), 418–420. <https://doi.org/10.1111/jcpp.13589>
- Fletcher, J. M., Lyon, G. R., Fuchs, L. S., & Barnes, M. A. (2019). *Learning disabilities: From identification to intervention* (2nd ed.). Guilford Press.
- Forbes, M. K., Tackett, J. L., Markon, K. E., & Krueger, R. F. (2016). Beyond comorbidity: Toward a dimensional and hierarchical approach to understanding psychopathology across the life span. *Development and Psychopathology*, 28(4pt1, Pt. 1), 971–986. <https://doi.org/10.1017/S0954579416000651>
- Fortes, I. S., Paula, C. S., Oliveira, M. C., Bordin, I. A., de Jesus Mari, J., & Rohde, L. A. (2016). A cross-sectional study to assess the prevalence of DSM-5 specific learning disorders in representative school samples from the second to sixth grade in Brazil. *European Child and Adolescent Psychiatry*, 25(2), 195–207. <https://doi.org/10.1007/s00787-015-0708-2>. ***

- Fougnie, D. (2008). The relationship between attention and working memory. *New Research on Short-Term Memory*, 1, 45.
- Frades, I., & Matthiesen, R. (2010). Overview on techniques in cluster analysis. *Methods in Molecular Biology (Clifton, NJ)*, 593, 81–107. https://doi.org/10.1007/978-1-60327-194-3_5
- Francés, L., Caules, J., Ruiz, A., Soler, C. V., Hervás, A., Fernández, A., Rodríguez-Quiroga, A., & Quintero, J. (2023). An approach for prevention planning based on the prevalence and comorbidity of neurodevelopmental disorders in 6-year-old children receiving primary care consultations on the island of Menorca. *BMC Pediatrics*, 23(1), 32. <https://doi.org/10.1186/s12887-023-03844-2>. ***
- Friedman, L., & Sterling, A. (2019). A review of language, executive function, and intervention in autism spectrum disorder. *Seminars in Speech and Language*, 40(4), 291–304. <https://doi.org/10.1055/s-0039-1692964>
- Furuya-Kanamori, L., Barendregt, J. J., & Doi, S. A. R. (2018). A new improved graphical and quantitative method for detecting bias in meta-analysis. *International Journal of Evidence-Based Healthcare*, 16(4), 195–203. <https://doi.org/10.1097/XEB.0000000000000141>
- Games, P. A., & Howell, J. F. (1976). Pairwise Multiple Comparison Procedures with Unequal N's and/or Variances: A Monte Carlo Study. *Journal of Educational Statistics*, 1(2), 113–125. <https://doi.org/10.3102/10769986001002113>
- Gao, C. X., Dwyer, D., Zhu, Y., Smith, C. L., Du, L., Filia, K. M., Bayer, J., Menssink, J. M., Wang, T., Bergmeir, C., Wood, S., & Cotton, S. M. (2023). An overview of clustering methods with guidelines for application in mental health research. *Psychiatry Research*, 327, Article 115265. <https://doi.org/10.1016/j.psychres.2023.115265>
- Gartlehner, G., Affengruber, L., Titscher, V., Noel-Storr, A., Dooley, G., Ballarini, N., & König, F. (2020). Single-reviewer abstract screening missed 13 percent of relevant studies: A crowd-based, randomized controlled trial. *Journal of Clinical Epidemiology*, 121, 20–28. <https://doi.org/10.1016/j.jclinepi.2020.01.005>
- Garwood, J. D., & McKenna, J. W. (2020). Vocabulary interventions for students identified with emotional disturbance: A literature review and a call to action. *Education and Treatment of Children*, 43(2), 187–199. <https://doi.org/10.1007/s43494-020-00016-0>
- Gathercole, S. E., Brown, L., & Pickering, S. J. (2003). Working memory assessments at school entry as longitudinal predictors of National Curriculum attainment levels. *Educational and Child Psychology*, 20(3), 109–122. <https://doi.org/10.53841/bpsecp.2003.20.3.109>
- Geiger, B. C., & Kubin, G. (2012). Relative information loss in the PCA. IEEE information theory workshop. 562–566. IEEE.

- Germanò, E., Gagliano, A., & Curatolo, P. (2010). Comorbidity of ADHD and dyslexia. *Developmental Neuropsychology*, 35(5), 475–493. <https://doi.org/10.1080/87565641.2010.494748>
- Gershon, R. C., Cook, K. F., Mungas, D., Manly, J. J., Slotkin, J., Beaumont, J. L., & Weintraub, S. (2014). Language measures of the NIH Toolbox Cognition Battery. *Journal of the International Neuropsychological Society: JINS*, 20(6), 642–651. <https://doi.org/10.1017/S1355617714000411>
- Geurts, H., Sinzig, J., Booth, R., & Happé, F. (2014). Neuropsychological heterogeneity in executive functioning in autism spectrum disorders. *International Journal of Developmental Disabilities*, 60(3), 155–162. <https://doi.org/10.1179/2047387714Y.00000000047>
- Ghelani, K., Sidhu, R., Jain, U., & Tannock, R. (2004). Reading comprehension and reading related abilities in adolescents with reading disabilities and attention-deficit/hyperactivity disorder. *Dyslexia*, 10(4), 364–384. <https://doi.org/10.1002/dys.285>. ***
- Ghosh, S., & Halder, S. (2020). Emotional regulation and cognitive flexibility in young adults. *Journal of Psychosocial Research*, 15(2), 609–617. <https://doi.org/10.32381/JPR.2020.15.02.22>
- Gioia, G. A., Isquith, P. K., Guy, S. C., & Kenworthy, L. (2000). *Behavior Rating Inventory of Executive Function (BRIEF)* [Database record]. *APA PsycTests*. <https://doi.org/10.1037/t73087-000>
- Gjevik, E., Eldevik, S., Fjæran-Granum, T., & Sponheim, E. (2011). Kiddie-SADS reveals high rates of DSM-IV disorders in children and adolescents with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 41(6), 761–769. <https://doi.org/10.1007/s10803-010-1095-7>
- Gnanavel, S., Sharma, P., Kaushal, P., & Hussain, S. (2019). Attention deficit hyperactivity disorder and comorbidity: A review of literature. *World Journal of Clinical Cases*, 7(17), 2420–2426. <https://doi.org/10.12998/wjcc.v7.i17.2420>
- Goker, Z., Uneri, O. S., Guney, E., Dinc, G., & Hekim-Bozkurt, O. (2014). Clinical and sociodemographic features of children and adolescents with specific learning disorder (SLD). *American Journal of Psychiatry and Neuroscience*, 2(6), 90–95. <https://doi.org/10.11648/j.ajpn.20140206.12>. ***
- Goldin, D., Alvarez, L. M., & von Harscher, H. (2025). Cognitive flexibility: What mental health professionals need to know. *Journal of Psychosocial Nursing and Mental Health Services*, 63(4), 20–27. <https://doi.org/10.3928/02793695-20241205-03>
- Goldston, D. B., Walsh, A., Mayfield Arnold, E., Reboussin, B., Sergent Daniel, S., Erkanli, A., Nutter, D., Hickman, E., Palmes, G., Snider, E., & Wood, F. B. (2007). Reading problems, psychiatric disorders, and functional impairment from mid- to late

- adolescence. *Journal of the American Academy of Child and Adolescent Psychiatry*, 46(1), 25–32. <https://doi.org/10.1097/01.chi.0000242241.77302.f4>. ***
- Gooch, D., Snowling, M., & Hulme, C. (2011). Time perception, executive function and phonological skills in children with attention deficit hyperactivity disorder (ADHD) and reading disorder (RD). *European Child and Adolescent Psychiatry*, 19. ***
- Goodman, A., & Goodman, R. (2009). Strengths and difficulties questionnaire as a dimensional measure of child mental health. *Journal of the American Academy of Child and Adolescent Psychiatry*, 48(4), 400–403. <https://doi.org/10.1097/CHI.0b013e3181985068>
- Goodman, D. W., Cortese, S., & Faraone, S. V. (2024). Why is ADHD so difficult to diagnose in older adults? *Expert Review of Neurotherapeutics*, 24(10), 941–944. <https://doi.org/10.1080/14737175.2024.2385932>
- Goodman, R. (1997). The Strengths and Difficulties Questionnaire: A research note. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 38(5), 581–586. <https://doi.org/10.1111/j.1469-7610.1997.tb01545.x>
- Goodman, R. (2001). Psychometric properties of the strengths and difficulties questionnaire. *Journal of the American Academy of Child and Adolescent Psychiatry*, 40(11), 1337–1345. <https://doi.org/10.1097/00004583-200111000-00015>
- Gordon, R., Smith-Spark, J. H., Newton, E. J., & Henry, L. A. (2018). Executive function and academic achievement in primary school children: The use of task-related processing speed. *Frontiers in Psychology*, 9, 582. <https://doi.org/10.3389/fpsyg.2018.00582>
- Grant, M., Salsman, N. L., & Berking, M. (2018). The assessment of successful emotion regulation skills use: Development and validation of an English version of the Emotion Regulation Skills Questionnaire. *PLOS One*, 13(10), Article e0205095. <https://doi.org/10.1371/journal.pone.0205095>
- Graziano, P. A., & Garcia, A. (2016). Attention-deficit hyperactivity disorder and children's emotion dysregulation: A meta-analysis. *Clinical Psychology Review*, 46, 106–123. <https://doi.org/10.1016/j.cpr.2016.04.011>
- Greenacre, M., Groenen, P. J. F., Hastie, T., d'Enza, A. I., Markos, A., & Tuzhilina, E. (2022). Principal component analysis. *Nature Reviews Methods Primers*, 2(1), 100. <https://doi.org/10.1038/s43586-022-00184-w>
- Greenberg, D. L., & Verfaellie, M. (2010). Interdependence of episodic and semantic memory: Evidence from neuropsychology. *Journal of the International Neuropsychological Society*, 16(5), 748–753. <https://doi.org/10.1017/S1355617710000676>
- Gregg, N., Bandalos, D. L., Coleman, C., Davis, J. M., Robinson, K., & Blake, J. (2008). The validity of a battery of phonemic and orthographic awareness tasks for adults with and

- without dyslexia and attention deficit/hyperactivity disorder. *Remedial and Special Education*, 29(3), 175–190. <https://doi.org/10.1177/0741932508315951>. ***
- Grogan, K., Gormley, C. I., Rooney, B., Whelan, R., Kiiski, H., Naughton, M., & Bramham, J. (2018). Differential diagnosis and comorbidity of ADHD and anxiety in adults. *British Journal of Clinical Psychology*, 57(1), 99–115. <https://doi.org/10.1111/bjc.12156>
- Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology*, 85(2), 348–362. <https://doi.org/10.1037/0022-3514.85.2.348>
- Hall, S. J., Halperin, J. M., Schwartz, S. T., & Newcorn, J. H. (1997). Behavioral and executive functions in children with attention-deficit hyperactivity disorder and reading disability. *Journal of Attention Disorders*, 1(4), 235–243. <https://doi.org/10.1177/108705479700100404>. ***
- Harvey, A. G., Watkins, E., & Mansell, W. (2004). *Cognitive behavioural processes across psychological disorders: A transdiagnostic approach to research and treatment*. Oxford University Press.
- Hawkins, E., Gathercole, S., Astle, D., The Calm Team, & Holmes, J. (2016). Language problems and ADHD symptoms: How specific are the links? *Brain Sciences*, 6(4), 50. <https://doi.org/10.3390/brainsci6040050>
- Hechtman, L., Etcovitch, J., Platt, R., Arnold, L. E., Abikoff, H. B., Newcorn, J. H., Hoza, B., Hinshaw, S. P., Kraemer, H. C., Wells, K., Conners, K., Elliott, G., Greenhill, L. L., Jensen, P. S., March, J. S., Molina, B., Pelham, W. E., Severe, J. B., Swanson, J. M., . . . & Wigal, T. (2005). Does multimodal treatment of ADHD decrease other diagnoses? *Clinical Neuroscience Research*, 5(5–6), 273–282. <https://doi.org/10.1016/j.cnr.2005.09.007>. ***
- Heikkilä, R., Torppa, M., Aro, M., Närhi, V., & Ahonen, T. (2016). Double-deficit hypothesis in a clinical sample: Extension beyond reading. *Journal of Learning Disabilities*, 49(5), 546–560. <https://doi.org/10.1177/0022219415572895>. ***
- Helland, W. A., Posserud, M.-B., Helland, T., Heimann, M., & Lundervold, A. J. (2016). Language impairments in children with ADHD and in children with reading disorder. *Journal of Attention Disorders*, 20(7), 581–589. <https://doi.org/10.1177/1087054712461530>. ***
- Hens, K., & Van Goidsenhoven, L. (2022). Developmental diversity: Putting the development back into research about developmental conditions. *Frontiers in Psychiatry*, 13, Article 986732. <https://doi.org/10.3389/fpsy.2022.986732>
- Herrera Gutiérrez, E., Conesa Conesa, M. R., & López Ortuño, J. (2021). *Study of reading comprehension in students with ADHD*. ***.

- Higgins, J. P. T., & Thompson, S. G. (2002). Quantifying heterogeneity in a meta-analysis. *Statistics in Medicine*, 21(11), 1539–1558. <https://doi.org/10.1002/sim.1186>
- Higgins, J. P. T., Thomas, J., Chandler, J., Cumpston, M., Li, T., Page, M. J., & Welch, V. A. (Eds.). (2024). *Cochrane handbook for systematic reviews of interventions*. Cochrane. <https://www.training.cochrane.org/handbook> ((version 6.5). updated August 2024).
- Hinshaw, S. P. (1987). On the distinction between attentional deficits/hyperactivity and conduct problems/aggression in child psychopathology. *Psychological Bulletin*, 101(3), 443–463. <https://doi.org/10.1037/0033-2909.101.3.443>
- Hinshaw, S. P. (2002). Preadolescent girls with attention-deficit/hyperactivity disorder: I. Background characteristics, comorbidity, cognitive and social functioning, and parenting practices. *Journal of Consulting and Clinical Psychology*, 70(5), 1086–1098. <https://doi.org/10.1037/0022-006x.70.5.1086>. ***
- Ho, C. S.-H., Chan, D. W.-O., Leung, P. W. L., Lee, S.-H., & Tsang, S.-M. (2005). Reading-related cognitive deficits in developmental dyslexia, attention-deficit/hyperactivity disorder, and developmental coordination disorder among Chinese children. *Reading Research Quarterly*, 40(3), 318–337. <https://doi.org/10.1598/RRQ.40.3.2> ***
- Hobson, H. M., & Lee, A. (2023). Camouflaging in developmental language disorder: The views of Speech and Language pathologists and parents. *Communication Disorders Quarterly*, 44(4), 247–256. <https://doi.org/10.1177/15257401221120937>
- Holmes, J., Gathercole, S. E., & Dunning, D. L. (2010). Poor working memory: Impact and interventions. *Advances in Child Development and Behavior*, 39, 1–43. <https://doi.org/10.1016/b978-0-12-374748-8.00001-9>
- Holmes, J., Guy, J., Kievit, R. A., Bryant, A., Mareva, S., CALM Team, & Gathercole, S. E. (2021). Cognitive dimensions of learning in children with problems in attention, learning, and memory. *Journal of Educational Psychology*, 113(7), 1454–1480. <https://doi.org/10.1037/edu0000644>
- Horowitz-Kraus, T. (2016). Can the error-monitoring system differentiate ADHD from ADHD with reading disability? Reading and executive dysfunction as reflected in error monitoring. *Journal of Attention Disorders*, 20(10), 889–902. <https://doi.org/10.1177/1087054713488440>. ***
- Horowitz-Kraus, T., Hershey, A., Kay, B., & DiFrancesco, M. (2019). Differential effect of reading training on functional connectivity in children with reading difficulties with and without ADHD comorbidity. *Journal of Neurolinguistics*, 49, 93–108. <https://doi.org/10.1016/j.jneuroling.2018.09.002>. ***
- Howard, J., Herold, B., Major, S., Leahy, C., Ramseur, K., 2nd, Franz, L., Deaver, M., Vermeer, S., Carpenter, K. L., Murias, M., Huang, W. A., & Dawson, G. (2023). Associations between executive function and attention abilities and language and social

- communication skills in young autistic children. *Autism: The International Journal of Research and Practice*, 27(7), 2135–2144. <https://doi.org/10.1177/13623613231154310>
- Hoza, B. (2007). Peer functioning in children with ADHD. *Journal of Pediatric Psychology*, 32(6), 655–663. <https://doi.org/10.1093/jpepsy/jsm024>
- Huang, Y.-F., Chang, H.-L., Wu, Y.-Y., Huang, Y.-S., & Liang, H.-Y. (2007). Clinical characteristics of children with learning disorders in Taiwan. *Chang Gung Medical Journal*, 30(5), 423–429. ***.
- Hutt Vater, C., DiSalvo, M., Ehrlich, A., Parker, H., O'Connor, H., Faraone, S. V., & Biederman, J. (2024). ADHD in adults: Does age at diagnosis matter? *Journal of Attention Disorders*, 28(5), 614–624. <https://doi.org/10.1177/10870547231218450>
- Hwang, Y. I. J., Arnold, S., Srasuebkul, P., & Trollor, J. (2020). Understanding anxiety in adults on the autism spectrum: An investigation of its relationship with intolerance of uncertainty, sensory sensitivities and repetitive behaviours. *Autism: The International Journal of Research and Practice*, 24(2), 411–422. <https://doi.org/10.1177/1362361319868907>
- IBM Corporation. (2023). *IBM SPSS Statistics for windows* (version 29.0) [Computer software]. Armonk, NY.
- Insel, T., Cuthbert, B., Garvey, M., Heinssen, R., Pine, D. S., Quinn, K., Sanislow, C., & Wang, P. (2010). Research domain criteria (RDoC): Toward a new classification framework for research on mental disorders. *The American Journal of Psychiatry*, 167(7), 748–751. <https://doi.org/10.1176/appi.ajp.2010.09091379>
- Jacobson, L. A., Ryan, M., Martin, R. B., Ewen, J., Mostofsky, S. H., Denckla, M. B., & Mahone, E. M. (2011). Working memory influences processing speed and reading fluency in ADHD. *Child Neuropsychology: A Journal on Normal and Abnormal Development in Childhood and Adolescence*, 17(3), 209–224. <https://doi.org/10.1080/09297049.2010.532204>
- Jaisle, E. M., Groves, N. B., Black, K. E., & Kofler, M. J. (2023). Linking ADHD and ASD symptomatology with social impairment: The role of emotion dysregulation. *Research on Child and Adolescent Psychopathology*, 51(1), 3–16. <https://doi.org/10.1007/s10802-022-00982-6>
- Jakobson, A., & Kikas, E. (2007). Cognitive functioning in children with and without attention-deficit/hyperactivity disorder with and without comorbid learning disabilities. *Journal of Learning Disabilities*, 40(3), 194–202. <https://doi.org/10.1177/00222194070400030101>. ***
- Jakobsson Støre, S., Van Zalk, N., Granander Schwartz, W., Nilsson, V., & Tillfors, M. (2024). The relationship between social anxiety disorder and ADHD in adolescents and adults: A systematic review. *Journal of Attention Disorders*, 28(9), 1299–1319. <https://doi.org/10.1177/10870547241247448>

- Jensen, C. M., & Steinhausen, H.-C. (2015). Comorbid mental disorders in children and adolescents with attention-deficit/hyperactivity disorder in a large nationwide study. *Attention Deficit and Hyperactivity Disorders*, 7(1), 27–38. <https://doi.org/10.1007/s12402-014-0142-1>
- Johnson, B. D., Altmaier, E. M., & Richman, L. C. (1999). Attention deficits and reading disabilities: Are immediate memory defects additive? *Developmental Neuropsychology*, 15(2), 213–226. <https://doi.org/10.1080/87565649909540746>. ***
- Johnson, M., Johnels, J. Å., Östlund, S., Cedergren, K., Omanovic, Z., Hjalmarsson, K., Jakobsson, K., Högstedt, J., & Billstedt, E. (2021). Long-term medication for ADHD and development of cognitive functions in children and adolescents. *Journal of Psychiatric Research*, 142, 204–209. <https://doi.org/10.1016/j.jpsychires.2021.07.055>. ***
- Jolliffe, I. T. (2002). *Principal component analysis for special types of data*. 338–372. Springer.
- Jolliffe, I. T., & Cadima, J. (2016). Principal component analysis: A review and recent developments. *Philosophical Transactions. Series A, Mathematical, Physical, and Engineering Sciences*, 374(2065), Article 20150202. <https://doi.org/10.1098/rsta.2015.0202>
- Jones, D. R., Dallman, A., Harrop, C., Whitten, A., Pritchett, J., Lecavalier, L., Bodfish, J. W., & Boyd, B. A. (2022). Evaluating the feasibility of the NIH toolbox cognition battery for autistic children and adolescents. *Journal of Autism and Developmental Disorders*, 52(2), 689–699. <https://doi.org/10.1007/s10803-021-04965-2>
- Jones, P. J., Ma, R., & McNally, R. J. (2021). Bridge centrality: A network approach to understanding comorbidity. *Multivariate Behavioral Research*, 56(2), 353–367. <https://doi.org/10.1080/00273171.2019.1614898>
- Jones, P. J., Mair, P., & McNally, R. J. (2018). Visualizing psychological networks: A tutorial in R. *Frontiers in Psychology*, 9, 1742. <https://doi.org/10.3389/fpsyg.2018.01742>
- Jones, P., & Jones, M. P. (2018). *Package 'networktools'*.
- Kadesjö, B., & Gillberg, C. (2001). The comorbidity of ADHD in the general population of Swedish school-age children. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 42(4), 487–492. <https://doi.org/10.1111/1469-7610.00742>
- Kadesjö, B., & Gillberg, C. (2001). The comorbidity of ADHD in the general population of Swedish school-age children. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 42(4), 487–492. <https://doi.org/10.1111/1469-7610.00742>. ***
- Kallitsoglou, A. (2018). Executive function variation in children with conduct problems: Influences of coexisting reading difficulties. *Journal of Learning Disabilities*, 51(2), 168–180. <https://doi.org/10.1177/0022219417691834>

- Kanne, S. M., & Mazurek, M. O. (2011). Aggression in children and adolescents with ASD: Prevalence and risk factors. *Journal of Autism and Developmental Disorders*, 41(7), 926–937. <https://doi.org/10.1007/s10803-010-1118-4>
- Kaplan, B. J., Crawford, S. G., Dewey, D. M., & Fisher, G. C. (2000). The IQs of children with ADHD are normally distributed. *Journal of Learning Disabilities*, 33(5), 425–432. <https://doi.org/10.1177/002221940003300503>. ***
- Kaplan, B. J., Dewey, D. M., Crawford, S. G., & Wilson, B. N. (2001). The term comorbidity is of questionable value in reference to developmental disorders: Data and theory. *Journal of Learning Disabilities*, 34(6), 555–565. <https://doi.org/10.1177/002221940103400608>. ***
- Karbach, J., Strobach, T., & Schubert, T. (2015). Adaptive working-memory training benefits reading, but not mathematics in middle childhood. *Child Neuropsychology: A Journal on Normal and Abnormal Development in Childhood and Adolescence*, 21(3), 285–301. <https://doi.org/10.1080/09297049.2014.899336>
- Kashdan, T. B., Barrios, V., Forsyth, J. P., & Steger, M. F. (2006). Experiential avoidance as a generalized psychological vulnerability: Comparisons with coping and emotion regulation strategies. *Behaviour Research and Therapy*, 44(9), 1301–1320. <https://doi.org/10.1016/j.brat.2005.10.003>
- Katz, L. J., Brown, F. C., Roth, R. M., & Beers, S. R. (2011). Processing speed and working memory performance in those with both ADHD and a reading disorder compared with those with ADHD alone. *Archives of Clinical Neuropsychology*, 26(5), 425–433. <https://doi.org/10.1093/arclin/acr026>. ***
- Kaufman, L., & Rousseeuw, P. J. (2009). *Finding groups in data: An introduction to cluster analysis*. John Wiley & Sons.
- Kibby, M. Y., & Cohen, M. J. (2008). Memory functioning in children with reading disabilities and/or attention deficit/hyperactivity disorder: A clinical investigation of their working memory and long-term memory functioning. *Child Neuropsychology: A Journal on Normal and Abnormal Development in Childhood and Adolescence*, 14(6), 525–546. <https://doi.org/10.1080/09297040701821752>. ***
- Kibby, M. Y., Newsham, G., Imre, Z., & Schlak, J. E. (2021). Is executive dysfunction a potential contributor to the comorbidity between basic reading disability and attention-deficit/hyperactivity disorder? *Child Neuropsychology: A Journal on Normal and Abnormal Development in Childhood and Adolescence*, 27(7), 888–910. <https://doi.org/10.1080/09297049.2021.1908532>. ***
- Kibby, M. Y., Vadnais, S. A., & Jagger-Rickels, A. C. (2019). Which components of processing speed are affected in ADHD subtypes? *Child Neuropsychology: A Journal on Normal and Abnormal Development in Childhood and Adolescence*, 25(7), 964–979. <https://doi.org/10.1080/09297049.2018.1556625>

- King, J. S. (2020). Sad mood, emotion regulation, and response inhibition. *Journal of Cognitive Psychology*, 32(5–6), 573–579. <https://doi.org/10.1080/20445911.2020.1777418>
- Kita, Y., Ashizawa, F., & Inagaki, M. (2020). Prevalence estimates of neurodevelopmental disorders in Japan: A community sample questionnaire study. *Psychiatry and Clinical Neurosciences*, 74(2), 118–123. <https://doi.org/10.1111/pcn.12950>. ***
- Klorman, R., Hazel-Fernandez, L. A., Shaywitz, S. E., Fletcher, J. M., Marchione, K. E., Holahan, J. M., Stuebing, K. K., & Shaywitz, B. A. (1999). Executive functioning deficits in attention-deficit/hyperactivity disorder are independent of oppositional defiant or reading disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 38(9), 1148–1155. <https://doi.org/10.1097/00004583-199909000-00020>. ***
- Koerner, K., J., Visser, L., Rothe, J., Schulte-Koerne, G., & Hasselhorn, M. (2021). Gender differences in the comorbidity of ADHD symptoms and specific learning disorders in a population-based sample. *Sustainability*, 13(15), 8440. <https://doi.org/10.3390/su13158440>. ***
- Kofler, M. J., Rapport, M. D., Bolden, J., Sarver, D. E., & Raiker, J. S. (2010). ADHD and working memory: The impact of central executive deficits and exceeding storage/rehearsal capacity on observed inattentive behavior. *Journal of Abnormal Child Psychology*, 38(2), 149–161. <https://doi.org/10.1007/s10802-009-9357-6>
- Koster, E. H. W., Hoorelbeke, K., Onraedt, T., Owens, M., & Derakshan, N. (2017). Cognitive control interventions for depression: A systematic review of findings from training studies. *Clinical Psychology Review*, 53, 79–92. <https://doi.org/10.1016/j.cpr.2017.02.002>
- Kotov, R. (2016). The quantitative classification of mental illness: Emerging solution to boundary problems. In E. J. Bromet (Ed.), *Long-term outcomes in psychopathology research: Rethinking the scientific agenda* (pp. 140–157). Oxford University Press.
- Kotov, R., Krueger, R. F., Watson, D., Achenbach, T. M., Althoff, R. R., Bagby, R. M., Brown, T. A., Carpenter, W. T., Caspi, A., Clark, L. A., Eaton, N. R., Forbes, M. K., Forbush, K. T., Goldberg, D., Hasin, D., Hyman, S. E., Ivanova, M. Y., Lynam, D. R., Markon, K., . . . & Zimmerman, M. (2017). The Hierarchical Taxonomy of Psychopathology (HiTOP): A dimensional alternative to traditional nosologies. *Journal of Abnormal Psychology*, 126(4), 454–477. <https://doi.org/10.1037/abn0000258>
- Kouvava, S., Antonopoulou, K., Kokkinos, C. M., Ralli, A. M., & Maridaki-Kassotaki, K. (2022). Friendship quality, emotion understanding, and emotion regulation of children with and without attention deficit/hyperactivity disorder or specific learning disorder. *Emotional and Behavioural Difficulties*, 27(1), 3–19. <https://doi.org/10.1080/13632752.2021.2001923>
- Koyuncu, A., Ayan, T., Ince Guliyev, E., Erbilgin, S., & Deveci, E. (2022). ADHD and anxiety disorder comorbidity in children and adults: Diagnostic and therapeutic

challenges. *Current Psychiatry Reports*, 24(2), 129–140. <https://doi.org/10.1007/s11920-022-01324-5>

- Kozak, M. J., & Cuthbert, B. N. (2016). The NIMH research domain criteria initiative: Background, issues, and pragmatics. *Psychophysiology*, 53(3), 286–297. <https://doi.org/10.1111/psyp.12518>
- Kuja-Halkola, R., Lichtenstein, P., D’Onofrio, B. M., & Larsson, H. (2015). Codevelopment of ADHD and externalizing behavior from childhood to adulthood. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 56(6), 640–647. <https://doi.org/10.1111/jcpp.12340>
- Laasonen, M., Kauppinen, J., Leppämäki, S., Tani, P., Harno, H., Hokkanen, L., & Wikgren, J. (2012). Project DyAdd: Classical eyeblink conditioning in adults with dyslexia and ADHD. *Experimental Brain Research*, 223(1), 19–32. <https://doi.org/10.1007/s00221-012-3237-y>. ***
- Laasonen, M., Leppämäki, S., Tani, P., & Hokkanen, L. (2009). Adult dyslexia and attention deficit disorder in Finland—Project DyAdd: WAIS-III cognitive profiles. *Journal of Learning Disabilities*, 42(6), 511–527. <https://doi.org/10.1177/0022219409345013>. ***
- Lage, C., Smith, E. S., & Lawson, R. P. (2024). A meta-analysis of cognitive flexibility in autism spectrum disorder. *Neuroscience and Biobehavioral Reviews*, 157, Article 105511. <https://doi.org/10.1016/j.neubiorev.2023.105511>
- Lamminmäki, T., Ahonen, T., Närhi, V., Lyytinen, H., & de Barra, H. T. (1995). Attention deficit hyperactivity disorder subtypes: Are there differences in academic problems? *Developmental Neuropsychology*, 11(3), 297–310. <https://doi.org/10.1080/87565649509540621>. ***
- Langberg, J. M., Vaughn, A. J., Brinkman, W. B., Froehlich, T., & Epstein, J. N. (2010). Clinical utility of the Vanderbilt A D H D Rating Scale for ruling out comorbid learning disorders. *Pediatrics*, 126(5), Article e1033–e1038. <https://doi.org/10.1542/peds.2010-1267-e1038>. ***
- Langer, N., Benjamin, C., Becker, B. L. C., & Gaab, N. (2019). Comorbidity of reading disabilities and ADHD: Structural and functional brain characteristics. *Human Brain Mapping*, 40(9), 2677–2698. <https://doi.org/10.1002/hbm.24552>. ***
- Langley, K., Del Pozo-Banos, M., Daalsgard, S., Paranjothy, S., Riglin, L., John, A., & Thapar, A. (2023). Can a nation-wide e-cohort of ADHD and ASD in childhood be established using Welsh routinely available datasets? *BMJ Open*, 13(8), Article e071851. <https://doi.org/10.1136/bmjopen-2023-071851>
- Lee, C. S. C. (2024). Executive functions underlie word reading and reading fluency in Chinese children with attention deficit/hyperactivity disorder, reading disabilities, and comorbid attention deficit/hyperactivity disorder and reading disabilities. *Child*

Neuropsychology: A Journal on Normal and Abnormal Development in Childhood and Adolescence, 30(1), 60–86. <https://doi.org/10.1080/09297049.2023.2179981>. ***

- Lefèvre, E., Law, J. M., Prado, J., Anders, R., & Cavalli, E. (2025). Reading comprehension resiliency in adolescents with and without dyslexia relates to vocabulary, listening comprehension and socioeconomic status. *Learning and Instruction*, 96, Article 102081. <https://doi.org/10.1016/j.learninstruc.2025.102081>
- Lei, J., Charman, T., Leigh, E., Russell, A., Mohamed, Z., & Hollocks, M. J. (2022). Examining the relationship between cognitive inflexibility and internalizing and externalizing symptoms in autistic children and adolescents: A systematic review and meta-analysis. *Autism Research: Official Journal of the International Society for Autism Research*, 15(12), 2265–2295. <https://doi.org/10.1002/aur.2826>
- Leib, S. I., Keezer, R. D., Cerny, B. M., Holbrook, L. R., Gallagher, V. T., Jennette, K. J., Ovsiew, G. P., & Soble, J. R. (2021). Distinct latent profiles of working memory and processing speed in adults with ADHD. *Developmental Neuropsychology*, 46(8), 574–587. <https://doi.org/10.1080/87565641.2021.1999454>
- Lensen, S. (2023). When to pool data in a meta-analysis (and when not to)? *Fertility and Sterility*, 119(6), 902–903. <https://doi.org/10.1016/j.fertnstert.2023.03.015>
- Lewandowska, M., Milner, R., Ganc, M., Włodarczyk, E., & Skarżyński, H. (2014). Attention dysfunction subtypes of developmental dyslexia. *Medical Science Monitor: International Medical Journal of Experimental and Clinical Research*, 20, 2256–2268. <https://doi.org/10.12659/MSM.890969>
- Li, M., Georgiou, G., Kirby, J. R., Frijters, J. C., Zhao, W., & Wang, T. (2023). Reading fluency in Chinese children with reading disabilities and/or ADHD: A key role for morphology. *Journal of Learning Disabilities*, 56(6), 467–482. <https://doi.org/10.1177/00222194221131569>. ***
- Li, M., Truong, D. T., DeMille, M., Malins, J. G., Lovett, M. W., Bosson-Heenan, J., Gruen, J. R., & Frijters, J. C. (2020). Effect of READ1 on latent profiles of reading disorder and comorbid attention and language impairment subtypes. *Child Neuropsychology: A Journal on Normal and Abnormal Development in Childhood and Adolescence*, 26(2), 145–169. <https://doi.org/10.1080/09297049.2019.1648642>. ***
- Lipinski, S., Boegl, K., Blanke, E. S., Suenkel, U., & Dziobek, I. (2022). A blind spot in mental healthcare? Psychotherapists lack education and expertise for the support of adults on the autism spectrum. *Autism: The International Journal of Research and Practice*, 26(6), 1509–1521. <https://doi.org/10.1177/13623613211057973>
- Little, M., & McLennan, J. D. (2010). Teacher perceived mental and learning problems of children referred to a school mental health service. *Journal of the Canadian Academy of Child and Adolescent Psychiatry*, 19(2), 94–99. ***.

- Liu, J. (2004). Childhood externalizing behavior: Theory and implications. *Journal of Child and Adolescent Psychiatric Nursing*, 17(3), 93–103. <https://doi.org/10.1111/j.1744-6171.2004.tb00003.x>
- Liu, L., Wang, Y., Chen, W., Gao, Y., Li, H., Wang, Y., Chan, R. C. K., & Qian, Q. (2022). Network analysis of 18 attention-deficit/hyperactivity disorder symptoms suggests the importance of “Distracted” and “Fidget” as central symptoms: Invariance across age, gender, and subtype presentations. *Frontiers in Psychiatry*, 13, Article 974283. <https://doi.org/10.3389/fpsyt.2022.974283>
- Livingston, L. A., Shah, P., Milner, V., & Happé, F. (2020). Quantifying compensatory strategies in adults with and without diagnosed autism. *Molecular Autism*, 11(1), 15. <https://doi.org/10.1186/s13229-019-0308-y>
- Livingston, R. L., Dykman, R. A., & Ackerman, P. T. (1990). The frequency and significance of additional self-reported psychiatric diagnoses in children with attention deficit disorder. *Journal of Abnormal Child Psychology*, 18(5), 465–478. <https://doi.org/10.1007/BF00911102>. ***
- Llewellyn, A., & Hogan, K. (2000). The use and abuse of models of disability. *Disability and Society*, 15(1), 157–165. <https://doi.org/10.1080/09687590025829>
- Lonergan, A., Doyle, C., Cassidy, C., MacSweeney Mahon, S., Roche, R. A. P., Boran, L., & Bramham, J. (2019). A meta-analysis of executive functioning in dyslexia with consideration of the impact of comorbid ADHD. *Journal of Cognitive Psychology*, 31(7), 725–749. <https://doi.org/10.1080/20445911.2019.1669609>
- Loo, S. K., Fisher, S. E., Francks, C., Ogdie, M. N., MacPhie, I. L., Yang, M., McCracken, J. T., McGough, J. J., Nelson, S. F., Monaco, A. P., & Smalley, S. L. (2004). Genome-wide scan of reading ability in affected sibling pairs with attention-deficit/hyperactivity disorder: Unique and shared genetic effects. *Molecular Psychiatry*, 9(5), 485–493. <https://doi.org/10.1038/sj.mp.4001450>. ***
- Luoni, C., Balottin, U., Zaccagnino, M., Brembilla, L., Livetti, G., & Termine, C. (2015). Reading difficulties and attention-deficit/hyperactivity behaviours: Evidence of an early association in a nonclinical sample. *Journal of Research in Reading*, 38(1), 73–90. <https://doi.org/10.1111/j.1467-9817.2013.01555.x>. ***
- Luyster, R. J., Seery, A., Talbott, M. R., & Tager-Flusberg, H. (2011). Identifying early-risk markers and developmental trajectories for language impairment in neurodevelopmental disorders. *Developmental Disabilities Research Reviews*, 17(2), 151–159. <https://doi.org/10.1002/ddrr.1109>
- Lyon, G. R., Shaywitz, S. E., & Shaywitz, B. A. (2003). A definition of dyslexia. *Annals of Dyslexia*, 53(1), 1–14. <https://doi.org/10.1007/s11881-003-0001-9>
- Maassen, B. A. M., Krikhaar, E., van der Leij, A., & Fikkert, P. (2022). Early productive vocabulary composition as precursor of dyslexia. *Journal of Speech, Language, and*

Hearing Research: JSLHR, 65(2), 760–774. https://doi.org/10.1044/2021_JSLHR-20-00599

- MacCullagh, L., Bosanquet, A., & Badcock, N. A. (2017). University students with dyslexia: A qualitative exploratory study of learning practices, challenges and strategies. *Dyslexia*, 23(1), 3–23. <https://doi.org/10.1002/dys.1544>
- Maehler, C., & Schuchardt, K. (2016). Working memory in children with specific learning disorders and/or attention deficits. *Learning and Individual Differences*, 49, 341–347. <https://doi.org/10.1016/j.lindif.2016.05.007>. ***
- Maestrini, L., Hui, F. K., & Welsh, A. H. (2024). *Restricted maximum likelihood estimation in generalized linear mixed models*. *arXiv* 2402.12719. <https://doi.org/10.48550/arXiv.2402.12719>
- Maister, L., Simons, J. S., & Plaisted-Grant, K. (2013). Executive functions are employed to process episodic and relational memories in children with autism spectrum disorders. *Neuropsychology*, 27(6), 615–627. <https://doi.org/10.1037/a0034492>
- Mammarella, I. C., Toffalini, E., Caviola, S., Colling, L., & Szűcs, D. (2021). No evidence for a core deficit in developmental dyscalculia or mathematical learning disabilities. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 62(6), 704–714. <https://doi.org/10.1111/jcpp.13397>
- Mano, Q. R., Jastrowski Mano, K. E., Denton, C. A., Epstein, J. N., & Tamm, L. (2017). Gender moderates association between emotional-behavioral problems and text comprehension in children with both reading difficulties and ADHD. *Psychology in the Schools*, 54(5), 504–518. <https://doi.org/10.1002/pits.22011>. ***
- Mareva, S., Akarca, D., CALM Team, & Holmes, J. (2023). Transdiagnostic profiles of behaviour and communication relate to academic and socioemotional functioning and neural white matter organisation. *Journal of child psychology and psychiatry, and allied disciplines*, 64(2), 217–233. <https://doi.org/10.1111/jcpp.13685>
- Mareva, S., CALM team, & Holmes, J. (2019). Transdiagnostic associations across communication, cognitive, and behavioural problems in a developmentally at-risk population: A network approach. *BMC Pediatrics*, 19(1), 452. <https://doi.org/10.1186/s12887-019-1818-7>
- Mareva, S., CALM Team, & Holmes, J. (2024). Mapping neurodevelopmental diversity in executive function. *Cortex; a Journal Devoted to the Study of the Nervous System and Behavior*, 172, 204–221. <https://doi.org/10.1016/j.cortex.2023.11.021>
- Margari, L., Buttiglione, M., Craig, F., Cristella, A., de Giambattista, C., Matera, E., Operto, F., & Simone, M. (2013). Neuropsychopathological comorbidities in learning disorders. *BMC Neurology*, 13, 198. <https://doi.org/10.1186/1471-2377-13-198>

- Marín-Martínez, F., & Sánchez-Meca, J. (2010). Weighting by inverse variance or by sample size in random-effects meta-analysis. *Educational and Psychological Measurement*, 70(1), 56–73. <https://doi.org/10.1177/0013164409344534>
- Markon, K. E., Chmielewski, M., & Miller, C. J. (2011). The reliability and validity of discrete and continuous measures of psychopathology: A quantitative review. *Psychological Bulletin*, 137(5), 856–879. <https://doi.org/10.1037/a0023678>
- Marshall, R. M., Hynd, G. W., Handwerk, M. J., & Hall, J. (1997). Academic underachievement in ADHD subtypes. *Journal of Learning Disabilities*, 30(6), 635–642. <https://doi.org/10.1177/002221949703000607>. ***
- Martel, M. M., Levinson, C. A., Lee, C. A., & Smith, T. E. (2017). Impulsivity symptoms as core to the developmental externalizing spectrum. *Journal of Abnormal Child Psychology*, 45(1), 83–90. <https://doi.org/10.1007/s10802-016-0148-6>
- Martella, D., Aldunate, N., Fuentes, L. J., & Sánchez-Pérez, N. (2020). Arousal and executive alterations in attention deficit hyperactivity disorder (ADHD). *Frontiers in Psychology*, 11, 1991. <https://doi.org/10.3389/fpsyg.2020.01991>
- Martinussen, R., & Mackenzie, G. (2015). Reading comprehension in adolescents with ADHD: Exploring the poor comprehender profile and individual differences in vocabulary and executive functions. *Research in Developmental Disabilities*, 38, 329–337. <https://doi.org/10.1016/j.ridd.2014.12.007> ***
- Martinussen, R., Hayden, J., Hogg-Johnson, S., & Tannock, R. (2005). A meta-analysis of working memory impairments in children with attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 44(4), 377–384. <https://doi.org/10.1097/01.chi.0000153228.72591.73>
- Marulis, L. M., & Neuman, S. B. (2010). The effects of vocabulary intervention on young children's word learning: A meta-analysis. *Review of Educational Research*, 80(3), 300–335. <https://doi.org/10.3102/0034654310377087>
- Marx, I., Hübner, T., Herpertz, S. C., Berger, C., Reuter, E., Kircher, T., Herpertz-Dahlmann, B., & Konrad, K. (2010). Cross-sectional evaluation of cognitive functioning in children, adolescents and young adults with ADHD. *Journal of Neural Transmission*, 117(3), 403–419. <https://doi.org/10.1007/s00702-009-0345-3>
- Masten, A. S., & Cicchetti, D. (2010). Developmental cascades. *Development and Psychopathology*, 22(3), 491–495. <https://doi.org/10.1017/S0954579410000222>
- Mayes, S. D., & Calhoun, S. L. (2006). Frequency of reading, math, and writing disabilities in children with clinical disorders. *Learning and Individual Differences*, 16(2), 145–157. <https://doi.org/10.1016/j.lindif.2005.07.004>. ***

- Mayes, S. D., Calhoun, S. L., & Crowell, E. W. (2000). Learning disabilities and ADHD: Overlapping spectrum disorders. *Journal of Learning Disabilities*, 33(5), 417–424. <https://doi.org/10.1177/002221940003300502>
- Mayes, S. D., Waschbusch, D. A., Calhoun, S. L., & Mattison, R. E. (2020). How common are academic overachievement and underachievement in children with autism or ADHD? *Journal of Developmental and Physical Disabilities*, 32(5), 775–783. <https://doi.org/10.1007/s10882-019-09719-8>. ***
- Mazefsky, C. A. (2015). Emotion regulation and emotional distress in autism spectrum disorder: Foundations and considerations for future research. *Journal of Autism and Developmental Disorders*, 45(11), 3405–3408. <https://doi.org/10.1007/s10803-015-2602-7>
- Mazefsky, C. A., Herrington, J., Siegel, M., Scarpa, A., Maddox, B. B., Scahill, L., & White, S. W. (2013). The role of emotion regulation in autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52(7), 679–688. <https://doi.org/10.1016/j.jaac.2013.05.006>
- McAuley, T., & White, D. A. (2011). A latent variables examination of processing speed, response inhibition, and working memory during typical development. *Journal of Experimental Child Psychology*, 108(3), 453–468. <https://doi.org/10.1016/j.jecp.2010.08.009>
- McAuley, T., Chen, S., Goos, L., Schachar, R., & Crosbie, J. (2010). Is the Behavior Rating Inventory of Executive Function more strongly associated with measures of impairment or executive function? *Journal of the International Neuropsychological Society: JINS*, 16(3), 495–505. <https://doi.org/10.1017/S1355617710000093>
- McGee, R. A., Clark, S. E., & Symons, D. K. (2000). Does the Conners' continuous performance test aid in ADHD diagnosis? *Journal of Abnormal Child Psychology*, 28(5), 415–424. <https://doi.org/10.1023/a:1005127504982>. ***
- McGrath, L. M., Pennington, B. F., Shanahan, M. A., Santerre-Lemmon, L. E., Barnard, H. D., Willcutt, E. G., Defries, J. C., & Olson, R. K. (2011). A multiple deficit model of reading disability and attention-deficit/hyperactivity disorder: Searching for shared cognitive deficits. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 52(5), 547–557. <https://doi.org/10.1111/j.1469-7610.2010.02346.x>
- McGuinness, L. A., & Higgins, J. P. T. (2020). Risk-Of-Bias VISualization (robvis): an R package and Shiny web app for visualizing risk-of-bias assessments. *Research Synthesis Methods*, 2020, 1-7. <https://doi.org/10.1002/jrsm.1411>
- McHugh, M. L. (2012). Interrater reliability: The kappa statistic. *Biochemia Medica*, 22(3), 276–282. <https://doi.org/10.11613/BM.2012.031>
- McInnes, A., Humphries, T., Hogg-Johnson, S., & Tannock, R. (2003). Listening comprehension and working memory are impaired in attention-deficit hyperactivity

- disorder irrespective of language impairment. *Journal of Abnormal Child Psychology*, 31(4), 427–443. <https://doi.org/10.1023/a:1023895602957>
- McKay, E., Kirk, H., Martin, R., & Cornish, K. (2023). Social difficulties in adolescent attention deficit hyperactivity disorder: Social motivation, social anxiety and symptom severity as contributing factors. *Journal of Clinical Psychology*, 79(4), 1113–1129. <https://doi.org/10.1002/jclp.23462>
- McKechnie, D. G. J., O’Nions, E., Dunsmuir, S., & Petersen, I. (2023). Attention-deficit hyperactivity disorder diagnoses and prescriptions in UK primary care, 2000–2018: Population-based cohort study. *BJPsych Open*, 9(4), e121. <https://doi.org/10.1192/bjo.2023.512>
- Melby-Lervåg, M., Lyster, S.-A. H., & Hulme, C. (2012). Phonological skills and their role in learning to read: A meta-analytic review. *Psychological Bulletin*, 138(2), 322–352. <https://doi.org/10.1037/a0026744>
- Méndez, M. A., Hödar, C., Vulpe, C., González, M., & Cambiazo, V. (2002). Discriminant analysis to evaluate clustering of gene expression data. *FEBS Letters*, 522(1–3), 24–28. [https://doi.org/10.1016/s0014-5793\(02\)02873-9](https://doi.org/10.1016/s0014-5793(02)02873-9)
- Michellini, G., Carlisi, C. O., Eaton, N. R., Elison, J. T., Haltigan, J. D., Kotov, R., Krueger, R. F., Latzman, R. D., Li, J. J., Levin-Aspenson, H. F., Salum, G. A., South, S. C., Stanton, K., Waldman, I. D., & Wilson, S. (2024). Where do neurodevelopmental conditions fit in transdiagnostic psychiatric frameworks? Incorporating a new neurodevelopmental spectrum. *World Psychiatry*, 23(3), 333–357. <https://doi.org/10.1002/wps.21225>
- Michellini, G., Palumbo, I. M., DeYoung, C. G., Latzman, R. D., & Kotov, R. (2021). Linking RDoC and HiTOP: A new interface for advancing psychiatric nosology and neuroscience. *Clinical Psychology Review*, 86, Article 102025. <https://doi.org/10.1016/j.cpr.2021.102025>
- Miranda, A., Mercader, J., Fernández, M. I., & Colomer, C. (2017). Reading performance of young adults with ADHD diagnosed in childhood. *Journal of Attention Disorders*, 21(4), 294–304. <https://doi.org/10.1177/1087054713507977>. ***
- Miyake, A., & Friedman, N. P. (2012). The nature and organization of individual differences in executive functions: Four general conclusions. *Current Directions in Psychological Science*, 21(1), 8–14. <https://doi.org/10.1177/0963721411429458>
- Miyake, A., & Shah, P. (1999). *Models of working memory* (pp. 442–481). Cambridge University Press.
- Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex “frontal lobe” tasks: A latent variable analysis. *Cognitive Psychology*, 41(1), 49–100. <https://doi.org/10.1006/cogp.1999.0734>

- Mohammadi, M. R., Ahmadi, N., Khaleghi, A., Zarafshan, H., Mostafavi, S.-A., Kamali, K., Rahgozar, M., Ahmadi, A., Hooshyari, Z., Alavi, S. S., Shakiba, A., Salmanian, M., Molavi, P., Sarraf, N., Hojjat, S. K., Mohammadzadeh, S., Amiri, S., Arman, S., & Ghanizadeh, A. (2019). Prevalence of autism and its comorbidities and the relationship with maternal psychopathology: A national population-based study. *Archives of Iranian Medicine*, 22(10), 546–553.
- Mohl, B., Ofen, N., Jones, L. L., Casey, J. E., & Stanley, J. A. (2018). Novel reading index for identifying disordered reading skill development: A preliminary study. *Applied Neuropsychology. Child*, 7(4), 287–297.
<https://doi.org/10.1080/21622965.2017.1328681>. ***
- Molavi, P., Nadermohammadi, M., Salvat Ghojehbeiglou, H., Vicario, C. M., Nitsche, M. A., & Salehinejad, M. A. (2020). ADHD subtype-specific cognitive correlates and association with self-esteem: A quantitative difference. *BMC Psychiatry*, 20(1), 502.
<https://doi.org/10.1186/s12888-020-02887-4>
- Molitor, S. J., Langberg, J. M., & Evans, S. W. (2016). The written expression abilities of adolescents with attention-deficit/hyperactivity disorder. *Research in Developmental Disabilities*, 51–52, 49–59. <https://doi.org/10.1016/j.ridd.2016.01.005>. ***
- Montaser, J., Umeano, L., Pujari, H. P., Nasiri, S. M. Z., Parisapogu, A., Shah, A., & Khan, S. (2023). Correlations between the development of social anxiety and individuals with autism spectrum disorder: A systematic review. *Cureus*, 15(9), Article e44841.
<https://doi.org/10.7759/cureus.44841>
- Moola, S., Munn, Z., Tufanaru, C., Aromataris, E., Sears, K., Sfetcu, R., Currie, M., Qureshi, R., Mattis, P., Lisy, K., & Mu, P.-F. (2020). Chapter 7. Systematic reviews of etiology and risk. In E. Aromataris & Z. Munn (Eds.), *JB1 manual for evidence synthesis*, JBI.
- Moretti, L., Semenza, C., & Vallesi, A. (2018). General Slowing and education mediate task switching performance across the life-span. *Frontiers in Psychology*, 9, 630.
<https://doi.org/10.3389/fpsyg.2018.00630>
- Morris, D., & Turnbull, P. (2007). A survey-based exploration of the impact of dyslexia on career progression of UK registered nurses. *Journal of Nursing Management*, 15(1), 97–106. <https://doi.org/10.1111/j.1365-2934.2006.00649.x>
- Mostert, J. C., Hoogman, M., Onnink, A. M. H., van Rooij, D., von Rhein, D., van Hulzen, K. J. E., Dammers, J., Kan, C. C., Buitelaar, J. K., Norris, D. G., & Franke, B. (2018). Similar subgroups based on cognitive performance parse heterogeneity in adults with ADHD and healthy controls. *Journal of Attention Disorders*, 22(3), 281–292.
<https://doi.org/10.1177/1087054715602332>
- Mostert, J. C., Onnink, A. M. H., Klein, M., Dammers, J., Harneit, A., Schulten, T., van Hulzen, K. J. E., Kan, C. C., Slaats-Willemse, D., Buitelaar, J. K., Franke, B., & Hoogman, M. (2015). Cognitive heterogeneity in adult attention deficit/hyperactivity disorder: A systematic analysis of neuropsychological measurements. *European*

Neuropsychopharmacology, 25(11), 2062–2074.
<https://doi.org/10.1016/j.euroneuro.2015.08.010>

Moura, O., Pereira, M., Alfaiate, C., Fernandes, E., Fernandes, B., Nogueira, S., Moreno, J., & Simões, M. R. (2017). Neurocognitive functioning in children with developmental dyslexia and attention-deficit/hyperactivity disorder: Multiple deficits and diagnostic accuracy. *Journal of Clinical and Experimental Neuropsychology*, 39(3), 296–312.
<https://doi.org/10.1080/13803395.2016.1225007>. ***

Mungas, D., Heaton, R., Tulsky, D., Zelazo, P. D., Slotkin, J., Blitz, D., Lai, J.-S., & Gershon, R. (2014). Factor structure, convergent validity, and discriminant validity of the NIH Toolbox Cognitive Health Battery (NIHTB-CHB) in adults. *Journal of the International Neuropsychological Society: JINS*, 20(6), 579–587.
<https://doi.org/10.1017/S1355617714000307>

Munn, Z., Moola, S., Lisy, K., Riitano, D., & Tufanaru, C. (2015). Methodological guidance for systematic reviews of observational epidemiological studies reporting prevalence and cumulative incidence data. *International Journal of Evidence-Based Healthcare*, 13(3), 147–153. <https://doi.org/10.1097/XEB.0000000000000054>

Nakashima, N., Yamashita, Y., Hirata, R., Nagamitsu, S., & Matsuishi, T. (2012). Kana reading disability and Das–Naglieri Cognitive Assessment System findings in children with attention deficit hyperactivity disorder. *Pediatrics International*, 54(6), 849–853.
<https://doi.org/10.1111/j.1442-200X.2012.03694.x>. ***

Naples, L. H., & Tuckwiller, E. D. (2021). Taking students on a strengths safari: A multidimensional pilot study of school-based wellbeing for young neurodiverse children. *International Journal of Environmental Research and Public Health*, 18(13), 6947.
<https://doi.org/10.3390/ijerph18136947>

Närhi, V., & Ahonen, T. (1995). Reading disability with or without, attention deficit hyperactivity, disorder: Do attentional problems, make a difference? *Developmental Neuropsychology*, 11(3), 337–349. <https://doi.org/10.1080/87565649509540624>. ***

Naz, S., & Najam, N. (2019). Neurological deficits and comorbidity in children with reading disorder. *Psychiatry and Clinical Psychopharmacology*, 29(4), 674–681.
<https://doi.org/10.1080/24750573.2019.1589174>. ***

Nelson, J. M., & Harwood, H. (2011). Learning disabilities and anxiety: A meta-analysis. *Journal of Learning Disabilities*, 44(1), 3–17.
<https://doi.org/10.1177/0022219409359939>

Nigg, J. T. (1999). The ADHD response-inhibition deficit as measured by the stop task: Replication with DSM–IV combined type, extension, and qualification. *Journal of Abnormal Child Psychology*, 27(5), 393–402. <https://doi.org/10.1023/a:1021980002473>.

- Nigg, J. T., Hinshaw, S. P., Carte, E. T., & Treuting, J. J. (1998). Neuropsychological correlates of childhood attention-deficit/hyperactivity disorder: Explainable by comorbid disruptive behavior or reading problems? *Journal of Abnormal Psychology*, 107(3), 468–480. <https://doi.org/10.1037/0021-843X.107.3.468>. ***
- Nigg, J. T., Sibley, M. H., Thapar, A., & Karalunas, S. L. (2020). Development of ADHD: Etiology, heterogeneity, and early life course. *Annual Review of Developmental Psychology*, 2(1), 559–583. <https://doi.org/10.1146/annurev-devpsych-060320-093413>
- Okoye, C., Obialo-Ibeawuchi, C. M., Obajeun, O. A., Sarwar, S., Tawfik, C., Waleed, M. S., Wasim, A. U., Mohamoud, I., Afolayan, A. Y., & Mbaezue, R. N. (2023). Early diagnosis of autism spectrum disorder: A review and analysis of the risks and benefits. *Cureus*, 15(8), Article e43226. <https://doi.org/10.7759/cureus.43226>
- Olofsson, Å., Taube, K., & Ahl, A. (2015). Academic achievement of university students with dyslexia. *Dyslexia*, 21(4), 338–349. <https://doi.org/10.1002/dys.1517>
- Onandia-Hinchado, I., Pardo-Palenzuela, N., & Diaz-Orueta, U. (2021). Cognitive characterization of adult attention deficit hyperactivity disorder by domains: A systematic review. *Journal of Neural Transmission*, 128(7), 893–937. <https://doi.org/10.1007/s00702-021-02302-6>
- Orm, S., Andersen, P. N., Fossum, I. N., Øie, M. G., & Skogli, E. W. (2022). Brief report: Autism spectrum disorder diagnostic persistence in a 10-year longitudinal study. *Research in Autism Spectrum Disorders*, 97, Article 102007. <https://doi.org/10.1016/j.rasd.2022.102007>
- Özerk, G., Özerk, K., & Silveira-Zaldivara, T. (2021). Developing social skills and social competence in children with autism. *International Electronic Journal of Elementary Education*, 13(3), 341–363. <https://doi.org/10.26822/iejee.2021.195>
- Pacheco, J., Garvey, M. A., Sarampote, C. S., Cohen, E. D., Murphy, E. R., & Friedman-Hill, S. R. (2022). Annual Research Review: The contributions of the RDoC research framework on understanding the neurodevelopmental origins, progression and treatment of mental illnesses. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 63(4), 360–376. <https://doi.org/10.1111/jcpp.13543>
- Page, M. J., McKenzie, J. E., Bossuyt, P. M., Boutron, I., Hoffmann, T. C., Mulrow, C. D., Shamseer, L., Tetzlaff, J. M., Akl, E. A., Brennan, S. E., Chou, R., Glanville, J., Grimshaw, J. M., Hróbjartsson, A., Lalu, M. M., Li, T., Loder, E. W., Mayo-Wilson, E., McDonald, S., . . . Moher, D. (2021). The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. *BMJ*, 372, n71. <https://doi.org/10.1136/bmj.n71>
- Pagirsky, M. S., Koriakin, T. A., Avitia, M., Costa, M., Marchis, L., Maykel, C., Sassu, K., Bray, M. A., & Pan, X. (2017). Do the kinds of achievement errors made by students diagnosed with ADHD vary as a function of their reading ability? *Journal of Psychoeducational Assessment*, 35(1–2), 124–137. <https://doi.org/10.1177/0734282916669020>. ***

- Papaioannou, S., Mouzaki, A., Sideridis, G. D., Antoniou, F., Padeliadu, S., & Simos, P. G. (2016). Cognitive and academic abilities associated with symptoms of attention-deficit/hyperactivity disorder: A comparison between subtypes in a Greek non-clinical sample. *Educational Psychology*, 36(1), 138–158. <https://doi.org/10.1080/01443410.2014.915931>. ***
- Parke, E. M., Thaler, N. S., Etcoff, L. M., & Allen, D. N. (2020). Intellectual profiles in children with ADHD and comorbid learning and motor disorders. *Journal of Attention Disorders*, 24(9), 1227–1236. <https://doi.org/10.1177/1087054715576343>. ***
- Pate, A., Riley, R. D., Collins, G. S., Van Smeden, M., Van Calster, B., Ensor, J., & Martin, G. P. (2023). Minimum sample size for developing a multivariable prediction model using multinomial logistic regression. *Statistical Methods in Medical Research*, 32(3), 555–571. <https://doi.org/10.1177/09622802231151220>
- Patros, C. H. G., Tarle, S. J., Alderson, R. M., Lea, S. E., & Arrington, E. F. (2019). Planning deficits in children with attention-deficit/hyperactivity disorder (ADHD): A meta-analytic review of tower task performance. *Neuropsychology*, 33(3), 425–444. <https://doi.org/10.1037/neu0000531>
- Pauc, R. (2005). Comorbidity of dyslexia, dyspraxia, attention deficit disorder (ADD), attention deficit hyperactive disorder (ADHD), obsessive compulsive disorder (OCD) and Tourette's syndrome in children: A prospective epidemiological study. *Clinical Chiropractic*, 8(4), 189–198. <https://doi.org/10.1016/j.clch.2005.09.007>. ***
- Pellicano, E., & den Houting, J. (2022). Annual Research Review: Shifting from “normal science” to neurodiversity in autism science. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 63(4), 381–396. <https://doi.org/10.1111/jcpp.13534>
- Pellicano, E., Dinsmore, A., & Charman, T. (2014). What should autism research focus upon? Community views and priorities from the United Kingdom. *Autism: The International Journal of Research and Practice*, 18(7), 756–770. <https://doi.org/10.1177/1362361314529627>
- Peng, P., Barnes, M., Wang, C., Wang, W., Li, S., Swanson, H. L., Dardick, W., & Tao, S. (2018). A meta-analysis on the relation between reading and working memory. *Psychological Bulletin*, 144(1), 48–76. <https://doi.org/10.1037/bul0000124>
- Pennington, B. F., & Lefly, D. L. (2001). Early reading development in children at family risk for dyslexia. *Child Development*, 72(3), 816–833. <https://doi.org/10.1111/1467-8624.00317>
- Pennington, B. F., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 37(1), 51–87. <https://doi.org/10.1111/j.1469-7610.1996.tb01380.x>

- Pennington, B. F., Groisser, D., & Welsh, M. C. (1993). Contrasting cognitive deficits in attention deficit hyperactivity disorder versus reading disability. *Developmental Psychology*, 29(3), 511–523. <https://doi.org/10.1037/0012-1649.29.3.511>. ***
- Pereira, E. E. L. D., Minervino, C. A. S. M., Cruz, L. F. P. D., Roama-Alves, R. J., & Andrade, J. M. (2020). Executive Functions in children with ADHD and/or reading difficulty. *Psicologia: Teoria e Pesquisa*, 36, Article e3623. <https://doi.org/10.1590/0102.3772e3623>. ***
- Perkins, E. R., Latzman, R. D., & Patrick, C. J. (2020). Interfacing neural constructs with the Hierarchical Taxonomy of Psychopathology: “Why” and “how”. *Personality and Mental Health*, 14(1), 106–122. <https://doi.org/10.1002/pmh.1460>
- Peterson, R. L., Pennington, B. F., Shriberg, L. D., & Boada, R. (2009). What influences literacy outcome in children with speech sound disorder? *Journal of Speech, Language, and Hearing Research: JSLHR*, 52(5), 1175–1188. [https://doi.org/10.1044/1092-4388\(2009/08-0024\)](https://doi.org/10.1044/1092-4388(2009/08-0024))
- Pisecco, S., Baker, D. B., Silva, P. A., & Brooke, M. (2001). Boys with reading disabilities and/or ADHD: Distinctions in early childhood. *Journal of Learning Disabilities*, 34(2), 98–106. <https://doi.org/10.1177/002221940103400201>. ***
- Pividori, M., Ritchie, M. D., Milone, D. H., & Greene, C. S. (2024). An efficient, not-only-linear correlation coefficient based on clustering. *Cell Systems*, 15(9), 854–868.e3. <https://doi.org/10.1016/j.cels.2024.08.005>
- Pons, P., & Latapy, M. (2006). Computing communities in large networks using random walks. *Journal of Graph Algorithms and Applications*, 10(2), 191–218. <https://doi.org/10.7155/jgaa.00124>
- Poon, K., & Ho, C. S.-H. (2014). Contrasting deficits on executive functions in Chinese delinquent adolescents with attention deficit and hyperactivity disorder symptoms and/or reading disability. *Research in Developmental Disabilities*, 35(11), 3046–3056. <https://doi.org/10.1016/j.ridd.2014.07.046>. ***
- Poon, K., Ho, M. S. H., & Wang, L.-C. (2021). Examining distinctive working memory profiles in Chinese children with predominantly inattentive subtype of attention-deficit/hyperactivity disorder and/or reading difficulties. *Frontiers in Psychology*, 12, Article 718112. <https://doi.org/10.3389/fpsyg.2021.718112>. ***
- Preece, D. A., Mehta, A., Petrova, K., Sikka, P., Bjureberg, J., Chen, W., Becerra, R., Allan, A., Robinson, K., & Gross, J. J. (2023). The Perth alexithymia Questionnaire-Short Form (PAQ-S): A 6-item measure of alexithymia. *Journal of Affective Disorders*, 325, 493–501. <https://doi.org/10.1016/j.jad.2023.01.036>
- Pritchard, A. E., Koriakin, T., Carey, L., Bellows, A., Jacobson, L., & Mahone, E. M. (2016). Academic testing accommodations for ADHD: Do they help? *Learning Disabilities*, 21(2), 67–78. <https://doi.org/10.18666/LDMJ-2016-V21-I2-7414>. ***

- Punt, M., De Jong, M., De Groot, E., & Hadders-Algra, M. I. J. N. A. (2010). Minor neurological dysfunction in children with dyslexia. *Developmental Medicine and Child Neurology*, 52(12), 1127–1132. <https://doi.org/10.1111/j.1469-8749.2010.03712.x>. ***
- Purvis, K. L., & Tannock, R. (1997). Language abilities in children with attention deficit hyperactivity disorder, reading disabilities, and normal controls. *Journal of abnormal child psychology*, 25(2), 133–144. <https://doi.org/10.1023/a:1025731529006>
- Purvis, K. L., & Tannock, R. (2000). Phonological processing, not inhibitory control, differentiates ADHD and reading disability. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39(4), 485–494. <https://doi.org/10.1097/00004583-200004000-00018>. ***
- R Core Team R. (2024). *A language and environment for statistical computing*. R Foundation for Statistical Computing.
- Raiker, J. S., Rapport, M. D., Kofler, M. J., & Sarver, D. E. (2012). Objectively-measured impulsivity and attention-deficit/hyperactivity disorder (ADHD): Testing competing predictions from the working memory and behavioral inhibition models of ADHD. *Journal of Abnormal Child Psychology*, 40(5), 699–713. <https://doi.org/10.1007/s10802-011-9607-2>
- Ran, X., Xi, Y., Lu, Y., Wang, X., & Lu, Z. (2023). Comprehensive survey on hierarchical clustering algorithms and the recent developments. *Artificial Intelligence Review*, 56(8), 8219–8264. <https://doi.org/10.1007/s10462-022-10366-3>
- Rapport, M. D., Bolden, J., Kofler, M. J., Sarver, D. E., Raiker, J. S., & Alderson, R. M. (2009). Hyperactivity in boys with attention-deficit/hyperactivity disorder (ADHD): A ubiquitous core symptom or manifestation of working memory deficits? *Journal of Abnormal Child Psychology*, 37(4), 521–534. <https://doi.org/10.1007/s10802-008-9287-8>
- Raskin, J. D., Maynard, D., & Gayle, M. C. (2022). Psychologist attitudes toward DSM-5 and its alternatives. *Professional Psychology: Research and Practice*, 53(6), 553–563. <https://doi.org/10.1037/pro0000480>
- Raudenbush, S. W. (2009). Analyzing effect sizes: Random-effects models. In H. Cooper, L. V. Hedges, & J. C. Valentine (Eds.), *The handbook of research synthesis and meta-analysis* (2nd ed.,). 295–315. Russell Sage Foundation.
- Reis, A., Araújo, S., Morais, I. S., & Faisca, L. (2020). Reading and reading-related skills in adults with dyslexia from different orthographic systems: A review and meta-analysis. *Annals of Dyslexia*, 70(3), 339–368. <https://doi.org/10.1007/s11881-020-00205-x>
- Revelle, W., & Revelle, M. W. (2015). Package “psych”. *The Comprehensive R Archive Network*, 337(338), 161–165.
- Riley, R. D., Higgins, J. P. T., & Deeks, J. J. (2011). Interpretation of random effects meta-analyses. *BMJ*, 342, d549. <https://doi.org/10.1136/bmj.d549>

- Rivard, M., Mestari, Z., Morin, D., Coulombe, P., Mello, C., & Morin, M. (2023). Cluster analysis of clinical features of children suspected to have neurodevelopmental disorders. *Journal of Autism and Developmental Disorders*, 53(6), 2409–2420. <https://doi.org/10.1007/s10803-022-05533-y>
- Robbins, T. W., James, M., Owen, A. M., Sahakian, B. J., McInnes, L., & Rabbitt, P. (1994). Cambridge Neuropsychological Test Automated Battery (CANTAB): A factor analytic study of a large sample of normal elderly volunteers. *Dementia*, 5(5), 266–281. <https://doi.org/10.1159/000106735>
- Robinson, S., Howlin, P., & Russell, A. (2017). Personality traits, autobiographical memory and knowledge of self and others: A comparative study in young people with autism spectrum disorder. *Autism*, 21(3), 357–367. <https://doi.org/10.1177/1362361316645429>
- Rodgers, J., & Ofield, A. (2018). Understanding, recognising and treating co-occurring anxiety in autism. *Current Developmental Disorders Reports*, 5(1), 58–64. <https://doi.org/10.1007/s40474-018-0132-7>
- Rónai, L., Hann, F., Kéri, S., Ettinger, U., & Polner, B. (2024). Emotions under control? Better cognitive control is associated with reduced negative emotionality but increased negative emotional reactivity within individuals. *Behaviour Research and Therapy*, 173, Article 104462. <https://doi.org/10.1016/j.brat.2023.104462>
- Rong, Y., Yang, C.-J., Jin, Y., & Wang, Y. (2021). Prevalence of attention-deficit/hyperactivity disorder in individuals with autism spectrum disorder: A meta-analysis. *Research in Autism Spectrum Disorders*, 83, Article 101759. <https://doi.org/10.1016/j.rasd.2021.101759>
- Rose, S. A., Feldman, J. F., & Jankowski, J. J. (2011). Modeling a cascade of effects: The role of speed and executive functioning in preterm/full-term differences in academic achievement. *Developmental Science*, 14(5), 1161–1175. <https://doi.org/10.1111/j.1467-7687.2011.01068.x>
- Roshani, F., Piri, R., Malek, A., Michel, T. M., & Vafaei, M. S. (2020). Comparison of cognitive flexibility, appropriate risk-taking and reaction time in individuals with and without adult ADHD. *Psychiatry Research*, 284, Article 112494. <https://doi.org/10.1016/j.psychres.2019.112494>
- Rouquette, A., & Falissard, B. (2011). Sample size requirements for the internal validation of psychiatric scales. *International Journal of Methods in Psychiatric Research*, 20(4), 235–249. <https://doi.org/10.1002/mpr.352>
- Rucklidge, J. J., & Tannock, R. (2002). Neuropsychological profiles of adolescents with ADHD: Effects of reading difficulties and gender. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 43(8), 988–1003. <https://doi.org/10.1111/1469-7610.00227>. ***

- Ruggero, C. J., Kotov, R., Hopwood, C. J., First, M., Clark, L. A., Skodol, A. E., Mullins-Sweatt, S. N., Patrick, C. J., Bach, B., Cicero, D. C., Docherty, A., Simms, L. J., Bagby, R. M., Krueger, R. F., Callahan, J. L., Chmielewski, M., Conway, C. C., De Clercq, B., Dornbach-Bender, A., . . . Zimmermann, J. (2019). Integrating the Hierarchical Taxonomy of Psychopathology (HiTOP) into clinical practice. *Journal of Consulting and Clinical Psychology*, 87(12), 1069–1084. <https://doi.org/10.1037/ccp0000452>
- Russell, G., Ryder, D., Norwich, B., & Ford, T. (2015). Behavioural difficulties that co-occur with specific word reading difficulties: A UK population-based cohort study. *Dyslexia*, 21(2), 123–141. <https://doi.org/10.1002/dys.1496>. ***
- Sadozai, A. K., Sun, C., Demetriou, E. A., Lampit, A., Munro, M., Perry, N., Boulton, K. A., & Guastella, A. J. (2024). Executive function in children with neurodevelopmental conditions: A systematic review and meta-analysis. *Nature Human Behaviour*, 8(12), 2357–2366. <https://doi.org/10.1038/s41562-024-02000-9>
- Sadusky, A., Reupert, A. E., Freeman, N. C., & Berger, E. P. (2021). Diagnosing adults with dyslexia: Psychologists' experiences and practices. *Dyslexia*, 27(4), 468–485. <https://doi.org/10.1002/dys.1689>
- Saha, S., Chant, D., Welham, J., & McGrath, J. (2005). A systematic review of the prevalence of schizophrenia. *PLOS Medicine*, 2(5), e141. <https://doi.org/10.1371/journal.pmed.0020141>
- Samuelsson, S., Lundberg, I., & Herkner, B. (2004). ADHD and reading disability in male adults: Is there a connection? *Journal of Learning Disabilities*, 37(2), 155–168. <https://doi.org/10.1177/00222194040370020601>. ***
- Sánchez-Morán, M., Hernández, J. A., Duñabeitia, J. A., Estévez, A., Bárcena, L., González-Lahera, A., Bajo, M. T., Fuentes, L. J., Aransay, A. M., & Carreiras, M. (2018). Genetic association study of dyslexia and ADHD candidate genes in a Spanish cohort: Implications of comorbid samples. *PLOS One*, 13(10), Article e0206431. <https://doi.org/10.1371/journal.pone.0206431>. ***
- Savitz, J. B., & Jansen, P. (2003). The Stroop Color-Word Interference Test as an indicator of ADHD in poor readers. *The Journal of Genetic Psychology*, 164(3), 319–333. <https://doi.org/10.1080/00221320309597986>. ***
- Scarcella, I., Marino, F., Failla, C., Doria, G., Chilà, P., Minutoli, R., Vetrano, N., Vagni, D., Pignolo, L., Di Cara, M., Settimo, C., Quartarone, A., Cerasa, A., & Pioggia, G. (2023). Information and communication technologies-based interventions for children with autism spectrum conditions: A systematic review of randomized control trials from a positive technology perspective. *Frontiers in Psychiatry*, 14, Article 1212522. <https://doi.org/10.3389/fpsy.2023.1212522>
- Schachar, R. J., Chen, S., Logan, G. D., Ornstein, T. J., Crosbie, J., Ickowicz, A., & Pakulak, A. (2004). Evidence for an error monitoring deficit in attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology*, 32(3), 285–293. <https://doi.org/10.1023/b:jacp.0000026142.11217.f2>. ***

- Scheeren, A. M., Buil, J. M., Howlin, P., Bartels, M., & Begeer, S. (2022). Objective and subjective psychosocial outcomes in adults with autism spectrum disorder: A 6-year longitudinal study. *Autism: The International Journal of Research and Practice*, 26(1), 243–255. <https://doi.org/10.1177/13623613211027673>
- Schippers, L. M., Greven, C. U., & Hoogman, M. (2024). Associations between ADHD traits and self-reported strengths in the general population. *Comprehensive Psychiatry*, 130, Article 152461. <https://doi.org/10.1016/j.comppsy.2024.152461>
- Schmeichel, B. J., & Tang, D. (2015). Individual differences in executive functioning and their relationship to emotional processes and responses. *Current Directions in Psychological Science*, 24(2), 93–98. <https://doi.org/10.1177/0963721414555178>
- Schuerholz, L. J., Harris, E. L., Baumgardner, T. L., Reiss, A. L., Freund, L. S., Church, R. P., Mohr, J., & Denckla, M. B. (1995). An analysis of two discrepancy-based models and a processing-deficit approach in identifying learning disabilities. *Journal of learning disabilities*, 28(1), 18–29. <https://doi.org/10.1177/002221949502800104>
- Schwarzer, G., Carpenter, J. R., & Rücker, G. (2023). *metasens: Statistical methods for sensitivity analysis in meta-analysis. Version R package version*, 1–5.
- Seidman, L. J., Biederman, J., Monuteaux, M. C., Doyle, A. E., & Faraone, S. V. (2001). Learning disabilities and executive dysfunction in boys with attention-deficit/hyperactivity disorder. *Neuropsychology*, 15(4), 544–556. <https://doi.org/10.1037//0894-4105.15.4.544>. ***
- Seiffert, S., Weber, S., Sack, U., & Keller, T. (2024). Use of logit transformation within statistical analyses of experimental results obtained as proportions: Example of method validation experiments and EQA in flow cytometry. *Frontiers in Molecular Biosciences*, 11, Article 1335174. <https://doi.org/10.3389/fmolb.2024.1335174>
- Semrud-Clikeman, M., Biederman, J., Sprich-Buckminster, S., Lehman, B. K., Faraone, S. V., & Norman, D. (1992). Comorbidity between ADDH and learning disability: A review and report in a clinically referred sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 31(3), 439–448. <https://doi.org/10.1097/00004583-199205000-00009>. ***
- Sesma, H. W., Mahone, E. M., Levine, T., Eason, S. H., & Cutting, L. E. (2009). The contribution of executive skills to reading comprehension. *Child Neuropsychology: A Journal on Normal and Abnormal Development in Childhood and Adolescence*, 15(3), 232–246. <https://doi.org/10.1080/09297040802220029>. ***
- Sevincok, D., Ozbay, H. C., Ozbek, M. M., Tunagur, M. T., & Aksu, H. (2020). ADHD symptoms in relation to internalizing and externalizing symptoms in children: The mediating role of sluggish cognitive tempo. *Nordic Journal of Psychiatry*, 74(4), 265–272. <https://doi.org/10.1080/08039488.2019.1697746>

- Sexton, C. C., Gelhorn, H. L., Bell, J. A., & Classi, P. M. (2012). The co-occurrence of reading disorder and ADHD: Epidemiology, treatment, psychosocial impact, and economic burden. *Journal of Learning Disabilities*, 45(6), 538–564. <https://doi.org/10.1177/0022219411407772>
- Shanahan, M. A., Pennington, B. F., Yerys, B. E., Scott, A., Boada, R., Willcutt, E. G., Olson, R. K., & DeFries, J. C. (2006). Processing speed deficits in attention deficit/hyperactivity disorder and reading disability. *Journal of Abnormal Child Psychology*, 34(5), 585–602 <https://doi.org/10.1007/s10802-006-9037-8>. ***
- Shaywitz, B. A., Fletcher, J. M., Holahan, J. M., Shneider, A. E., Marchione, K. E., Stuebing, K. K., Francis, D. J., Shankweiler, D. P., Katz, L., Liberman, I. Y., & Shaywitz, S. E. (1995). Interrelationships between reading disability and attention-deficit/hyperactivity disorder. *Child Neuropsychology*, 1(3), 170–186. <https://doi.org/10.1080/09297049508400223>. ***
- Sheikhi, A. R., Martin, N., Hay, D., & Piek, J. P. (2013). Phenotype refinement for comorbid attention deficit hyperactivity disorder and reading disability. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics*, 162B(1), 44–54. <https://doi.org/10.1002/ajmg.b.32119>. ***
- Shi, L., & Lin, L. (2019). The trim-and-fill method for publication bias: Practical guidelines and recommendations based on a large database of meta-analyses. *Medicine*, 98(23), Article e15987. <https://doi.org/10.1097/MD.00000000000015987>
- Shin, M.-S., Kim, Y.-H., Cho, S.-C., & Kim, B.-N. (2003). Neuropsychologic characteristics of children with attention-deficit hyperactivity disorder (ADHD), learning disorder, and tic disorder on the Rey-Osterreith Complex Figure. *Journal of Child Neurology*, 18(12), 835–844. <https://doi.org/10.1177/088307380301801203>. ***
- Skodzik, T., Holling, H., & Pedersen, A. (2017). Long-term memory performance in adult ADHD. *Journal of Attention Disorders*, 21(4), 267–283. <https://doi.org/10.1177/1087054713510561>
- Skowronek, J. S., Leichtman, M. D., & Pillemer, D. B. (2008). Long-term episodic memory in children with attention-deficit/hyperactivity disorder. *Learning Disabilities Research and Practice*, 23(1), 25–35. <https://doi.org/10.1111/j.1540-5826.2007.00260.x>
- Smith-Spark, J. H., & Fisk, J. E. (2007). Working memory functioning in developmental dyslexia. *Memory (Hove, England)*, 15(1), 34–56. <https://doi.org/10.1080/09658210601043384>
- Smith-Spark, J. H., Henry, L. A., Messer, D. J., Edvardsdottir, E., & Zięcik, A. P. (2016). Executive functions in adults with developmental dyslexia. *Research in Developmental Disabilities*, 53–54, 323–341. <https://doi.org/10.1016/j.ridd.2016.03.001>

- Snowling, M. J., Gallagher, A., & Frith, U. (2003). Family risk of dyslexia is continuous: Individual differences in the precursors of reading skill. *Child Development*, 74(2), 358–373. <https://doi.org/10.1111/1467-8624.7402003>
- Snowling, M. J., Hulme, C., & Nation, K. (2020). Defining and understanding dyslexia: Past, present and future. *Oxford Review of Education*, 46(4), 501–513. <https://doi.org/10.1080/03054985.2020.1765756>
- Soke, G. N., Rosenberg, S. A., Hamman, R. F., Fingerlin, T., Robinson, C., Carpenter, L., Giarelli, E., Lee, L.-C., Wiggins, L. D., Durkin, M. S., & DiGuseppi, C. (2016). Brief report: Prevalence of self-injurious behaviors among children with autism spectrum disorder-A population-based study. *Journal of Autism and Developmental Disorders*, 46(11), 3607–3614. <https://doi.org/10.1007/s10803-016-2879-1>
- Soler-Gutiérrez, A.-M., Pérez-González, J.-C., & Mayas, J. (2023). Evidence of emotion dysregulation as a core symptom of adult ADHD: A systematic review. *PLOS One*, 18(1), Article e0280131. <https://doi.org/10.1371/journal.pone.0280131>
- Sonuga-Barke, E., Bitsakou, P., & Thompson, M. (2010). Beyond the dual pathway model: Evidence for the dissociation of timing, inhibitory, and delay-related impairments in attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 49(4), 345–355. <https://doi.org/10.1016/j.jaac.2009.12.018>
- South, M., & Rodgers, J. (2017). Sensory, emotional and cognitive contributions to anxiety in autism spectrum disorders. *Frontiers in Human Neuroscience*, 11, 20. <https://doi.org/10.3389/fnhum.2017.00020>
- Stack-Cutler, H. L., Parrila, R. K., & Torppa, M. (2016). University students with reading difficulties: Do perceived supports and comorbid difficulties predict Well-being and GPA? *Learning Disabilities Research and Practice*, 31(1), 45–55. <https://doi.org/10.1111/ldrp.12092>. ***
- Stark, S., Geertsema, S., Le Roux, M., & Bothma, E. (2022). Investigating the prevalence and comorbidity of attention deficit hyperactivity disorder and developmental dyslexia in learners in a South African practice. *South African Journal of Childhood Education*, 12(1), 1085. <https://doi.org/10.4102/sajce.v12i1.1085>. ***
- Steele, S. C., & Mills, M. T. (2011). Vocabulary intervention for school-age children with language impairment: A review of evidence and good practice. *Child Language Teaching and Therapy*, 27(3), 354–370. <https://doi.org/10.1177/0265659011412247>
- Steinley, D., & Brusco, M. J. (2008). Selection of variables in cluster analysis: An empirical comparison of eight procedures. *Psychometrika*, 73(1), 125–144. <https://doi.org/10.1007/s11336-007-9019-y>
- Stenneken, P., Egetemeir, J., Schulte-Körne, G., Müller, H. J., Schneider, W. X., & Finke, K. (2011). Slow perceptual processing at the core of developmental dyslexia: A parameter-

- based assessment of visual attention. *Neuropsychologia*, 49(12), 3454–3465.
<https://doi.org/10.1016/j.neuropsychologia.2011.08.021>
- Stevenson, J., Langley, K., Pay, H., Payton, A., Worthington, J., Ollier, W., & Thapar, A. (2005). Attention deficit hyperactivity disorder with reading disabilities: Preliminary genetic findings on the involvement of the ADRA2A gene. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 46(10), 1081–1088.
<https://doi.org/10.1111/j.1469-7610.2005.01533.x>. ***
- Stoll, C. R. T., Izadi, S., Fowler, S., Green, P., Suls, J., & Colditz, G. A. (2019). The value of a second reviewer for study selection in systematic reviews. *Research Synthesis Methods*, 10(4), 539–545. <https://doi.org/10.1002/jrsm.1369>
- Stubenrauch, C., Freund, J., Alecu de Flers, S., Scharke, W., Braun, M., Jacobs, A. M., & Konrad, K. (2014). Nonword reading and Stroop interference: What differentiates attention-deficit/hyperactivity disorder and reading disability? *Journal of Clinical and Experimental Neuropsychology*, 36(3), 244–260.
<https://doi.org/10.1080/13803395.2013.878690>. ***
- Sunde, H. F., Kleppestø, T. H., Gustavson, K., Nordmo, M., Reme, B.-A., & Torvik, F. A. (2022). The ADHD deficit in school performance across sex and parental education: A prospective sibling-comparison register study of 344,152 Norwegian adolescents. *JCPP Advances*, 2(1), Article e12064. <https://doi.org/10.1002/jcv2.12064>
- Swanson, H. L., Mink, J., & Bocian, K. M. (1999). Cognitive processing deficits in poor readers with symptoms of reading disabilities and ADHD: More alike than different? *Journal of Educational Psychology*, 91(2), 321–333. <https://doi.org/10.1037/0022-0663.91.2.321>. ***
- Swanson, J. M., Schuck, S., Porter, M. M., Carlson, C., Hartman, C. A., Sergeant, J. A., Clevenger, W., Wasdell, M., McCleary, R., Lakes, K., & Wigal, T. (2012). Categorical and dimensional definitions and evaluations of symptoms of ADHD: History of the SNAP and the SWAN rating scales. *The International Journal of Educational and Psychological Assessment*, 10(1), 51–70.
- Tajik-Parvinchi, D., Farmus, L., Tablon Modica, P., Cribbie, R. A., & Weiss, J. A. (2021). The role of cognitive control and emotion regulation in predicting mental health problems in children with neurodevelopmental disorders. *Child: Care, Health and Development*, 47(5), 608–617. <https://doi.org/10.1111/cch.12868>
- Tan, D. W., Crane, L., Haar, T., Heyworth, M., Poulsen, R., & Pellicano, E. (2025). Reporting community involvement in autism research: Findings from the journal autism. *Autism*, 29(2), 490–503. <https://doi.org/10.1177/13623613241275263>
- Tannock, R., Martinussen, R., & Frijters, J. (2000). Naming speed performance and stimulant effects indicate effortful, semantic processing deficits in attention-deficit/hyperactivity disorder. *Journal of Abnormal Child Psychology*, 28(3), 237–252.
<https://doi.org/10.1023/a:1005192220001>. ***

- Tarpey, T. (2007). Linear transformations and the k-means clustering algorithm: Applications to clustering curves. *The American Statistician*, 61(1), 34–40. <https://doi.org/10.1198/000313007X171016>
- Taylor, H. G., Orchinik, L., Fristad, M. A., Minich, N., Klein, N., Espy, K. A., Schluchter, M., & Hack, M. (2019). Associations of attention deficit hyperactivity disorder (ADHD) at school entry with early academic progress in children born prematurely and full-term controls. *Learning and Individual Differences*, 69, 1–10. <https://doi.org/10.1016/j.lindif.2018.10.008>
- Taylor, J. L., & Seltzer, M. M. (2010). Changes in the autism behavioral phenotype during the transition to adulthood. *Journal of Autism and Developmental Disorders*, 40(12), 1431–1446. <https://doi.org/10.1007/s10803-010-1005-z>
- Tharwat, A., Gaber, T., Ibrahim, A., & Hassanien, A. E. (2017). Linear discriminant analysis: A detailed tutorial. *AI Communications*, 30(2), 169–190. <https://doi.org/10.3233/AIC-170729>
- The jamovi project. (2023). *jamovi* (version 2.3) [Computer software]. <https://www.jamovi.org>.
- Theiling, J., & Petermann, F. (2016). Neuropsychological profiles on the WAIS-IV of adults with ADHD. *Journal of Attention Disorders*, 20(11), 913–924. <https://doi.org/10.1177/1087054713518241>
- Thomas, R., Sanders, S., Doust, J., Beller, E., & Glasziou, P. (2015). Prevalence of attention-deficit/hyperactivity disorder: A systematic review and meta-analysis. *Pediatrics*, 135(4), 994–1001. <https://doi.org/10.1542/peds.2014-3482>
- Thompson, R. A., Lewis, M. D., & Calkins, S. D. (2008). Reassessing emotion regulation. *Child Development Perspectives*, 2(3), 124–131. <https://doi.org/10.1111/j.1750-8606.2008.00054.x>
- Thomson, J. B., Chenault, B., Abbott, R. D., Raskind, W. H., Richards, T., Aylward, E., & Berninger, V. W. (2005). Converging evidence for attentional influences on the orthographic word form in child dyslexics. *Journal of Neurolinguistics*, 18(2), 93–126. <https://doi.org/10.1016/j.jneuroling.2004.11.005>. ***
- Thorndike, R. L. (1953). Who belongs in the family? *Psychometrika*, 18(4), 267–276. <https://doi.org/10.1007/BF02289263>
- Tibshirani, R., Walther, G., & Hastie, T. (2001). Estimating the number of clusters in a data set via the gap statistic. *Journal of the Royal Statistical Society Series B*, 63(2), 411–423. <https://doi.org/10.1111/1467-9868.00293>
- Tiffin-Richards, M. C., Hasselhorn, M., Woerner, W., Rothenberger, A., & Banaschewski, T. (2008). Phonological short-term memory and central executive processing in attention-deficit/hyperactivity disorder with/without dyslexia – evidence of cognitive overlap.

Journal of Neural Transmission, 115(2), 227–234. <https://doi.org/10.1007/s00702-007-0816-3>. ***

Toffalini, E., Buono, S., & Cornoldi, C. (2022). The structure, profile, and diagnostic significance of intelligence in children with ADHD are impressively similar to those of children with a specific learning disorder. *Research in Developmental Disabilities*, 129, Article 104306. <https://doi.org/10.1016/j.ridd.2022.104306> ***

Tolin, D. F. (2010). Is cognitive-behavioral therapy more effective than other therapies? A meta-analytic review. *Clinical Psychology Review*, 30(6), 710–720. <https://doi.org/10.1016/j.cpr.2010.05.003>

Tomblin, J. B., Records, N. L., Buckwalter, P., Zhang, X., Smith, E., & O'Brien, M. (1997). Prevalence of specific language impairment in kindergarten children. *Journal of Speech, Language, and Hearing Research: JSLHR*, 40(6), 1245–1260. <https://doi.org/10.1044/jslhr.4006.1245>

Toplak, M. E., Rucklidge, J. J., Hetherington, R., John, S. C., & Tannock, R. (2003). Time perception deficits in attention-deficit/hyperactivity disorder and comorbid reading difficulties in child and adolescent samples. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 44(6), 888–903. <https://doi.org/10.1111/1469-7610.00173> ***

Tosto, C., Hasegawa, T., Mangina, E., Chifari, A., Treacy, R., Merlo, G., & Chiazese, G. (2021). Exploring the effect of an augmented reality literacy programme for reading and spelling difficulties for children diagnosed with ADHD. *Virtual Reality*, 25(3), 879–894. <https://doi.org/10.1007/s10055-020-00485-z>. ***

Tulsky, D. S., Carlozzi, N., Chiaravalloti, N. D., Beaumont, J. L., Kisala, P. A., Mungas, D., Conway, K., & Gershon, R. (2014). NIH Toolbox Cognition Battery (NIHTB-CB): List sorting test to measure working memory. *Journal of the International Neuropsychological Society*, 20(6), 599–610. <https://doi.org/10.1017/S135561771400040X>

Tureck, K., Matson, J. L., May, A., & Turygin, N. (2013). Externalizing and tantrum behaviours in children with ASD and ADHD compared to children with ADHD. *Developmental Neurorehabilitation*, 16(1), 52–57. <https://doi.org/10.3109/17518423.2012.719245>

Turker, S., Seither-Preisler, A., Reiterer, S. M., & Schneider, P. (2019). Cognitive and behavioural weaknesses in children with reading disorder and AD(H)D. *Scientific Reports*, 9(1), Article 15185. <https://doi.org/10.1038/s41598-019-51372-w>. ***

Ullman, M. T., & Pullman, M. Y. (2015). A compensatory role for declarative memory in neurodevelopmental disorders. *Neuroscience and Biobehavioral Reviews*, 51, 205–222. <https://doi.org/10.1016/j.neubiorev.2015.01.008>

Underwood, J. F. G., Kendall, K. M., Berrett, J., Lewis, C., Anney, R., van den Bree, M. B. M., & Hall, J. (2019). Autism spectrum disorder diagnosis in adults: Phenotype and

- genotype findings from a clinically derived cohort. *The British Journal of Psychiatry*, 215(5), 647–653. <https://doi.org/10.1192/bjp.2019.30>
- Vakil, E., Blachstein, H., Wertman-Elad, R., & Greenstein, Y. (2012). Verbal learning and memory as measured by the Rey-Auditory Verbal Learning Test: ADHD with and without learning disabilities. *Child Neuropsychology: A Journal on Normal and Abnormal Development in Childhood and Adolescence*, 18(5), 449–466. <https://doi.org/10.1080/09297049.2011.613816>. ***
- van de Velden, M., D’Enza, A. I., & Yamamoto, M. (2019). Special feature: Dimension reduction and cluster analysis. *Behaviormetrika*, 46(2), 239–241. <https://doi.org/10.1007/s41237-019-00092-6>
- Van De Voorde, S., Roeyers, H., Verté, S., & Wiersema, J. R. (2010). Working memory, response inhibition, and within-subject variability in children with attention-deficit/hyperactivity disorder or reading disorder. *Journal of Clinical and Experimental Neuropsychology*, 32(4), 366–379. <https://doi.org/10.1080/13803390903066865>. ***
- Van Der Meer, J. M. J., Oerlemans, A. M., Van Steijn, D. J., Lappenschaar, M. G. A., De Sonnevile, L. M. J., Buitelaar, J. K., & Rommelse, N. N. J. (2012). Are autism spectrum disorder and attention-deficit/hyperactivity disorder different manifestations of one overarching disorder? Cognitive and symptom evidence from a clinical and population-based sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 51(11), 1160–1172.e3. <https://doi.org/10.1016/j.jaac.2012.08.024>
- van der Putten, W. J., Mol, A. J. J., Groenman, A. P., Radhoe, T. A., Torenvliet, C., van Rentergem, J. A. A., & Geurts, H. M. (2024). Is camouflaging unique for autism? A comparison of camouflaging between adults with autism and ADHD. *Autism Research: Official Journal of the International Society for Autism Research*, 17(4), 812–823. <https://doi.org/10.1002/aur.3099>
- Van Steensel, F. J. A., Bögels, S. M., & de Bruin, E. I. (2013). Psychiatric comorbidity in children with autism spectrum disorders: A comparison with children with ADHD. *Journal of Child and Family Studies*, 22(3), 368–376. <https://doi.org/10.1007/s10826-012-9587-z>
- Varanda, C. A., & Fernandes, F. D. M. (2017). Cognitive flexibility training intervention among children with autism: A longitudinal study. *Psicologia, Reflexao e Critica: Revista Semestral Do Departamento de Psicologia da UFRGS*, 30(1), 15. <https://doi.org/10.1186/s41155-017-0069-5>
- Vehtari, A., Gelman, A., & Gabry, J. (2017). Practical Bayesian model evaluation using leave-one-out cross-validation and WAIC. *Statistics and Computing*, 27(5), 1413–1432. <https://doi.org/10.1007/s11222-016-9696-4>
- Verté, S., Geurts, H. M., Roeyers, H., Oosterlaan, J., & Sergeant, J. A. (2006). Executive functioning in children with an autism spectrum disorder: Can we differentiate within the spectrum? *Journal of Autism and Developmental Disorders*, 36(3), 351–372. <https://doi.org/10.1007/s10803-006-0074-5>

- Verwimp, C., Tijms, J., Snellings, P., Haslbeck, J. M. B., & Wiers, R. W. (2023). A network approach to dyslexia: Mapping the reading network. *Development and Psychopathology*, 35(3), 1011–1025. <https://doi.org/10.1017/S0954579421000365>
- Viechtbauer, W. (2005). Bias and efficiency of meta-analytic variance estimators in the random-effects model. *Journal of Educational and Behavioral Statistics*, 30(3), 261–293. <https://doi.org/10.3102/10769986030003261>
- Viechtbauer, W. (2010). Conducting meta-analyses in R with the metafor package. *Journal of Statistical Software*, 36, 1–48. <https://doi.org/10.18637/jss.v036.i03>
- Visser, E. M., Berger, H. J., Van Schrojenstein Lantman-De Valk, H. M., Prins, J. B., & Teunisse, J. P. (2015). Cognitive shifting and externalising problem behaviour in intellectual disability and autism spectrum disorder. *Journal of Intellectual Disability Research: JIDR*, 59(8), 755–766. <https://doi.org/10.1111/jir.12182>
- Visser, L., Kalmar, J., Linkersdörfer, J., Görgen, R., Rothe, J., Hasselhorn, M., & Schulte-Körne, G. (2020). Comorbidities between specific learning disorders and psychopathology in elementary school children in Germany. *Frontiers in Psychiatry*, 11(292), 292. <https://doi.org/10.3389/fpsy.2020.00292>. ***
- Vogindroukas, I., Stankova, M., Chelas, E.-N., & Proedrou, A. (2022). Language and speech characteristics in autism. *Neuropsychiatric Disease and Treatment*, 18, 2367–2377. <https://doi.org/10.2147/NDT.S331987>
- Voigt, R. G., Katusic, S. K., Colligan, R. C., Killian, J. M., Weaver, A. L., & Barbaresi, W. J. (2017). Academic achievement in adults with a history of childhood attention-deficit/hyperactivity disorder: A population-based prospective study. *Journal of Developmental and Behavioral Pediatrics*, 38(1), 1–11. <https://doi.org/10.1097/DBP.0000000000000358>. ***
- Vos, M., Rommelse, N. N. J., Franke, B., Oosterlaan, J., Heslenfeld, D. J., Hoekstra, P. J., Klein, M., Faraone, S. V., Buitelaar, J. K., & Hartman, C. A. (2022). Characterizing the heterogeneous course of inattention and hyperactivity-impulsivity from childhood to young adulthood. *European Child and Adolescent Psychiatry*, 31(8), 1–11. <https://doi.org/10.1007/s00787-021-01764-z>
- Wadsworth, S. J., DeFries, J. C., Olson, R. K., & Willcutt, E. G. (2007). Colorado longitudinal twin study of reading disability. *Annals of Dyslexia*, 57(2), 139–160. <https://doi.org/10.1007/s11881-007-0009-7>. ***
- Wager, T. D., & Smith, E. E. (2003). Neuroimaging studies of working memory: A meta-analysis. *Cognitive, Affective and Behavioral Neuroscience*, 3(4), 255–274. <https://doi.org/10.3758/cabn.3.4.255>
- Wagner, R. K., Zirps, F. A., Edwards, A. A., Wood, S. G., Joyner, R. E., Becker, B. J., Liu, G., & Beal, B. (2020). The prevalence of dyslexia: A new approach to its estimation.

Journal of Learning Disabilities, 53(5), 354–365.
<https://doi.org/10.1177/0022219420920377>

Wang, L.-C., & Chung, K. K. H. (2018). Co-morbidities in Chinese children with attention deficit/hyperactivity disorder and reading disabilities. *Dyslexia*, 24(3), 276–293.
<https://doi.org/10.1002/dys.1579>. ***

Ware, C., Dautricourt, S., Gonneaud, J., & Chételat, G. (2021). Does second language learning promote neuroplasticity in aging? A systematic review of cognitive and neuroimaging studies. *Frontiers in Aging Neuroscience*, 13, Article 706672.
<https://doi.org/10.3389/fnagi.2021.706672>

Wassenberg, R., Hendriksen, J. G. M., Hurks, P. P. M., Feron, F. J. M., Vles, J. S. H., & Jolles, J. (2010). Speed of language comprehension is impaired in ADHD. *Journal of Attention Disorders*, 13(4), 374–385. <https://doi.org/10.1177/1087054708326111>. ***

Weintraub, S., Dikmen, S. S., Heaton, R. K., Tulsky, D. S., Zelazo, P. D., Bauer, P. J., Carlozzi, N. E., Slotkin, J., Blitz, D., Wallner-Allen, K., Fox, N. A., Beaumont, J. L., Mungas, D., Nowinski, C. J., Richler, J., Deocampo, J. A., Anderson, J. E., Manly, J. J., Borosh, B., . . . Gershon, R. C. (2013). Cognition assessment using the NIH Toolbox. *Neurology*, 80(11) (Suppl. 3), S54–S64.
<https://doi.org/10.1212/WNL.0b013e3182872ded>

Weintraub, S., Dikmen, S. S., Heaton, R. K., Tulsky, D. S., Zelazo, P. D., Slotkin, J., Carlozzi, N. E., Bauer, P. J., Wallner-Allen, K., Fox, N., Havlik, R., Beaumont, J. L., Mungas, D., Manly, J. J., Moy, C., Conway, K., Edwards, E., Nowinski, C. J., & Gershon, R. (2014). The cognition battery of the NIH toolbox for assessment of neurological and behavioral function: Validation in an adult sample. *Journal of the International Neuropsychological Society*, 20(6), 567–578.
<https://doi.org/10.1017/S1355617714000320>

Westervelt, D., Johnson, D. C., Westervelt, M. D., & Murrill, S. (1998). Changes in self-concept and academic skills during a multimodal summer camp program. *Annals of Dyslexia*, 48(1), 189–212. <https://doi.org/10.1007/s11881-998-0009-2>. ***

Westrupp, E. M., Reilly, S., McKean, C., Law, J., Mensah, F., & Nicholson, J. M. (2020). Vocabulary development and trajectories of behavioral and emotional difficulties via academic ability and peer problems. *Child Development*, 91(2), e365–e382.
<https://doi.org/10.1111/cdev.13219>

Whipple, B. D., & Nelson, J. M. (2016). Naming speed of adolescents and young adults with attention deficit hyperactivity disorder: Differences in alphanumeric versus color/object naming. *Archives of Clinical Neuropsychology*, 31(1), 66–78.
<https://doi.org/10.1093/arclin/acv061>. ***

Wickham, H., & Sievert, C. (2009). *ggplot2: Elegant graphics for data analysis*. Springer.
<https://doi.org/10.1007/978-0-387-98141-3>

- Wickham, H., Miller, E., & Smith, D. (2025). *haven: Import and Export 'SPSS'. 'Stata' and "SAS" Files. R Package Version 2.5.5*. <https://haven.tidyverse.org>
- Willcutt, E. G., & Pennington, B. F. (2000). Comorbidity of reading disability and attention-deficit/hyperactivity disorder: Differences by gender and subtype. *Journal of Learning Disabilities*, 33(2), 179–191. <https://doi.org/10.1177/002221940003300206>. ***
- Willcutt, E. G., Betjemann, R. S., McGrath, L. M., Chhabildas, N. A., Olson, R. K., DeFries, J. C., & Pennington, B. F. (2010). Etiology and neuropsychology of comorbidity between RD and ADHD: The case for multiple-deficit models. *Cortex; a Journal Devoted to the Study of the Nervous System and Behavior*, 46(10), 1345–1361. <https://doi.org/10.1016/j.cortex.2010.06.009>
- Willcutt, E. G., Pennington, B. F., Boada, R., Ogline, J. S., Tunick, R. A., Chhabildas, N. A., & Olson, R. K. (2001). A comparison of the cognitive deficits in reading disability and attention-deficit/hyperactivity disorder. *Journal of Abnormal Psychology*, 110(1), 157–172. <https://doi.org/10.1037//0021-843x.110.1.157> ***
- Wiśniewska, B., Baranowska, W., & Wendorff, J. (2007). The assessment of comorbid disorders in ADHD children and adolescents. *Advances in Medical Sciences*, 52(Suppl. 1), 215–217. ***.
- Wodka, E. L., Mahone, E. M., Blankner, J. G., Larson, J. C. G., Fotedar, S., Denckla, M. B., & Mostofsky, S. H. (2007). Evidence that response inhibition is a primary deficit in ADHD. *Journal of Clinical and Experimental Neuropsychology*, 29(4), 345–356. <https://doi.org/10.1080/13803390600678046>
- Wolff, B., Franco, V. R., Magiati, I., Cooper, M. N., Roberts, R., Skoss, R., & Glasson, E. J. (2023). Individual-level risk and resilience factors associated with mental health in siblings of individuals with neurodevelopmental conditions: A network analysis. *Developmental Neuropsychology*, 48(3), 112–134. <https://doi.org/10.1080/87565641.2023.2190119>
- World Health Organization. (2019). *International statistical classification of diseases and related health problems* (11th ed.). <https://icd.who.int/>
- Wu, K. K., Anderson, V., & Castiello, U. (2002). Neuropsychological evaluation of deficits in executive functioning for ADHD children with or without learning disabilities. *Developmental Neuropsychology*, 22(2), 501–531. https://doi.org/10.1207/S15326942DN2202_5. ***
- Yang, L., Li, C., Li, X., Zhai, M., An, Q., Zhang, Y., Zhao, J., & Weng, X. (2022). Prevalence of developmental dyslexia in primary school children: A systematic review and meta-analysis. *Brain Sciences*, 12(2), 240. <https://doi.org/10.3390/brainsci12020240>
- Ying, X. (2019). An overview of overfitting and its solutions. In *Journal of Physics: Conference Series*. IOP Publishing, 1168. <https://doi.org/10.1088/1742-6596/1168/2/022022>

- Yoshimasu, K., Barbaresi, W. J., Colligan, R. C., Killian, J. M., Voigt, R. G., Weaver, A. L., & Katusic, S. K. (2010). Gender, attention-deficit/hyperactivity disorder, and reading disability in a population-based birth cohort. *Pediatrics*, 126(4), e788–e795. <https://doi.org/10.1542/peds.2010-1187>. ***
- Yue, X., Liu, L., Chen, W., Preece, D. A., Liu, Q., Li, H., Wang, Y., & Qian, Q. (2022). Affective-cognitive-behavioral heterogeneity of attention-deficit/hyperactivity disorder (ADHD): Emotional dysregulation as a sentinel symptom differentiating “ADHD-simplex” and “ADHD-complex” syndromes? *Journal of Affective Disorders*, 307, 133–141. <https://doi.org/10.1016/j.jad.2022.03.065>
- Zahn-Waxler, C., Klimes-Dougan, B., & Slattery, M. J. (2000). Internalizing problems of childhood and adolescence: Prospects, pitfalls, and progress in understanding the development of anxiety and depression. *Development and Psychopathology*, 12(3), 443–466. <https://doi.org/10.1017/S0954579400003102>
- Zapparrata, N. M., Brooks, P. J., & Ober, T. M. (2023). Slower processing speed in autism spectrum disorder: A meta-analytic investigation of time-based tasks. *Journal of Autism and Developmental Disorders*, 53(12), 4618–4640. <https://doi.org/10.1007/s10803-022-05736-3>
- Zauche, L. H., Thul, T. A., Mahoney, A. E. D., & Stapel-Wax, J. L. (2016). Influence of language nutrition on children’s language and cognitive development: An integrated review. *Early Childhood Research Quarterly*, 36, 318–333. <https://doi.org/10.1016/j.ecresq.2016.01.015>
- Zdorovtsova, N., Jones, J., Akarca, D., Benhamou, E., The Calm Team, & Astle, D. E. (2023). Exploring neural heterogeneity in inattention and hyperactivity. *Cortex; a Journal Devoted to the Study of the Nervous System and Behavior*, 164, 90–111. <https://doi.org/10.1016/j.cortex.2023.04.001>
- Zelazo, P. D. (2006). The Dimensional Change Card Sort (DCCS): A method of assessing executive function in children. *Nature Protocols*, 1(1), 297–301. <https://doi.org/10.1038/nprot.2006.46>
- Zelazo, P. D., Anderson, J. E., Richler, J., Wallner-Allen, K., Beaumont, J. L., Conway, K. P., Gershon, R., & Weintraub, S. (2014). NIH Toolbox Cognition Battery (CB): Validation of executive function measures in adults. *Journal of the International Neuropsychological Society: JINS*, 20(6), 620–629. <https://doi.org/10.1017/S1355617714000472>
- Zhang, M., Huang, Y., Jiao, J., Yuan, D., Hu, X., Yang, P., Zhang, R., Wen, L., Situ, M., Cai, J., Sun, X., Guo, K., Huang, X., & Huang, Y. (2022). Transdiagnostic symptom subtypes across autism spectrum disorders and attention deficit hyperactivity disorder: Validated by measures of neurocognition and structural connectivity. *BMC Psychiatry*, 22(1), 102. <https://doi.org/10.1186/s12888-022-03734-4>
- Zhang, Z., Feng, J., Xue, Y., Jia, F., & Wang, T. (2022). Planning processing in ADHD with comorbid reading disabilities is worse than in ADHD: Based on Das-Naglieri Cognitive

Assessment System. *Frontiers in Pediatrics*, 10, Article 898348.
<https://doi.org/10.3389/fped.2022.898348>. ***

- Zhao, S., Zhang, B., Yang, J., Zhou, J., & Xu, Y. (2024). Linear discriminant analysis. *Nature Reviews Methods Primers*, 4(1), 70. <https://doi.org/10.1038/s43586-024-00346-y>
- Zheng, S., Hume, K. A., Able, H., Bishop, S. L., & Boyd, B. A. (2020). Exploring developmental and behavioral heterogeneity among preschoolers with ASD: A cluster analysis on principal components. *Autism Research: Official Journal of the International Society for Autism Research*, 13(5), 796–809. <https://doi.org/10.1002/aur.2263>
- Zhou, Q., Ye, X., Wei, C., Wu, Y., Ren, P., Lin, X., Li, L., Xiang, W., & Xiao, L. (2023). Network analysis of ADHD symptoms and cognitive profiles in children. *Neuropsychiatric Disease and Treatment*, 19, 1207–1219. <https://doi.org/10.2147/NDT.S409503>
- Zuppardo, L., Serrano, F., Pirrone, C., & Rodriguez-Fuentes, A. (2023). More than words: Anxiety, self-esteem, and behavioral problems in children and adolescents with dyslexia. *Learning Disability Quarterly*, 46(2), 77–91. <https://doi.org/10.1177/07319487211041103>

Supplementary Materials 1 (Chapter 2: Systematic Review)

Appendix A. Prisma 2020 Checklist

Section and Topic	Item #	Checklist item	Location where item is reported
TITLE			
Title	1	Identify the report as a systematic review.	X
ABSTRACT			
Abstract	2	See the PRISMA 2020 for Abstracts checklist.	X
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of existing knowledge.	X
Objectives	4	Provide an explicit statement of the objective(s) or question(s) the review addresses.	X
METHODS			
Eligibility criteria	5	Specify the inclusion and exclusion criteria for the review and how studies were grouped for the syntheses.	X
Information sources	6	Specify all databases, registers, websites, organisations, reference lists and other sources searched or consulted to identify studies. Specify the date when each source was last searched or consulted.	X
Search strategy	7	Present the full search strategies for all databases, registers and websites, including any filters and limits used.	X
Selection process	8	Specify the methods used to decide whether a study met the inclusion criteria of the review, including how many reviewers screened each record and each report retrieved, whether they worked independently, and if applicable, details of automation tools used in the process.	X
Data collection process	9	Specify the methods used to collect data from reports, including how many reviewers collected data from each report, whether they worked independently, any processes for obtaining or confirming data from study investigators, and if applicable, details of automation tools used in the process.	X
Data items	10a	List and define all outcomes for which data were sought. Specify whether all results that were compatible with each outcome domain in each study were sought (e.g. for all measures, time points, analyses), and if not, the methods used to decide which results to collect.	X
	10b	List and define all other variables for which data were sought (e.g. participant and intervention characteristics, funding sources). Describe any assumptions made about any missing or unclear information.	X
Study risk of bias assessment	11	Specify the methods used to assess risk of bias in the included studies, including details of the tool(s) used, how many reviewers assessed each study and whether they worked independently, and if applicable, details of automation tools used in the process.	X

Section and Topic	Item #	Checklist item	Location where item is reported
Effect measures	12	Specify for each outcome the effect measure(s) (e.g. risk ratio, mean difference) used in the synthesis or presentation of results.	X
Synthesis methods	13a	Describe the processes used to decide which studies were eligible for each synthesis (e.g. tabulating the study intervention characteristics and comparing against the planned groups for each synthesis (item #5)).	X
	13b	Describe any methods required to prepare the data for presentation or synthesis, such as handling of missing summary statistics, or data conversions.	X
	13c	Describe any methods used to tabulate or visually display results of individual studies and syntheses.	X
	13d	Describe any methods used to synthesize results and provide a rationale for the choice(s). If meta-analysis was performed, describe the model(s), method(s) to identify the presence and extent of statistical heterogeneity, and software package(s) used.	X
	13e	Describe any methods used to explore possible causes of heterogeneity among study results (e.g. subgroup analysis, meta-regression).	X
	13f	Describe any sensitivity analyses conducted to assess robustness of the synthesized results.	X
Reporting bias assessment	14	Describe any methods used to assess risk of bias due to missing results in a synthesis (arising from reporting biases).	X
Certainty assessment	15	Describe any methods used to assess certainty (or confidence) in the body of evidence for an outcome.	X
RESULTS			
Study selection	16a	Describe the results of the search and selection process, from the number of records identified in the search to the number of studies included in the review, ideally using a flow diagram.	X
	16b	Cite studies that might appear to meet the inclusion criteria, but which were excluded, and explain why they were excluded.	X
Study characteristics	17	Cite each included study and present its characteristics.	X
Risk of bias in studies	18	Present assessments of risk of bias for each included study.	X
Results of individual studies	19	For all outcomes, present, for each study: (a) summary statistics for each group (where appropriate) and (b) an effect estimate and its precision (e.g. confidence/credible interval), ideally using structured tables or plots.	X
Results of syntheses	20a	For each synthesis, briefly summarise the characteristics and risk of bias among contributing studies.	
	20b	Present results of all statistical syntheses conducted. If meta-analysis was done, present for each the summary estimate and its precision (e.g. confidence/credible interval) and measures of statistical heterogeneity. If comparing groups, describe the direction of the effect.	X
	20c	Present results of all investigations of possible causes of heterogeneity among study results.	X
	20d	Present results of all sensitivity analyses conducted to assess the robustness of the synthesized results.	X

Section and Topic	Item #	Checklist item	Location where item is reported
Reporting biases	21	Present assessments of risk of bias due to missing results (arising from reporting biases) for each synthesis assessed.	X
Certainty of evidence	22	Present assessments of certainty (or confidence) in the body of evidence for each outcome assessed.	X
DISCUSSION			
Discussion	23a	Provide a general interpretation of the results in the context of other evidence.	X
	23b	Discuss any limitations of the evidence included in the review.	X
	23c	Discuss any limitations of the review processes used.	X
	23d	Discuss implications of the results for practice, policy, and future research.	X
OTHER INFORMATION			
Registration and protocol	24a	Provide registration information for the review, including register name and registration number, or state that the review was not registered.	X
	24b	Indicate where the review protocol can be accessed, or state that a protocol was not prepared.	X
	24c	Describe and explain any amendments to information provided at registration or in the protocol.	X
Support	25	Describe sources of financial or non-financial support for the review, and the role of the funders or sponsors in the review.	X
Competing interests	26	Declare any competing interests of review authors.	X
Availability of data, code and other materials	27	Report which of the following are publicly available and where they can be found: template data collection forms; data extracted from included studies; data used for all analyses; analytic code; any other materials used in the review.	X

From: Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ 2021;372:n71. doi: 10.1136/bmj.n71

Appendix B. Full search strategy for all databases searched

Database	Search Strategy
Embase (1974 to present)	#1 attention deficit hyperactivity disorder/ #2 (adhd or adhs or addh or "ad/hd" or hkd).ti,ab,kw. #3 ((attention* or behavi*) adj3 (defic* or dysfunc* or disorder*)).ti,ab,kw. #4 ((disrupt* or defian*) adj3 (disorder* or behavi*)).ti,ab,kw. #5 (impulsiv* or inattention or inattentiv* or hyperkin* or hyper-kin*).ti,ab,kw. #6 (hyperactiv* or hyper-activ*).ti,ab,kw. #7 or/1-6 #8 Dyslexia/ or reading/ or dyscalculia/ or *learning disorder/ #9 dyslexi*.ti,ab,kw. #10 (reading* adj3 (comprehen* or disorder* or disabilit* or problem* or difficult* or deficient* or fluenc* or skill*)).ti,ab,kw. #11 ((word or text) adj1 blind*).ti,ab,kw. #12 dyscalculi*.ti,ab,kw. #13 ((math* or number* or arithmetic*) adj3 (comprehen* or disorder* or disabilit* or problem* or difficult* or deficient* or fluenc* or skill*)).ti,ab,kw. #14 or/8-13 #15 comorbidity/ #16 (co-occurr* adj4 (symptom* or difficult* or need* or condition* or problem* or disorder* or psycho* or diagnos*)).ti,ab,kw. #17 ((symptom* or condition* or disorder* or characteristic* or deficit* or neuro*) adj4 overlap*).ti,ab,kw. #18 (comorbid*).ti,ab,kw. #19 or/15-18 #20 7 and 14 and 19
MEDLINE (1946 to present)	#1 "attention deficit and disruptive behavior disorders"/ or attention deficit disorder with hyperactivity/ #2 (adhd or adhs or addh or "ad/hd" or hkd).ti,ab,kw. #3 ((attention* or behavi*) adj3 (defic* or dysfunc* or disorder*)).ti,ab,kw. #4 ((disrupt* or defian*) adj3 (disorder* or behavi*)).ti,ab,kw. #5 (impulsiv* or inattention or inattentiv* or hyperkin* or hyper-kin*).ti,ab,kw. #6 (hyperactiv* or hyper-activ*).ti,ab,kw. #7 or/1-6 #8 exp Dyslexia/ or reading/ or dyscalculia/ or learning disabilities/ #9 dyslexi*.ti,ab,kw. #10 (reading* adj3 (comprehen* or disorder* or disabilit* or problem* or difficult* or deficient* or fluenc* or skill*)).ti,ab,kw. #11 ((word or text) adj1 blind*).ti,ab,kw. #12 dyscalculi*.ti,ab,kw. #13 ((math* or number* or arithmetic*) adj3 (comprehen* or disorder* or disabilit* or problem* or difficult* or deficient* or fluenc* or skill*)).ti,ab,kw. #14 or/8-13 #15 exp comorbidity/ or epidemiologic factors/ #16 (co-occurr* adj4 (symptom* or difficult* or need* or condition* or problem* or disorder* or psycho* or diagnos*)).ti,ab,kw.

#17 ((symptom* or condition* or disorder* or characteristic* or deficit* or neuro*) adj4 overlap*).ti,ab,kw.

#18 (comorbid*).ti,ab,kw.

#19 or/15-18

#20 7 and 14 and 19

PsycINFO
(1806 to
present)

(MAINSUBJECT.EXACT.EXPLODE("Attention Deficit Disorder") OR abstract((adhd OR adhs OR addh OR "ad/hd" OR hkd) OR ((attention* OR behavi*) NEAR/2 (defic* OR dysfunc* OR disorder*)) OR ((disrupt* OR defian*) NEAR/2 (disorder* OR behavi*)) OR (impulsiv* OR inattention OR inattentiv* OR hyperkin* OR hyper-kin*) OR (hyperactiv* OR hyper-activ*))) AND ((MAINSUBJECT.EXACT.EXPLODE("Reading Disabilities") OR MAINSUBJECT.EXACT("Learning Disabilities") OR MAINSUBJECT.EXACT("Dyslexia")) OR abstract(dyslexi* OR (reading* NEAR/2 (comprehen* OR disorder* OR disabilit* OR problem* OR difficult* OR deficient* OR fluenc* OR skill*)) OR ((word OR text) NEAR/1 blind*) OR dyscalculi* OR ((math* OR number* OR arithmetic*) NEAR/2 (comprehen* OR disorder* OR disabilit* OR problem* OR difficult* OR deficient* OR fluenc* OR skill*)))) AND (MAINSUBJECT.EXACT("Comorbidity") OR abstract((co-occurr* NEAR/3 (symptom* OR difficult* OR need* OR condition* OR problem* OR disorder* OR psycho* OR diagnos*)) OR ((symptom* OR condition* OR disorder* OR characteristic* OR deficit* OR neuro*) NEAR/3 overlap*) OR comorbid*))

**Web of
Science**
(No time
period limit)

#1 (adhd or adhs or addh or "ad/hd" or hkd) or ((attention* or behavi*) NEAR/2 (defic* or dysfunc* or disorder*)) or ((disrupt* or defian*) NEAR/2 (disorder* or behavi*)) or (impulsiv* or inattention or inattentiv* or hyperkin* or hyper-kin*) or (hyperactiv* or hyper-activ*)
#2 dyslexi* or (reading* NEAR/2 (comprehen* or disorder* or disabilit* or problem* or difficult* or deficient* or fluenc* or skill*)) or ((word or text) NEAR/1 blind*) or dyscalculi* or ((math* or number* or arithmetic*) NEAR/2 (comprehen* or disorder* or disabilit* or problem* or difficult* or deficient* or fluenc* or skill*))
#3 (co-occurr* NEAR/3 (symptom* or difficult* or need* or condition* or problem* or disorder* or psycho* or diagnos*)) or ((symptom* or condition* or disorder* or characteristic* or deficit* or neuro*) NEAR/3 overlap*) or comorbid*
#4 1 AND 2 AND 3

CINAHL
(No time
period limit)

#1 (MH "Attention Deficit Hyperactivity Disorder") OR TI ((adhd or adhs or addh or "ad/hd" or hkd) or ((attention* or behavi*) NEAR/2 (defic* or dysfunc* or disorder*)) or ((disrupt* or defian*) NEAR/2 (disorder* or behavi*)) or (impulsiv* or inattention or inattentiv* or hyperkin* or hyper-kin*) or (hyperactiv* or hyper-activ*)) OR AB ((adhd or adhs or addh or "ad/hd" or hkd) or ((attention* or behavi*) NEAR/2 (defic* or dysfunc* or disorder*)) or ((disrupt* or defian*) NEAR/2 (disorder* or behavi*)) or (impulsiv* or inattention or inattentiv* or hyperkin* or hyper-kin*) or (hyperactiv* or hyper-activ*))
#2 ((MH "Dyscalculia") OR (MH "Reading Disorders+")) OR TI (dyslexi* or (reading* NEAR/2 (comprehen* or disorder* or disabilit* or problem* or difficult* or deficient* or fluenc* or skill*)) or ((word or text) NEAR/1 blind*) or dyscalculi* or ((math* or number* or arithmetic*) NEAR/2 (comprehen* or disorder* or disabilit* or problem* or difficult* or deficient* or fluenc* or skill*))) OR AB (dyslexi* or (reading* NEAR/2 (comprehen* or disorder* or disabilit* or problem* or difficult* or deficient* or fluenc* or skill*)) or ((word or text) NEAR/1 blind*) or dyscalculi* or ((math* or number* or

arithmetic*) NEAR/2 (comprehen* or disorder* or disabilit* or problem* or difficult* or deficien* or fluenc* or skill*))

#3 ((MH "Comorbidity") OR (MH "Epidemiology")) OR TI ((co-occurr* NEAR/3 (symptom* or difficult* or need* or condition* or problem* or disorder* or psycho* or diagnos*)) or ((symptom* or condition* or disorder* or characteristic* or deficit* or neuro*) NEAR/3 overlap*) or comorbid*) OR AB ((co-occurr* NEAR/3 (symptom* or difficult* or need* or condition* or problem* or disorder* or psycho* or diagnos*)) or ((symptom* or condition* or disorder* or characteristic* or deficit* or neuro*) NEAR/3 overlap*) or comorbid*)

#4 S1 AND S2 AND S3

PubMED (No time period limit) #1 (adhd or adhs or addh or "ad/hd" or hkd) or ((attention* or behavi*) AND (defic* or dysfunc* or disorder*)) or ((disrupt* or defian*) AND (disorder* or behavi*)) or (impulsiv* or inattention or inattentiv* or hyperkin* or hyper-kin*) or (hyperactiv* or hyper-activ*)

#2 dyslexi* or (reading* AND (comprehen* or disorder* or disabilit* or problem* or difficult* or deficien* or fluenc* or skill*)) or ((word or text) AND blind*) or dyscalculi* or ((math* or number* or arithmetic*) AND (comprehen* or disorder* or disabilit* or problem* or difficult* or deficien* or fluenc* or skill*))

#3 (co-occurr* AND (symptom* or difficult* or need* or condition* or problem* or disorder* or psycho* or diagnos*)) or ((symptom* or condition* or disorder* or characteristic* or deficit* or neuro*) AND overlap*) or comorbid*

Note. Key terms associated with dyscalculia were included in the search strategy. However, in the interest of simplifying the review, any studies relating to dyscalculia were later excluded (unless they included a separate ADHD+RD group).

JBI CRITICAL APPRAISAL CHECKLIST FOR STUDIES REPORTING PREVALENCE DATA

	Yes	No	Unclear	Not applicable
1. Was the sample frame appropriate to address the target population?	☐	☐	☐	☐
2. Were study participants sampled in an appropriate way?	☐	☐	☐	☐
3. Was the sample size adequate?	☐	☐	☐	☐
4. Were the study subjects and the setting described in detail?	☐	☐	☐	☐
5. Was the data analysis conducted with sufficient coverage of the identified sample?	☐	☐	☐	☐
6. Were valid methods used for the identification of the condition?	☐	☐	☐	☐
7. Was the condition measured in a standard, reliable way for all participants?	☐	☐	☐	☐
8. Was there appropriate statistical analysis?	☐	☐	☐	☐
9. Was the response rate adequate, and if not, was the low response rate managed appropriately?	☐	☐	☐	☐

Overall appraisal: Include ☐ Exclude ☐ Seek further info ☐

Comments (Including reason for exclusion)

JBI CRITICAL APPRAISAL CHECKLIST FOR STUDIES REPORTING PREVALENCE DATA

How to cite: Munn Z, Moola S, Lisy K, Riitano D, Tufanaru C. Methodological guidance for systematic reviews of observational epidemiological studies reporting prevalence and incidence data. Int J Evid Based Healthc. 2015;13(3):147–153.

Answers: Yes, No, Unclear or Not/Applicable

1. Was the sample frame appropriate to address the target population?

This question relies upon knowledge of the broader characteristics of the population of interest and the geographical area. If the study is of women with breast cancer, knowledge of at least the characteristics, demographics and medical history is needed. The term “target population” should not be taken to infer every individual from everywhere or with similar disease or exposure characteristics. Instead, give consideration to specific population characteristics in the study, including age range, gender, morbidities, medications, and other potentially influential factors. For example, a sample frame may not be appropriate to address the target population if a certain group has been used (such as those working for one organisation, or one profession) and the results then inferred to the target population (i.e. working adults). A sample frame may be appropriate when it includes almost all the members of the target population (i.e. a census, or a complete list of participants or complete registry data).

2. Were study participants recruited in an appropriate way?

Studies may report random sampling from a population, and the methods section should report how sampling was performed. Random probabilistic sampling from a defined subset of the population (sample frame) should be employed in most cases, however, random probabilistic sampling is not needed when everyone in the sampling frame will be included/analysed. For example, reporting on all the data from a good census is appropriate as a good census will identify everybody. When using cluster sampling, such as a random sample of villages within a region, the methods need to be clearly stated as the precision of the final prevalence estimate incorporates the clustering effect. Convenience samples, such as a street survey or interviewing lots of people at a public gatherings are not considered to provide a representative sample of the base population.

3. Was the sample size adequate?

The larger the sample, the narrower will be the confidence interval around the prevalence estimate, making the results more precise. An adequate sample size is important to ensure good precision of the final estimate. Ideally we are looking for evidence that the authors conducted a sample size calculation to determine an adequate sample size. This will estimate how many subjects are needed to produce a reliable estimate of the measure(s) of interest. For conditions with a low prevalence, a larger sample size is needed. Also consider sample sizes for subgroup (or characteristics) analyses, and whether these are appropriate. Sometimes, the study will be large enough (as in large national surveys) whereby a sample size calculation is not required. In these cases, sample size can be considered adequate.

When there is no sample size calculation and it is not a large national survey, the reviewers may consider conducting their own sample size analysis using the following formula: (Naing et al. 2006, Daniel 1999)

$$n = \frac{Z^2 P(1-P)}{d^2}$$

d²

Where:

n = sample size

Z = Z statistic for a level of confidence

P = Expected prevalence or proportion (in proportion of one; if 20%, P = 0.2)

d = precision (in proportion of one; if 5%, d=0.05)

Ref:

Naing L, Winn T, Rusli BN. Practical issues in calculating the sample size for prevalence studies Archives of Orofacial Sciences. 2006;1:9-14.

Daniel WW. Biostatistics: A Foundation for Analysis in the Health Sciences.

Edition. 7th ed. New York: John Wiley & Sons. 1999.

4. Were the study subjects and setting described in detail?

Certain diseases or conditions vary in prevalence across different geographic regions and populations (e.g. Women vs. Men, sociodemographic variables between countries). The study sample should be described in sufficient detail so that other researchers can determine if it is comparable to the population of interest to them.

5. Was data analysis conducted with sufficient coverage of the identified sample?

Coverage bias can occur when not all subgroups of the identified sample respond at the same rate. For instance, you may have a very high response rate overall for your study, but the response rate for a certain subgroup (i.e. older adults) may be quite low.

6. Were valid methods used for the identification of the condition?

Here we are looking for measurement or classification bias. Many health problems are not easily diagnosed or defined and some measures may not be capable of including or excluding appropriate levels or stages of the health problem. If the outcomes were assessed based on existing definitions or diagnostic criteria, then the answer to this question is likely to be yes. If the outcomes were assessed using observer reported, or self-reported scales, the risk of over- or under-reporting is increased, and objectivity is compromised. Importantly, determine if the measurement tools used were validated instruments as this has a significant impact on outcome assessment validity.

7. Was the condition measured in a standard, reliable way for all participants?

Considerable judgment is required to determine the presence of some health outcomes. Having established the validity of the outcome measurement instrument (see item 6 of this scale), it is important to establish how the measurement was conducted. Were those involved in collecting data trained or educated in the use of the instrument/s? If there was more than one data collector, were they similar in terms of level of education, clinical or research experience, or level of responsibility in the piece of research being appraised? When there was more than one observer or collector, was there comparison of results from across the observers? Was the condition measured in the same way for all participants?

8. Was there appropriate statistical analysis?

Importantly, the numerator and denominator should be clearly reported, and percentages should be given with confidence intervals. The methods section should be detailed enough for reviewers to identify the analytical technique used and how specific variables were measured. Additionally, it is also important to assess the appropriateness of the analytical strategy in terms of the assumptions associated with the approach as differing methods of analysis are based on differing assumptions about the data and how it will respond.

9. Was the response rate adequate, and if not, was the low response rate managed appropriately?

A large number of dropouts, refusals or “not founds” amongst selected subjects may diminish a study’s validity, as can a low response rates for survey studies. The authors should clearly discuss the response rate and any reasons for non-response and compare persons in the study to those not in the study, particularly with regards to their socio-demographic characteristics. If reasons for non-response appear to be unrelated to the outcome measured and the characteristics of non-responders are comparable to those who do respond in the study (addressed in question 5, coverage bias), the researchers may be able to justify a more modest response rate.

JBI CRITICAL APPRAISAL CHECKLIST FOR ANALYTICAL CROSS SECTIONAL STUDIES

	Yes	No	Unclear	Not applicable
1. Were the criteria for inclusion in the sample clearly defined?	☐	☐	☐	☐
2. Were the study subjects and the setting described in detail?	☐	☐	☐	☐
3. Was the exposure measured in a valid and reliable way?	☐	☐	☐	☐
4. Were objective, standard criteria used for measurement of the condition?	☐	☐	☐	☐
5. Were confounding factors identified?	☐	☐	☐	☐
6. Were strategies to deal with confounding factors stated?	☐	☐	☐	☐
7. Were the outcomes measured in a valid and reliable way?	☐	☐	☐	☐
8. Was appropriate statistical analysis used?	☐	☐	☐	☐

Overall appraisal: Include ☐ Exclude ☐ Seek further info ☐

Comments (Including reason for exclusion)

Explanation of analytical cross sectional studies critical appraisal

How to cite: Moola S, Munn Z, Tufanaru C, Aromataris E, Sears K, Sfetcu R, Currie M, Qureshi R, Mattis P, Lisy K, Mu P-F. Chapter 7: Systematic reviews of etiology and risk . In: Aromataris E, Munn Z (Editors). *JBI Manual for Evidence Synthesis*. JBI, 2020. Available from <https://synthesismanual.jbi.global>

Analytical cross sectional studies Critical Appraisal Tool

Answers: Yes, No, Unclear or Not/Applicable

1. Were the criteria for inclusion in the sample clearly defined?

The authors should provide clear inclusion and exclusion criteria that they developed prior to recruitment of the study participants. The inclusion/exclusion criteria should be specified (e.g., risk, stage of disease progression) with sufficient detail and all the necessary information critical to the study.

2. Were the study subjects and the setting described in detail?

The study sample should be described in sufficient detail so that other researchers can determine if it is comparable to the population of interest to them. The authors should provide a clear description of the population from which the study participants were selected or recruited, including demographics, location, and time period.

3. Was the exposure measured in a valid and reliable way?

The study should clearly describe the method of measurement of exposure. Assessing validity requires that a 'gold standard' is available to which the measure can be compared. The validity of exposure measurement usually relates to whether a current measure is appropriate or whether a measure of past exposure is needed.

Reliability refers to the processes included in an epidemiological study to check repeatability of measurements of the exposures. These usually include intra-observer reliability and inter-observer reliability.

4. Were objective, standard criteria used for measurement of the condition?

It is useful to determine if patients were included in the study based on either a specified diagnosis or definition. This is more likely to decrease the risk of bias. Characteristics are another useful approach to matching groups, and studies that did not use specified diagnostic methods or definitions should provide evidence on matching by key characteristics

5. Were confounding factors identified?

Confounding has occurred where the estimated intervention exposure effect is biased by the presence of some difference between the comparison groups (apart from the exposure investigated/of interest). Typical confounders include baseline characteristics, prognostic factors, or concomitant exposures (e.g. smoking). A confounder is a difference between the comparison groups and it influences the direction of the study results. A high quality study at the level of cohort design will identify the potential confounders and measure them (where possible). This is difficult for studies where behavioral, attitudinal or lifestyle factors may impact on the results.

6. Were strategies to deal with confounding factors stated?

Strategies to deal with effects of confounding factors may be dealt within the study design or in data analysis. By matching or stratifying sampling of participants, effects of confounding factors can be adjusted for. When dealing with adjustment in data analysis, assess the statistics used in the study. Most will be some form of multivariate regression analysis to account for the confounding factors measured.

7. Were the outcomes measured in a valid and reliable way?

Read the methods section of the paper. If for e.g. lung cancer is assessed based on existing definitions or diagnostic criteria, then the answer to this question is likely to be yes. If lung cancer is assessed using observer reported, or self-reported scales, the risk of over- or under-reporting is increased, and objectivity is compromised. Importantly, determine if the measurement tools used were validated instruments as this has a significant impact on outcome assessment validity.

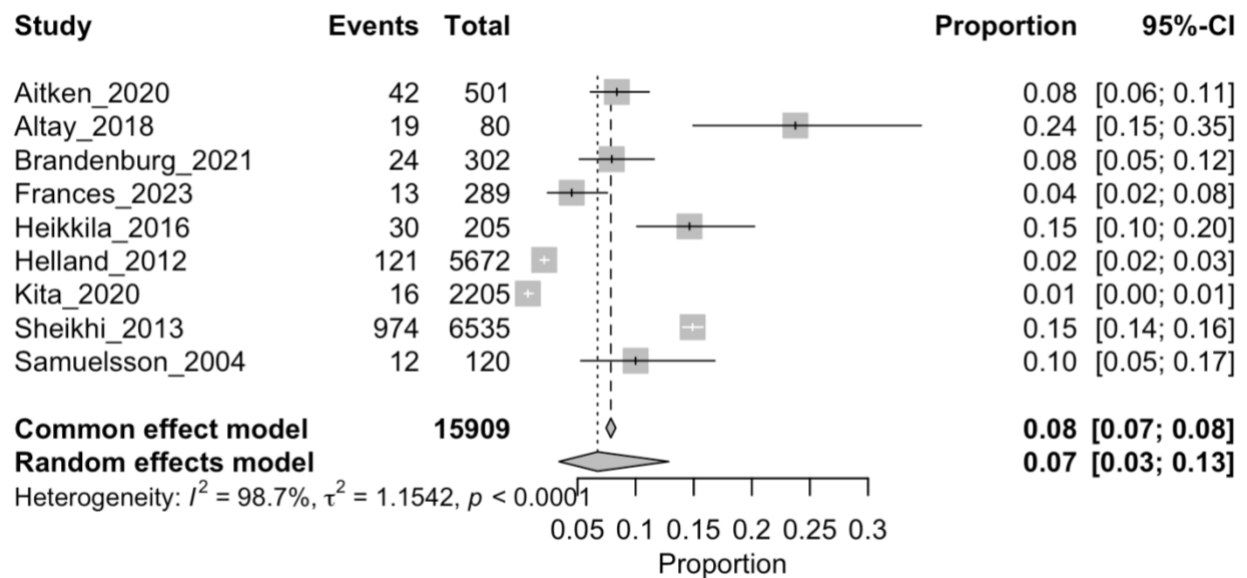
Having established the objectivity of the outcome measurement (e.g. lung cancer) instrument, it's important to establish how the measurement was conducted. Were those involved in collecting data trained or educated in the use of the instrument/s? (e.g. radiographers). If there was more than one data collector, were they similar in terms of level of education, clinical or research experience, or level of responsibility in the piece of research being appraised?

8. Was appropriate statistical analysis used?

As with any consideration of statistical analysis, consideration should be given to whether there was a more appropriate alternate statistical method that could have been used. The methods section should be detailed enough for reviewers to identify which analytical techniques were used (in particular, regression or stratification) and how specific confounders were measured. For studies utilizing regression analysis, it is useful to identify if the study identified which variables were included and how they related to the outcome. If stratification was the analytical approach used, were the strata of analysis defined by the specified variables? Additionally, it is also important to assess the appropriateness of the analytical strategy in terms of the assumptions associated with the approach as differing methods of analysis are based on differing assumptions about the data and how it will respond.

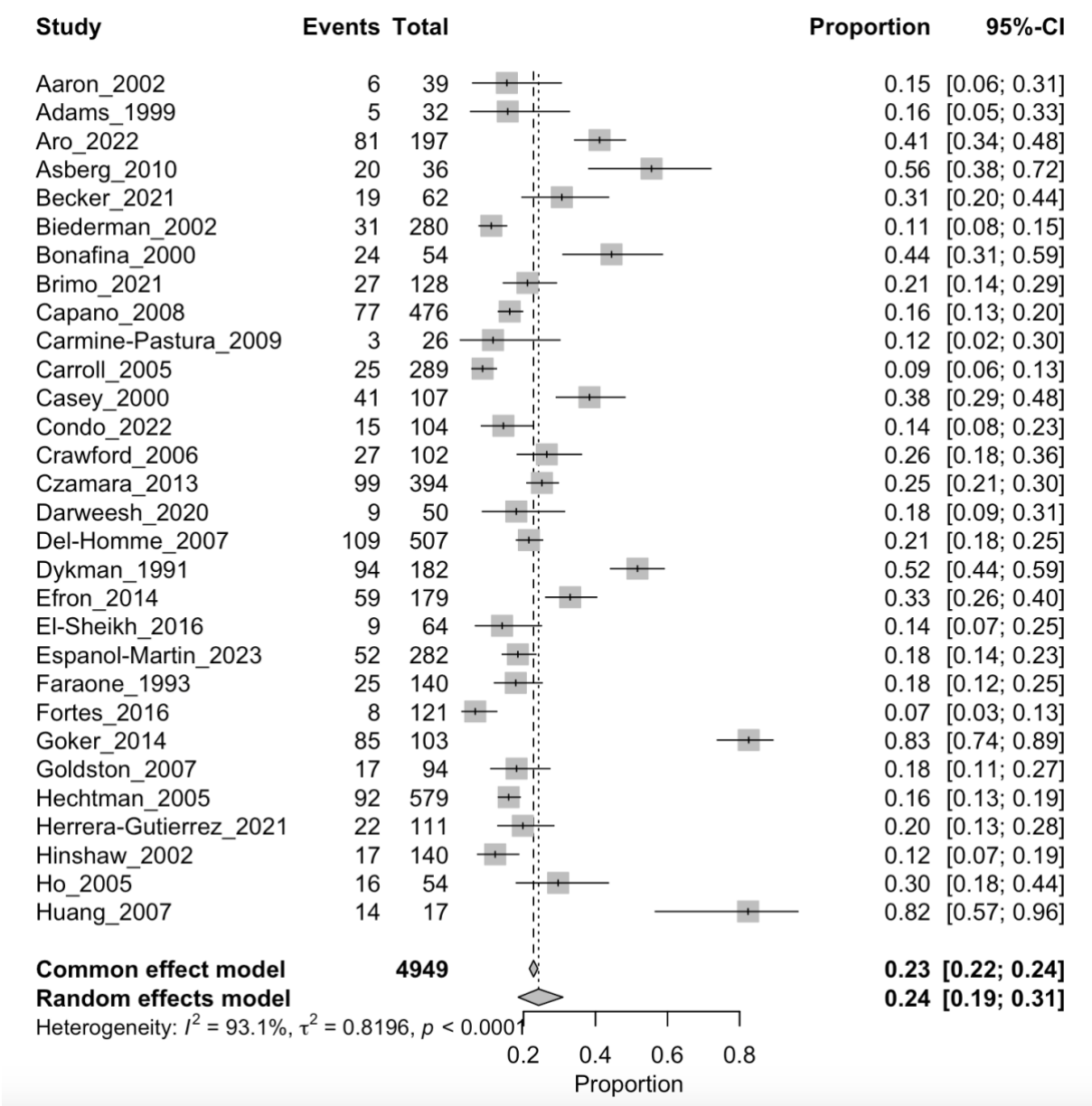
Appendix E. Forest plots displaying proportions of ADHD+RD and 95% confidence intervals, categorised by sampling method.

Figure 1. Mixed Sampling



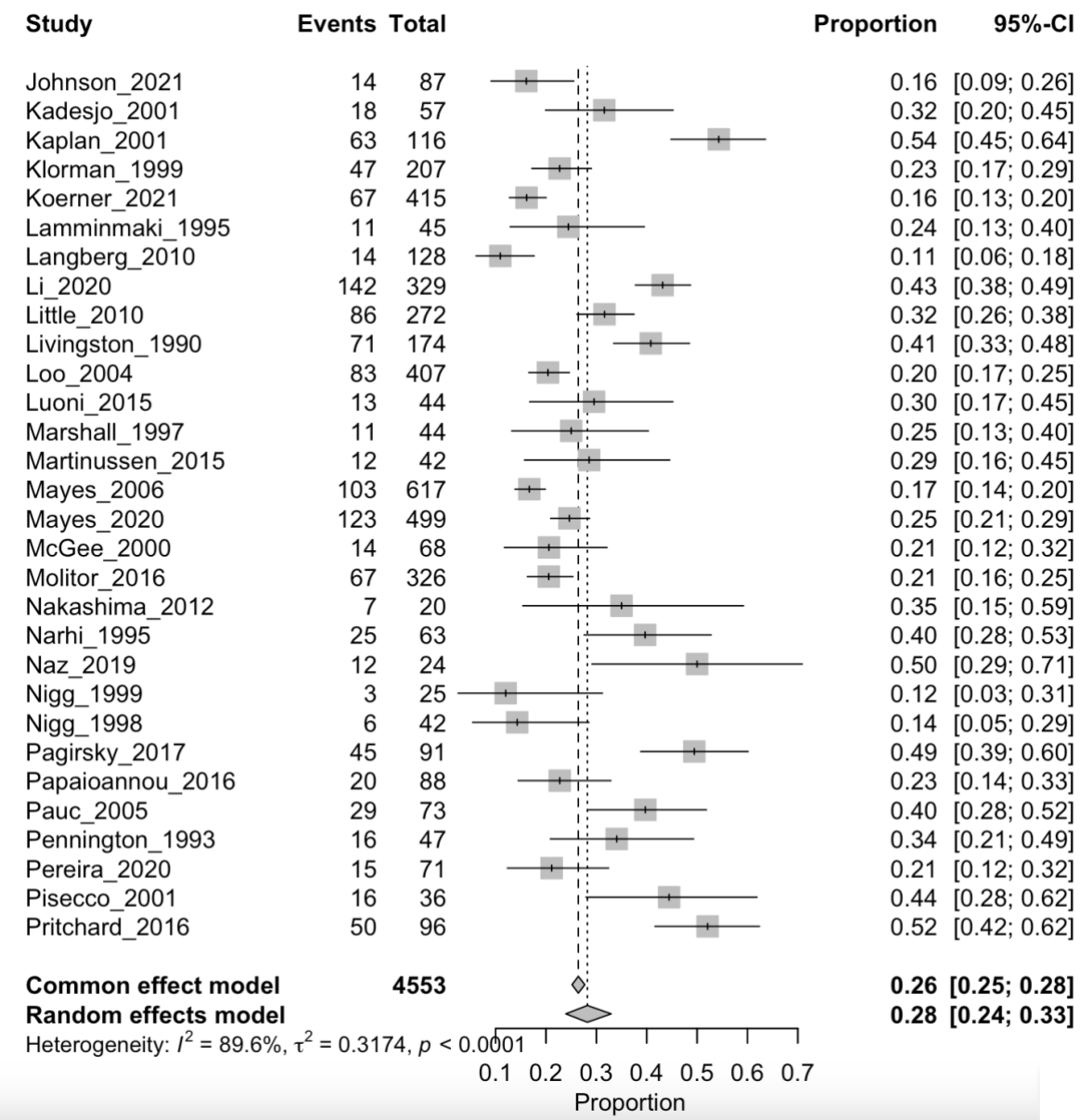
Note. ‘Events’ = number of individuals with ADHD+RD. ‘Total’ = sample from which the proportion of ADHD+RD was calculated.

Figure 2. Purposive Sampling



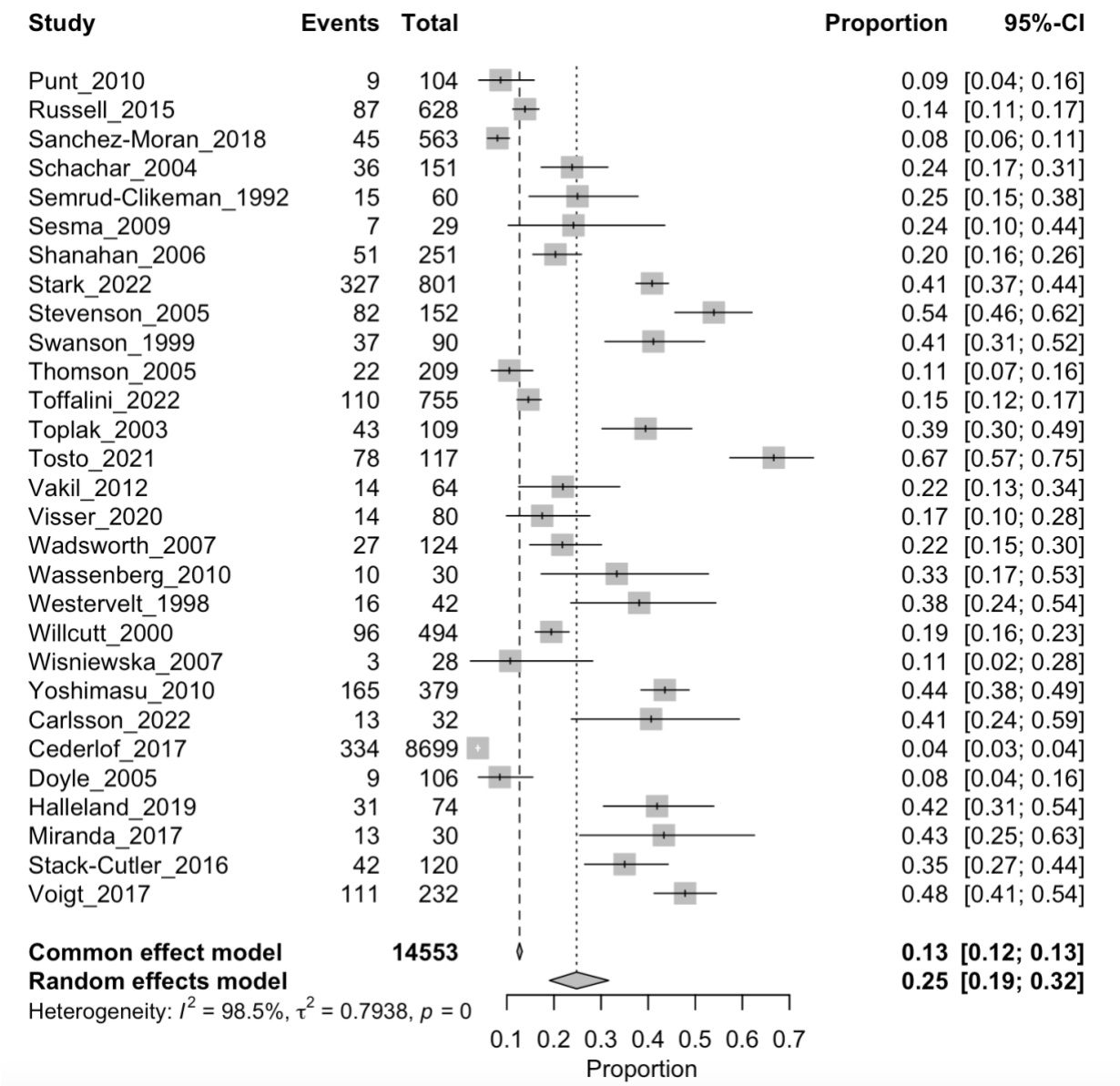
Note. ‘Events’ = number of individuals with ADHD+RD. ‘Total’ = sample from which the proportion of ADHD+RD was calculated.

Figure 2 Continued. Purposive Sampling



Note. ‘Events’ = number of individuals with ADHD+RD. ‘Total’ = sample from which the proportion of ADHD+RD was calculated.

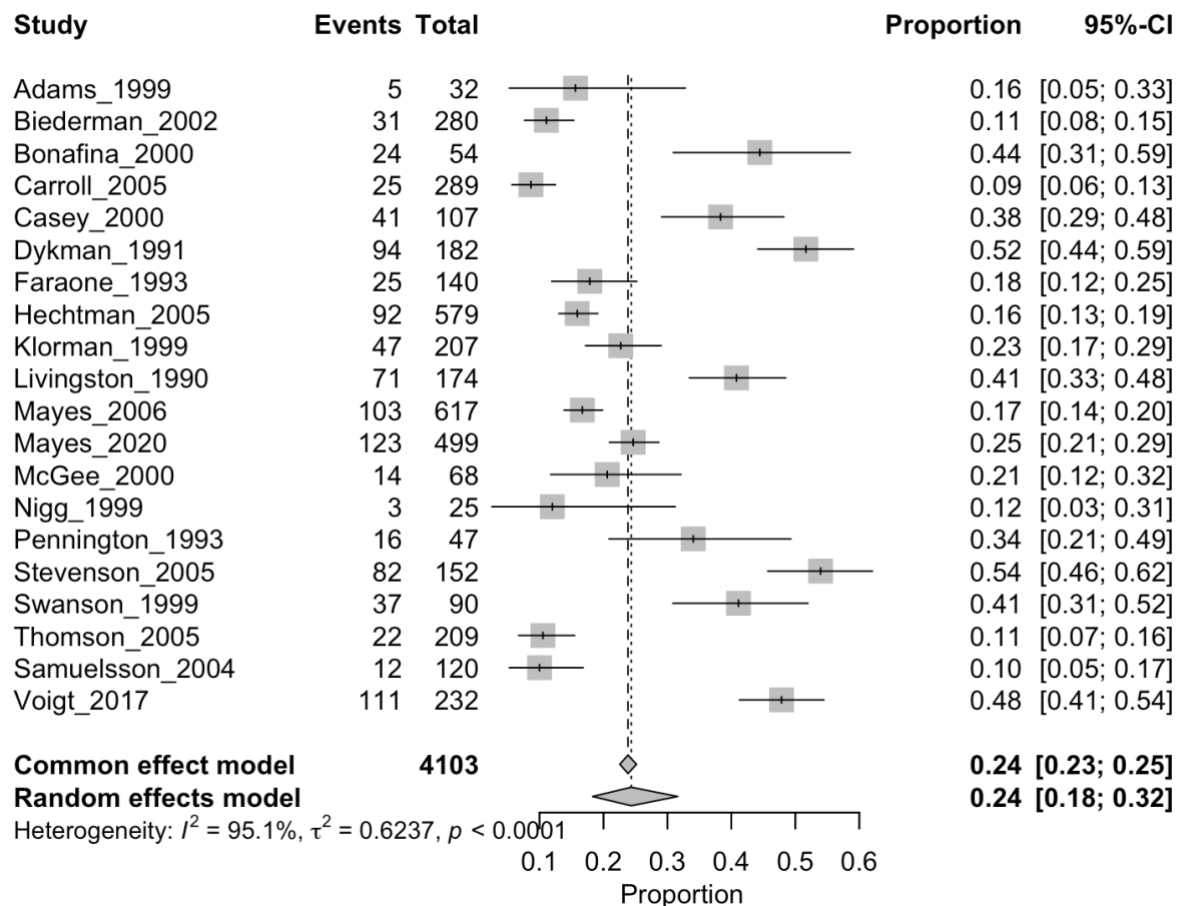
Figure 2 Continued. Purposive Sampling



Note. ‘Events’ = number of individuals with ADHD+RD. ‘Total’ = sample from which the proportion of ADHD+RD was calculated.

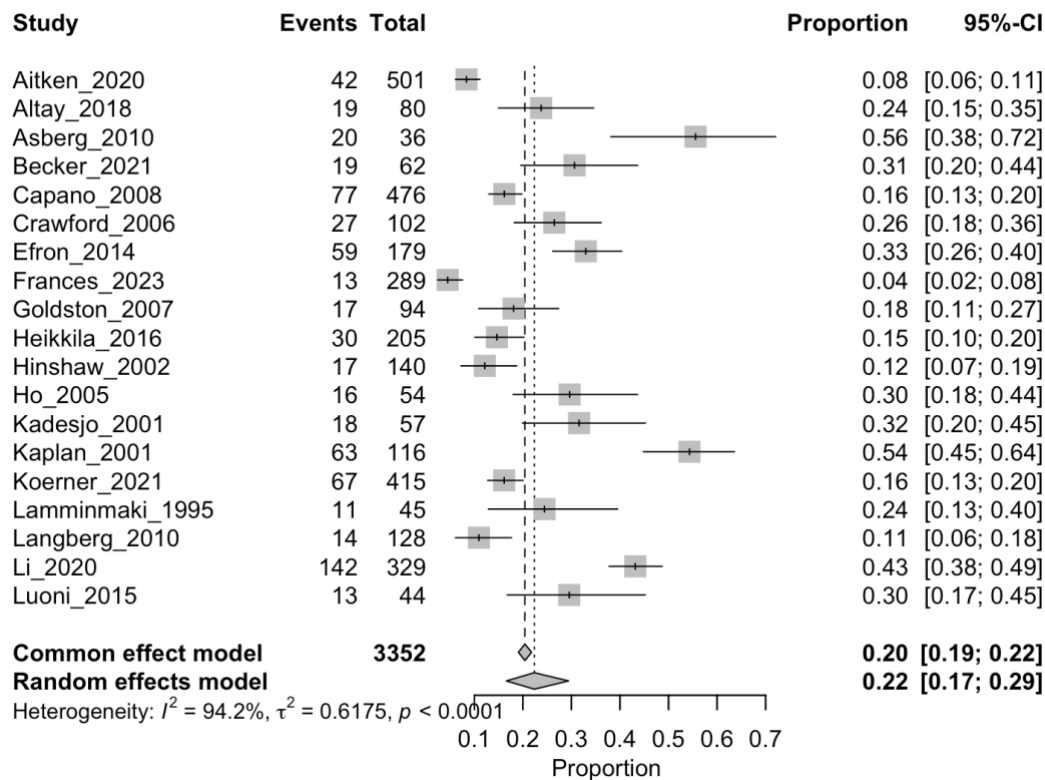
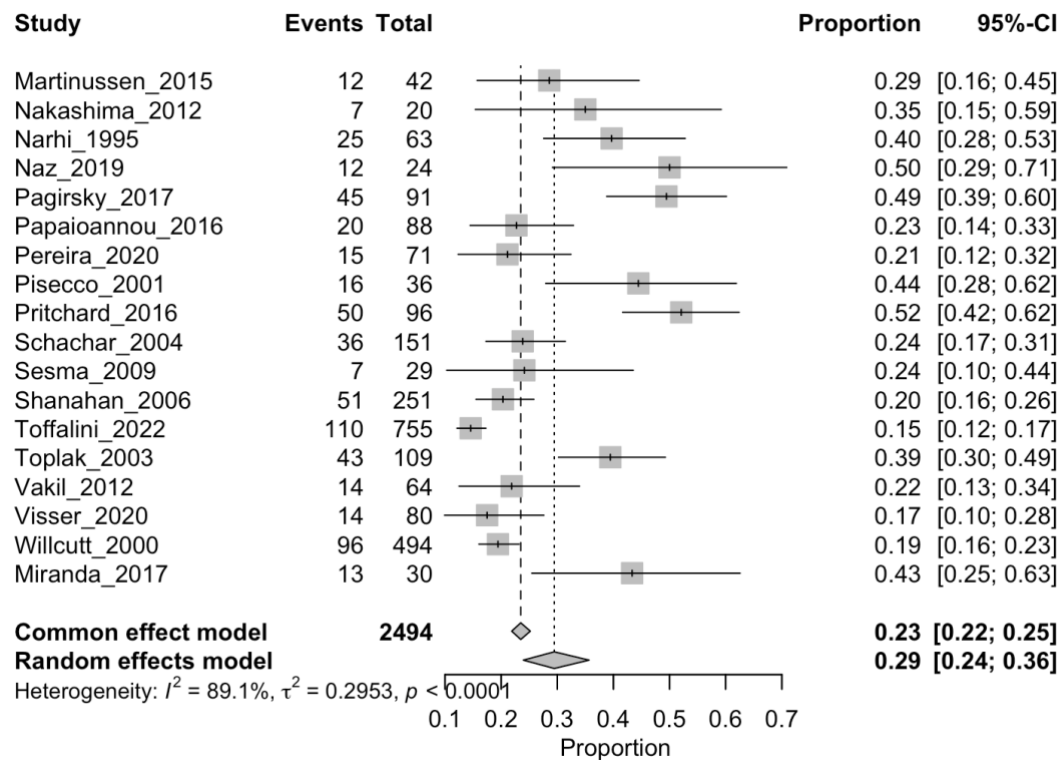
Appendix F. Forest plots displaying proportions of ADHD+RD and 95% confidence intervals, categorised by RD definition.

Figure 1. Discrepancy Model



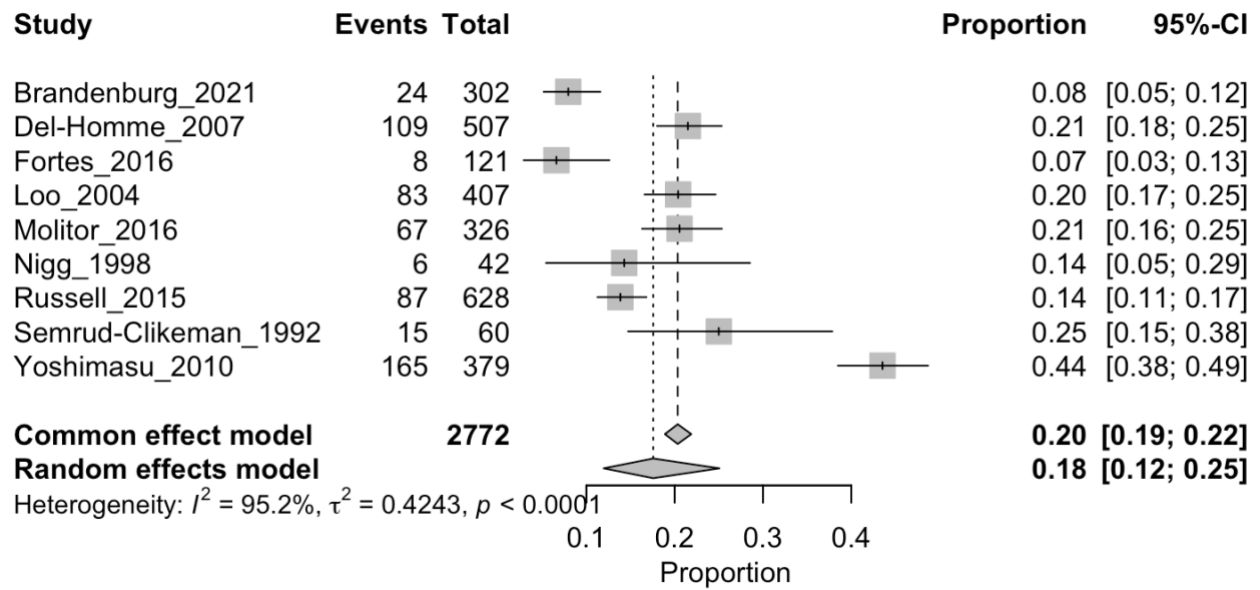
Note. ‘Events’ = number of individuals with ADHD+RD. ‘Total’ = sample from which the proportion of ADHD+RD was calculated.

Figure 2. Low Achievement Model



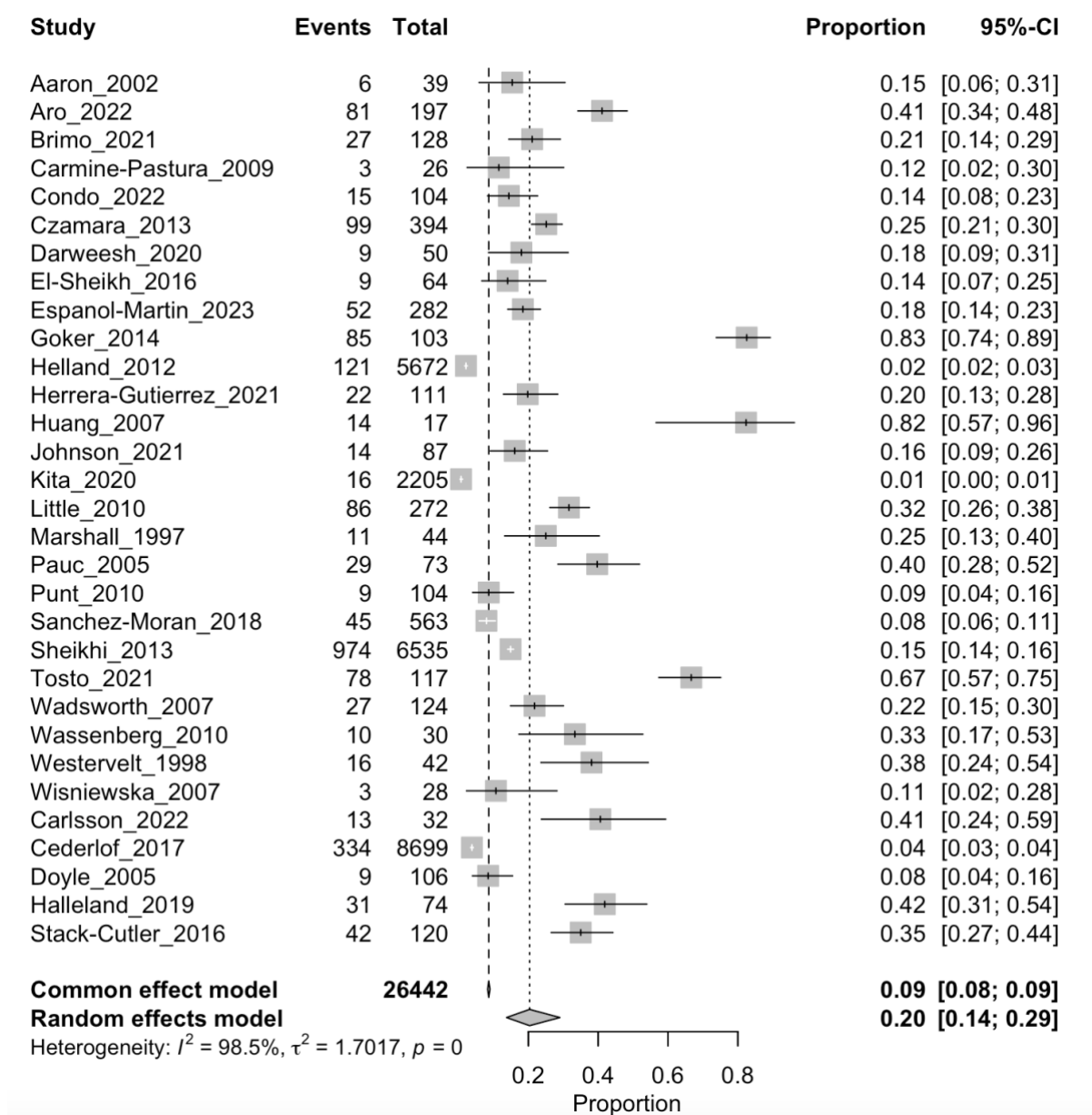
Note. ‘Events’ = number of individuals with ADHD+RD. ‘Total’ = sample from which the proportion of ADHD+RD was calculated.

Figure 3. Discrepancy and Low Achievement Model



Note. ‘Events’ = number of individuals with ADHD+RD. ‘Total’ = sample from which the proportion of ADHD+RD was calculated.

Figure 4. Unclear/Other Model



Note. ‘Events’ = number of individuals with ADHD+RD. ‘Total’ = sample from which the proportion of ADHD+RD was calculated.

Appendix G. Risk of Bias Assessment Justifications

Table 1. Detailed justifications for studies with unclear or high risk of bias domains (RQ1).

Study	Bias Domain	Rating	Justification
Aaron et al. (2002)	1. Generalisability 2. Sampling Methods 3. Sample Size 4. Statistical Analysis	1. Unclear 2. High 3. High 4. Unclear	1. Focused on children in grades 2-5 from a single school, limiting generalisability to broader population. 2. Participants selected by teachers based on perceived reading difficulty; sampling not random/systematic. 3. Sample size insufficient to address prevalence estimates adequately. 4. Prevalence rates established from the number of children who met criteria for both ADHD+RD in the given sample – this is not an epidemiological study.
Adams et al. (1999)	1. Generalisability 2. Sampling Methods 3. Sample Size 4. Diagnostic Assessment	1. High 2. High 3. Unclear 4. Unclear	1. Data collected from children in grades 3-6 in a single school, limits generalisability to wider population. 2. Did not specify how children were selected. 3. Sample size insufficient to address prevalence estimates adequately. 4. Identification of conditions relied on subjective reports by different teachers; inter-rater bias comparisons not addressed.
Aitken et al. (2020)	1. Generalisability 2. Sampling Methods 3. Sample Size 4. Sample Coverage 5. Diagnostic Assessment 6. Statistical Analysis	1. Unclear 2. Unclear 3. Unclear 4. Unclear 5. Unclear 6. Unclear	1. Data collected from several schools within a specific region. 2. Specific sampling procedures not described in detail. 3. Large sample size, but formal sample size calculation not reported. 4. It is reported that several participants were missing parent ratings. 5. Identification of conditions relied on subjective parent/teacher ratings. 6. Prevalence rates established using latent classes of ADHD+RD; may not be suitable for determining prevalence rates.
Altay et al. (2018)	1. Generalisability 2. Sampling Methods 3. Sample Size	1. Unclear 2. High 3. High	1. Data are based on students who were referred to a single clinic. 2. Participants were volunteers – sampling not random/systematic. 3. Sample size calculations not reported. Sample size insufficient to address prevalence estimates adequately.
Asberg et al. (2010)	1. Generalisability 2. Sampling Methods 3. Sample Size	1. High 2. High 3. High	1. Sample restricted to female participants. 2. Convenience sample. 3. Sample size insufficient to address prevalence estimates adequately.
Becker et al. (2021)	1. Generalisability 2. Sampling Methods 3. Sample Size	1. Unclear 2. Unclear 3. High	1. Sample recruited from a single counselling centre. 2. Sampling method unclear, possibly a convenience sample.

Study	Bias Domain	Rating	Justification
Biederman et al. (2002)	1. Sampling Methods 2. Sample Size	1. Unclear 2. Unclear	3. No sample size calculation. Sample size insufficient to address prevalence estimates adequately. 1. Participants selected based on consecutive referrals or being existing patients at two different clinics; exact sampling method not fully clarified. 2. No sample size calculation. Sample size insufficient to address prevalence estimates adequately.
Bonafina et al. (2000)	1. Generalisability 2. Sampling Methods 3. Sample Size 4. Statistical Analysis	1. High 2. High 3. High 4. Unclear	1. Majority of participants were male, and age range is limited. 2. Children clinically referred to research program, not an ideal recruitment method for determining prevalence. 3. Sample size insufficient to address prevalence estimates adequately. 4. Statistical analyses may not be suitable for determining prevalence rates. Prevalence established from number of participants with ADHD in 'poor reading' cluster.
Brandenburg et al. (2021)	1. Generalisability 2. Sampling Methods 3. Sample Size 4. Diagnostic Assessment 5. Statistical Analysis	1. Unclear 2. Unclear 3. High 4. Unclear 5. Unclear	1. Limited age range. 2. Participants recruited via screening of academic skills; those who met criteria were invited. This suggests convenience sampling, but details are unclear. 3. No sample size calculation. Sample size insufficient to address prevalence estimates adequately. 4. Unclear who administered diagnostic assessments and if this was consistent across all participants. 5. Statistical analyses may not be suitable for determining prevalence rates. Prevalence rates calculated from the number of participants with RD in the profile with a single deficit in visual attention.
Brimo et al. (2021)	1. Demographic Information	1. Unclear	1. The study does not report enough demographic information (e.g., exact age range has not been reported).
Capano et al. (2008)	1. Generalisability 2. Sampling Methods 3. Sample Size	1. Unclear 2. High 3. Unclear	1. The sample was drawn from consecutive referrals to a single clinic. 2. Convenience sample; participants were included based on specific criteria – sampling not randomised. 3. No sample size calculation. Sample size relatively large, but may not be sufficient enough to address prevalence rates in the wider population.
Carlsson et al. (2022)	1. Generalisability 2. Sampling Methods 3. Sample Size	1. High 2. High 3. High	1. Participants responded to advertisements in local newspaper, may not represent the target population well. 2. Opportunity sampling used, introducing potential selection bias.

Study	Bias Domain	Rating	Justification
	4. Statistical Analysis	4. Unclear	3. No sample size calculation. Sample size insufficient to address prevalence estimates adequately. 4. Statistical analyses may not be suitable for determining prevalence rates. Prevalence rates calculated from the number of participants with attentional difficulties who reported having reading disabilities. This is not an epidemiological study.
Carmine Pastura et al. (2009)	1. Generalisability 2. Sampling Methods 3. Sample Size 4. Sample Coverage 5. Statistical Analysis	1. Unclear 2. High 3. Unclear 4. Unclear 5. Unclear	1. Participants recruited from a single elementary school. 2. Convenience sampling used, introducing potential selection bias. 3. No sample size calculation. Sample size may be insufficient to address prevalence rates adequately. 4. Around 20% of parents did not provide consent for children to participate – analyses did not fully cover the identified sample. 5. Statistical analysis not described in enough detail, although percentages for RD occurrence in ADHD are reported.
Carroll et al. (2005)	1. Sample Coverage	1. Unclear	1. Report of refusals, missing information and failure to make contact. The analyses only included around 50% of the identified sample.
Casey et al. (2000)	1. Generalisability 2. Sampling Methods 3. Sample Size 4. Demographic Information 5. Statistical Analysis	1. Unclear 2. High 3. High 4. Unclear 5. Unclear	1. The study is based on children specifically selected from a larger study. All participants came from the same research centre. 2. Convenience sample used, introducing potential selection bias. 3. No sample size calculation. Sample size insufficient to address prevalence estimates adequately. 4. Limited information reported about the actual participants – the focus of the current study was on their parents, but prevalence rates are reported for the children with ADHD diagnoses. 5. Prevalence rates were not the primary outcome of this study; although percentages for ADHD+RD are reported, they are not directly assessed. May not be appropriate for addressing overall prevalence rates.
Cederlof et al. (2017)	1. Sample Coverage 2. Response Rates	1. Unclear 2. Unclear	1. Coverage percentage reported, but it is unclear whether this coverage is satisfactory; some tests were incomplete, but reasons are not provided. 2. Participation rate is reported, but there is no description or comparison between individuals who did and did not complete the academic test.
Condo et al. (2022)	1. Sampling Methods 2. Sample Size 3. Diagnostic Assessment	1. Unclear 2. High 3. Unclear	1. Convenience sample, introducing potential selection bias. 2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.

Study	Bias Domain	Rating	Justification
Crawford et al. (2006)	4. Response Rate	4. Unclear	3. Assessments for identifying ADHD and RD are not clearly reported. It is unclear who conducted diagnostic assessments.
	1. Sample Size	1. Unclear	4. Participation/response rate is not discussed.
Czamara et al. (2013)	2. Statistical Analysis	2. Unclear	1. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
			2. Statistical analysis not described in enough detail. Prevalence rates established from number of children meeting criteria for ADHD and RD. Analyses may not be appropriate to reliably estimate prevalence.
Darweesh et al. (2020)	1. Sample Coverage	1. Unclear	1. The final sample size included in the analyses is smaller than the identified sample, but this is not addressed in the paper.
	2. Diagnostic Assessment	2. Unclear	2. The presence of RD was not identified using standardised methods.
Del'Homme et al. (2007)	3. Response Rate	3. Unclear	3. Reasons for non-responses are not discussed.
			1. Sample seems to represent the target population well, but the sampling frame is not described in enough detail.
Doyle et al. (2005)	1. Generalisability	1. Unclear	2. Sampling methods not reported in enough detail.
	2. Sampling Methods	2. Unclear	3. No sample size calculation. Sample size is insufficient to address prevalence estimates adequately.
Dykman et al. (1991)	3. Sample Size	3. High	1. No sample size calculation, although sample is quite large.
			2. Some participants were excluded from the final analyses due to incomplete data.
			3. Response rate seems adequate, but missing data not addressed properly.
			1. Sample frame not described in enough detail.
			2. Convenience sample, introducing potential selection bias.
			3. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
			1. Participants recruited from a single clinic.
			2. Convenience sample, introducing potential selection bias.
			3. No sample size calculation. Sample size is insufficient to address prevalence estimates adequately.
			4. Unclear who administered diagnostic assessments.
			5. Statistical analyses not reported in detail. Analyses may not be appropriate to address prevalence rates adequately.

Study	Bias Domain	Rating	Justification
Efron et al. (2014)	1. Sample Coverage	1. High	1. Analyses did not include participants from disadvantaged areas as they were more likely to have missing data. Final analyses also did not include all participants with ADHD as consent was not given.
El Sheikh et al. (2016)	1. Diagnostic Assessment	1. Unclear	1. Inter-rater reliability was not reported.
Espanol-Martin et al. (2023)	1. Response Rate	1. Unclear	1. Response rate is reported, but reasons for missing data and lack of consent are not discussed.
Faraone et al. (1993)	1. Generalisability 2. Sampling Methods 3. Sample Size 4. Sample Coverage 5. Response Rate	1. High 2. High 3. High 4. Unclear 5. Unclear	1. Sample is restricted to males of higher SES recruited from two sources. 2. Convenience sample, introducing potential selection bias. 3. No sample size calculation. Sample size is insufficient to address prevalence estimates adequately. 4. Not all identified participants were included in the study. Some were ineligible and some refused to participate. 5. Rates of unreachable participants and refusals are reported, but reasons are not discussed.
Fortes et al. (2016)	1. Sample Coverage 2. Response Rate	1. Unclear 2. Unclear	1. There was some loss of participants, so it is unclear whether there was sufficient coverage of the identified sample. 2. Response rate was reported, but those who did not participate are not described in detail (in terms of participant characteristics).
Frances et al. (2023)	1. Sampling Methods 2. Response Rate	1. Unclear 2. Unclear	1. Consecutive sampling was used, which introduces potential selection bias. 2. Response rate and reasons reported, but those who did not participate or had missing data are not described in detail (in terms of participant characteristics).
Goker et al. (2014)	1. Sampling Methods 2. Sample Coverage 3. Diagnostic Assessment 4. Statistical Analysis 5. Response Rate	1. Unclear 2. Unclear 3. Unclear 4. Unclear 5. Unclear	1. Convenience sample used, introduces potential selection bias. 2. Adolescents were not covered by the analyses, but it is unclear whether this was due to low response rate or because the majority of participants admitted to the recruitment source were children. 3. Diagnostic assessment is not described in detail. Also unclear who confirmed diagnoses. 4. Statistical analyses may not be appropriate to address prevalence rates, as these were simply calculated from the number of participants who had both ADHD and RD (this is not an epidemiological study). 5. Refusals and response rates are not discussed.

Study	Bias Domain	Rating	Justification
Goldston et al. (2007)	1. Sampling Methods 2. Sample Size	1. Unclear 2. Unclear	1. Convenience sampling used, introduces potential selection bias. 2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
Halleland et al. (2019)	1. Sample Size 2. Statistical Analysis	1. Unclear 2. Unclear	1. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately. 2. Statistical analysis is not described in detail, although percentages are reported for co-occurring RD and ADHD.
Hechtman et al. (2005)	1. Generalisability 2. Sampling Methods 3. Sample Size 4. Demographic Information 5. Diagnostic Assessment 6. Response Rate	1. Unclear 2. Unclear 3. Unclear 4. Unclear 5. Unclear 6. Unclear	1. Sample frame not described in detail (signposts to another publication). 2. Unclear whether sampling was randomised. 3. Sample size is large, but no sample size calculation provided. 4. Some demographic information is missing (likely in previous publications using the same sample). 5. Unclear who administered diagnostic assessments. 6. States that anybody who would not be able to complete the full study was excluded, but response rates are not discussed further.
Heikkila et al. (2016)	1. Generalisability 2. Sampling Methods 3. Sample Size 4. Diagnostic Assessment 5. Statistical Analysis 6. Response Rate	1. Unclear 2. Unclear 3. Unclear 4. Unclear 5. Unclear 6. Unclear	1. Data drawn from a single clinic. 2. Convenience sample used, introduces potential selection bias. 3. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately. 4. Unclear who administered diagnostic assessments. 5. Statistical analyses may not appropriately address prevalence. Prevalence rates were calculated using the numbers reported for different combinations of co-occurring conditions (from the sample characterisation section). This is not an epidemiological study. 6. Response rates not discussed.
Helland et al. (2012)	1. Diagnostic Assessment 2. Statistical Analysis	1. Unclear 2. Unclear	1. RD was identified using a measure which was not validated. Parents and teachers provided subjective reports for diagnostic screening. It is unclear whether this was further confirmed by a professional. 2. Statistical analyses may not be appropriate for addressing prevalence. The number of children with ADHD+RD was reported for the purposes of sample characterisation (this is not an epidemiological study).
Herrera-Gutierrez et al. (2021)	1. Sampling Methods 2. Sample Size 3. Sample Coverage	1. Unclear 2. Unclear 3. Unclear	1. Unclear whether the sampling was random or convenience. 2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.

Study	Bias Domain	Rating	Justification
	4. Diagnostic Assessment 5. Statistical Analysis 6. Response Rate	4. Unclear 5. Unclear 6. Unclear	3. It is reported that the initial sample size was reduced, making it unclear whether the analyses sufficiently covered the identified sample. 4. Unclear how ADHD and RD were identified/diagnosed. 5. Statistical analyses are not described in enough detail and may not be appropriate for addressing prevalence rates. Percentages reported for the co-occurrence of ADHD and RD, but just for the purposes of sample characterisation (this is not an epidemiological study). 6. States that the sample size was reduced, but reasons for this are not reported.
Hinshaw (2002)	1. Sampling Methods 2. Sample Size 3. Response Rate	1. Unclear 2. Unclear 3. Unclear	1. Convenience sample used, introducing potential selection bias. 2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately. 3. Response rate reported and reasons given; however, those who declined participation are not further described (in terms of demographics).
Ho et al. (2005)	1. Generalisability 2. Sampling Methods 3. Sample Size 4. Diagnostic Assessment 5. Statistical Analysis 6. Response Rate	1. Unclear 2. Unclear 3. Unclear 4. Unclear 5. Unclear 6. Unclear	1. Small age range and all participants were recruited from one location. 2. Convenience sample used, introducing potential selection bias. 3. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately. 4. Unclear who administered diagnostic assessments/confirmed diagnoses. 5. Prevalence rates are reported, but it is stated that co-occurrence rates may be questionable. Analyses may not be appropriate to address prevalence. 6. Response rates not discussed.
Huang et al. (2007)	1. Sampling Methods 2. Sample Size 3. Sample Coverage 4. Diagnostic Assessment 5. Statistical Analysis 6. Response Rate	1. Unclear 2. Unclear 3. Unclear 4. Unclear 5. Unclear 6. Unclear	1. Convenience sampling used, introducing potential selection bias. 2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately. 3. Reports that participants with incomplete data were excluded from analyses, but this is not discussed further. 4. It is suggested that standardised diagnostic assessments were used, but it is not clearly reported who conducted these assessments. 5. Statistical analyses not described in enough detail. Percentages are reported for co-occurring ADHD and RD, but analyses are likely not appropriate for addressing prevalence rates adequately due to small sample size.

Study	Bias Domain	Rating	Justification
Johnson et al. (2021)	1. Sampling Methods	1. Unclear	6. It is reported that some participants did not complete assessments, but this is not discussed further.
	2. Sample Size	2. Unclear	1. Convenience sampling used, introducing potential selection bias.
	3. Diagnostic Assessment	3. Unclear	2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
	4. Statistical Analysis	4. Unclear	3. It is not reported how RD was identified.
	5. Response Rate	5. Unclear	4. Prevalence percentage of co-occurring ADHD+RD reported just for the purposes of baseline participant characteristics. May not be appropriate for addressing prevalence rates adequately as this is not an epidemiological study.
Kadesjo et al. (2001) Kaplan et al. (2001)	1. Sample Size	1. Unclear	5. Response rate not discussed. 1. Sample size seems large enough to address prevalence rates, but no sample size calculation provided.
	1. Sample Size	1. Unclear	1. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
	2. Sample Coverage	2. Unclear	2. States that a lot of participants were not included in the study; unclear whether the final analyses sufficiently cover the identified sample.
	3. Statistical Analysis	3. Unclear	3. Unclear whether the statistical analyses appropriately address prevalence rates; the number of children with ADHD+RD (plus other conditions) are reported, but not percentages. This is not an epidemiological study.
Kita et al. (2020)	1. Sampling Methods	1. Unclear	1. Convenience sample used, introduces potential selection bias.
	2. Sample Size	2. Unclear	2. No sample size calculation, although sample seems large enough to address prevalence.
	3. Demographic Information	3. Unclear	3. Some demographic information not described (e.g., gender).
	4. Sample Coverage	4. Unclear	4. The final analyses did not cover the whole identified sample.
	5. Response Rate	5. Unclear	5. Response rates reported with reasons, but non-respondents are not further described (in terms of demographics).
Klorman et al. (1999)	1. Sampling Methods	1. Unclear	1. Convenience sampling used, introduces potential selection bias.
	2. Sample Size	2. Unclear	2. Sample is quite large, but there is no sample size calculation.
	3. Diagnostic Assessment	3. Unclear	3. Unclear who administered diagnostic assessments.
	4. Statistical Analysis	4. Unclear	4. Statistical analyses may not be appropriate for addressing prevalence rates adequately. Percentages are not reported, prevalence rates calculated using the number of children with RD in each ADHD subtype.
	5. Response Rate	5. Unclear	5. Response rates not discussed.

Study	Bias Domain	Rating	Justification
Koerner et al. (2021)	- Sample Coverage - Diagnostic Assessment - Response Rate	- Unclear - Unclear - Unclear	- Reports that cases with incomplete data were excluded, and over half of the initial sample was included in the final analyses. However, it is not clear whether specific subgroups were sufficiently covered. - Diagnostic assessments were administered online, posing some reliability questions. - Reports the number of participants with missing data and those who quit, but they are not described in any more detail (i.e., demographics).
Lamminmaki et al. (1995)	- Generalisability - Sampling Methods - Sample Size - Sample Coverage - Diagnostic Assessment - Response Rate	- Unclear - High - Unclear - Unclear - Unclear - Unclear	- All participants were recruited from a single remediation centre. - Convenience sample used, introduces potential selection bias. - No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately. - Reports that participants who could not be categorised into a specific diagnostic group were excluded, so it is unclear whether the analyses sufficiently covered the identified sample. - Unclear who conducted academic assessments to identify RD. - Some participants were excluded from the study because they did not meet diagnostic criteria (these participants were not demographically compared to those who did meet the criteria).
Langberg et al. (2010)	- Sampling Methods - Sample Size - Diagnostic Assessment - Response Rate	- Unclear - Unclear - Unclear - Unclear	- Convenience sample used, introduces potential selection bias. - No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately. - It is not reported who administered assessments to confirm RD. - Reports that children with low IQ were excluded, but excluded children are not further compared to those who were included in the study (in terms of sociodemographic variables).
Li et al. (2020)	- Sampling Methods - Diagnostic Assessment - Statistical Analysis - Response Rate	- Unclear - Unclear - Unclear - Unclear	- Sampling method is unclear. - Unclear who administered diagnostic assessments. - Information about the prevalence of ADHD+RD is based on a latent class analysis (i.e., the class that matched the profile of the co-occurring conditions). The analysis may not be appropriate for addressing prevalence rates as this is not an epidemiological study. - Response rate not discussed.
Little et al. (2010)	- Sample Size - Diagnostic Assessment	- Unclear - Unclear	No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.

Study	Bias Domain	Rating	Justification
Livingston et al. (1990)	1. Generalisability	1. Unclear	2. RD was not diagnosed using a standardised assessment. Subjective teacher reports were used to identify RD; unclear if this was further confirmed or cross-compared.
	2. Sampling Methods	2. High	1. Reported data are restricted to white males.
	3. Sample Size	3. Unclear	2. Convenience sampling used, introduces selection bias.
	4. Sample Coverage	4. High	3. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
	5. Response Rate	5. Unclear	4. The analyses did not cover children of other ethnicities; these were explicitly excluded.
Loo et al. (2004)			5. There were many exclusions based on race and gender. Lack of representation was not managed appropriately. Some participants had missing data, but this is not discussed further.
	1. Sampling Methods	1. High	1. Convenience/opportunity sampling used, introduces selection bias.
	2. Sample Size	2. Unclear	2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
	3. Sample Coverage	3. Unclear	3. Reports that data were unavailable for some participants, but it is unclear which participants were not included in the final analysis.
	4. Diagnostic Assessment	4. Unclear	4. Unclear how diagnostic assessments were administered.
	5. Statistical Analysis	5. Unclear	5. Statistical analyses may not be appropriate to address prevalence rates. There is no specific percentage for the co-occurrence of ADHD+RD; this was established from numbers reported.
Luoni et al. (2015)	6. Response Rate	6. Unclear	6. Response rate is reported and discussed, but those who did not participate are not described in any further detail.
	1. Generalisability	1. Unclear	1. Limited age range (all participants were in their first year of primary school). All participants from the same location.
	2. Sampling Methods	2. Unclear	2. Sampling method unclear.
	3. Sample Size	3. Unclear	3. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
Marshall et al. (1997)	4. Sample Coverage	4. Unclear	4. Unclear whether there is a specific group that was not covered in the analyses; children without consent and those with psychiatric disorders were not included, but this is not explained further.
	1. Generalisability	1. Unclear	1. All participants recruited from a single clinic.
	2. Sampling Methods	2. High	2. Convenience sample used, introduces selection bias.
	3. Sample Size	3. Unclear	

Study	Bias Domain	Rating	Justification
	4. Sample Coverage 5. Response Rate	4. Unclear 5. Unclear	3. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately. 4. Some participants had missing data and were excluded from the final analyses. Unclear whether the analyses sufficiently covered the identified sample. 5. Response rate reported (i.e., missing data); however, this is not discussed further.
Martinussen et al. (2015)	1. Sampling Methods 2. Sample Size 3. Statistical Analysis	1. Unclear 2. Unclear 3. Unclear	1. Convenience sample used, introduces selection bias. 2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately. 3. Statistical analyses may not be appropriate to address prevalence. Prevalence rates were established from the number of participants with ADHD who were considered poor readers; this number was reported for the purposes of grouping as this is not a prevalence study.
Mayes et al. (2006)	1. Sampling Methods 2. Response Rate	1. Unclear 2. Unclear	1. Sampling method is unclear (possibly opportunity sampling). 2. Response rate/missing data not discussed.
Mayes et al. (2020)	1. Generalisability 2. Sampling Methods 3. Sample Size 4. Response Rate	1. Unclear 2. High 3. Unclear 4. Unclear	1. Participants recruited from a single clinic. 2. Convenience sample used, introduces selection bias. 3. Sample size seems large enough, but there is no sample size calculation. 4. Response rate/missing data not discussed.
McGee et al. (2000)	1. Generalisability 2. Sampling Methods 3. Sample Size 4. Statistical Analysis 5. Response Rate	1. Unclear 2. High 3. Unclear 4. Unclear 5. Unclear	1. Participants recruited from a single clinic. 2. Convenience sample used, introduces selection bias. 3. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately. 4. Statistical analyses may not adequately address prevalence. Prevalence rates established from the percentage of participants with ADHD+RD reported for the purposes of study grouping, as opposed to actual prevalence rates. This is not an epidemiological study. 5. Reports that there were some missing data (e.g., refusals and incomplete reports), but these participants are not described further.
Miranda et al. (2017)	1. Generalisability 2. Sampling Methods 3. Sample Size 4. Statistical Analysis	1. Unclear 2. Unclear 3. High 4. Unclear	1. Sample consists of males only. 2. Sampling method unclear (follow-up from a genetic study). 3. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.

Study	Bias Domain	Rating	Justification
	5. Response Rate	5. Unclear	4. Prevalence rates were established from the number of adults with ADHD+RD reported for the purposes of study grouping. May not appropriately address prevalence rates. 5. Response rates/refusals not discussed.
Molitor et al. (2016)	1. Sampling Methods 2. Sample Size 3. Response Rate	1. Unclear 2. Unclear 3. Unclear	1. Sampling method unclear. 2. Sample size seems large enough, but there is no sample size calculation. 3. The report suggests there may be some missing data, but this is not discussed further.
Nakashima et al. (2012)	1. Generalisability 2. Sampling Methods 3. Sample Size 4. Diagnostic Assessment 5. Statistical Analysis 6. Response Rate	1. High 2. High 3. High 4. Unclear 5. Unclear 6. Unclear	1. Participants recruited from a single clinic in a specific location. 2. Convenience sample used, introduces selection bias. 3. Very small sample. No sample size calculation. 4. Diagnostic assessments were not specified in enough detail (unclear whether these were standardised). 5. Percentage of ADHD+RD is reported, but this is not an epidemiological study, so may not be appropriate for estimating prevalence rates (especially due to small sample size). 6. Participation rate not discussed.
Narhi et al. (1995)	1. Generalisability 2. Sampling Methods 3. Sample Size 4. Sample Coverage 5. Statistical Analysis 6. Response Rate	1. High 2. High 3. High 4. Unclear 5. Unclear 6. Unclear	1. Sample consists of middle-class males from a single clinic. 2. Convenience sample used, introduces selection bias. 3. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately. 4. Unclear whether analyses sufficiently covered the identified sample as only a proportion of children were included due to data availability in the areas of interest. Those with unavailable data were not further discussed. 5. Prevalence rates were established from demographic information (i.e., how many participants with ADHD met criteria for RD). This is not an epidemiological study, so may not be adequate for estimating prevalence rates. 6. Reports that there were some missing data, but the reason for this is not discussed further, so response rate is unclear.
Naz et al. (2019)	1. Sampling Methods 2. Sample Size 3. Statistical Analysis 4. Response Rate	1. Unclear 2. Unclear 3. Unclear 4. Unclear	1. Sampling method unclear. 2. Sample size calculations conducted, but unclear whether the sample size is appropriate for estimating prevalence rates.

Study	Bias Domain	Rating	Justification
Nigg (1999)	1. Sampling Methods	1. Unclear	3. Percentages of co-occurring ADHD+RD reported for the purposes of study grouping. Unclear whether this is an adequate prevalence estimate as this is not an epidemiological study.
	2. Sample Size	2. High	4. Response rate/missing data not discussed.
	3. Sample Coverage	3. Unclear	1. Sampling method unclear.
	4. Response Rate	4. Unclear	2. Sample too small to address prevalence adequately. Sample size calculations conducted, but not for the purposes of prevalence estimates.
Nigg et al. (1998)	1. Generalisability	1. High	3. Children with a specific ADHD subtype were excluded; therefore, it is unclear whether the final analyses sufficiently covered the identified sample.
	2. Sampling Methods	2. Unclear	4. Reports there were missing data, and some participants were removed, but this is not discussed further.
	3. Sample Size	3. Unclear	1. Sample consisted of males only.
	4. Diagnostic Assessment	4. Unclear	2. Sampling method unclear.
Pagirsky et al. (2017)	1. Sampling Methods	1. Unclear	3. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
	2. Sample Size	2. Unclear	4. Unclear who conducted assessments to confirm diagnoses.
	3. Diagnostic Assessment	3. Unclear	1. Sampling method unclear.
	4. Statistical Analysis	4. Unclear	2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
	5. Response Rate	5. Unclear	3. Unclear who administered diagnostic assessments.
Papaioannou et al. (2016)	1. Sample Coverage	1. Unclear	4. The co-occurrence of ADHD+RD is reported for the purposes of study grouping. May not be appropriate for determining prevalence estimates as this is not an epidemiological study.
	2. Diagnostic Assessment	2. Unclear	5. Response rate not discussed.
Pauc (2005)	1. Generalisability	1. Unclear	1. States that only 42% of participants provided consent, but it is unclear what type of participants were not covered by the analyses.
	2. Sampling Methods	2. High	2. Unclear who administered reading assessments and who confirmed ADHD diagnoses.
	3. Sample Size	3. Unclear	1. Participants recruited from a single clinic.
	4. Diagnostic Assessment	4. Unclear	2. Convenience sampling used, introduces selection bias.
			3. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
			4. Diagnostic assessments used to identify the conditions are not specified.

Study	Bias Domain	Rating	Justification
Pennington et al. (1993)	1. Generalisability	1. High	1. Sample consists of males only.
	2. Sampling Methods	2. Unclear	2. Sampling method unclear.
	3. Sample Size	3. High	3. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
	4. Statistical Analysis	4. Unclear	4. Number of participants meeting criteria for ADHD+RD is reported for the purposes of study grouping as this is not an epidemiological study. May not be appropriate for estimating prevalence rates.
	5. Response Rate	5. Unclear	5. Response rate/refusals not discussed.
Pereira et al. (2020)	1. Generalisability	1. Unclear	1. Sampling frame not described in enough detail.
	2. Sampling Methods	2. Unclear	2. Unclear how participants were sampled.
	3. Sample Size	3. Unclear	3. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
	4. Sample Coverage	4. Unclear	4. States that participants were excluded if they withdrew during the administering of measures, but it is unclear how much of the identified sample was covered by the final analyses.
	5. Diagnostic Assessment	5. Unclear	5. Unclear who administered assessments/confirmed diagnoses.
	6. Response Rate	6. Unclear	6. Refusals/missing data not discussed.
Pisecco et al. (2001)	1. Sample Size	1. Unclear	1. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
	2. Demographic Information	2. Unclear	2. Not enough information about the sample.
	3. Statistical Analysis	3. Unclear	3. The number of participants with ADHD+RD is reported for the purposes of group identification. This may not be appropriate for estimating prevalence rates.
	4. Response Rate	4. Unclear	4. Attrition rates for this sample are reported in previous papers, but this information is unclear in the current paper.
Pritchard et al. (2016)	1. Sampling Methods	1. Unclear	1. Sampling method unclear, potentially convenience sampling (introduces selection bias).
	2. Sample Size	2. Unclear	2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
	3. Sample Coverage	3. Unclear	3. Reports that participants who received modified versions of the school assessment based on special educational needs were excluded from the study. It is unclear whether the analyses sufficiently covered the identified sample.
	4. Response Rate	4. Unclear	4. Response rate not discussed.

Study	Bias Domain	Rating	Justification
Punt et al. (2010)	1. Sampling Methods	1. Unclear	1. Convenience sampling used, introduces selection bias.
	2. Sample Size	2. Unclear	2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
	3. Sample Coverage	3. Unclear	3. The entire identified sample was not included in the final analyses.
	4. Diagnostic Assessment	4. Unclear	Reports that most of the sample frame met inclusion criteria, but it is unclear whether the final analyses had sufficient coverage.
	5. Response Rate	5. Unclear	4. Unclear what assessments were used to identify the conditions. 5. Some children were not included in the study because they did not meet diagnostic criteria for RD, but response rate is not discussed further.
Russell et al. (2015)	1. Sample Coverage	1. Unclear	1. Unclear which specific groups were not covered by the analyses.
	2. Diagnostic Assessment	2. Unclear	2. ADHD was identified via retrospective parent reports of previous clinical diagnoses and teacher reports of educational needs. No standardised diagnostic assessments used to confirm ADHD.
Samuelsson et al. (2004)	1. Generalisability	1. High	1. Sample frame is not generalisable – prison inmates.
	2. Sample Size	2. Unclear	2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
	3. Diagnostic Assessment	3. Unclear	3. Unclear whether assessments used to identify conditions were standardised.
Sanchez-Moran et al. (2018)	1. Sampling Methods	1. Unclear	1. Sampling method unclear.
	2. Sample Size	2. Unclear	2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
	3. Sample Coverage	3. Unclear	3. Reports that only participants of Spanish origin were included in the analyses; it is unclear who was excluded.
	4. Diagnostic Assessment	4. Unclear	4. Unclear whether standardised diagnostic assessments were used because identification of conditions was not based on DSM criteria. It is unclear who administered diagnostic assessments.
	5. Statistical Analysis	5. Unclear	5. The number of participants with ADHD+RD was reported for the purposes of sample characterisation. It is unclear whether this is appropriate for estimating prevalence rates.
	6. Response Rate	6. Unclear	6. Response rates not discussed.
Schachar et al. (2004)	1. Sampling Methods	1. Unclear	1. Convenience sampling used, introduces selection bias.
	2. Sample Size	2. Unclear	2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
	3. Response Rate	3. Unclear	3. Response rates/refusals not discussed.

Study	Bias Domain	Rating	Justification
Semrud-Clikeman et al. (1992)	1. Sampling Methods 2. Sample Size 3. Response Rate	1. Unclear 2. Unclear 3. Unclear	1. Convenience sampling used, introduces selection bias. 2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately. 3. Reports that some participants had unavailable data, but this is not discussed further.
Sesma et al. (2009)	1. Sampling Methods 2. Sample Size 3. Diagnostic Assessment 4. Statistical Analysis 5. Response Rate	1. Unclear 2. Unclear 3. Unclear 4. Unclear 5. Unclear	1. Sampling methods unclear – possibly convenience sampling, which introduces selection bias. 2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately. 3. Unclear who administered diagnostic assessments. 4. The number of participants with ADHD+RD was reported for the purposes of sample characterisation. May not be an accurate estimate of prevalence rates. 5. Response rates not discussed.
Shanahan et al. (2006)	1. Sampling Methods 2. Sample Size 3. Sample Coverage 4. Diagnostic Assessment 5. Statistical Analysis	1. Unclear 2. Unclear 3. Unclear 4. Unclear 5. Unclear	1. Opportunity sampling used, introduces selection bias. 2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately. 3. States that only one twin was included in the final analyses from each identified twin pair. Unclear what this means for the sample coverage of the analyses. 4. Unclear who administered RD assessments. 5. The number of participants with ADHD+RD was reported for the purposes of sample characterisation. May not be an accurate estimate of prevalence rates.
Sheikhi et al. (2013)	1. Sampling Methods 2. Diagnostic Assessment	1. Unclear 2. Unclear	1. Sampling method unclear – potentially opportunity sampling, which introduces selection bias. 2. Unclear who administered diagnostic assessments.
Stack-Cutler et al. (2016)	1. Generalisability 2. Sampling Methods 3. Sample Size 4. Diagnostic Assessment 5. Response Rate	1. High 2. High 3. Unclear 4. High 5. Unclear	1. Sample restricted to university students. 2. Unclear sampling method, but appears to be opportunity sampling (which introduces selection bias). 3. Sample size calculation reported, but not for the purposes of estimating prevalence rates. The sample size may be insufficient for this purpose.

Study	Bias Domain	Rating	Justification
Stark et al. (2022)	1. Sampling Method	1. Unclear	4. ADHD was not assessed via a standardised diagnostic assessment. Identification of conditions was based on self-report questionnaires, which are subject to bias.
	2. Diagnostic Assessment	2. Unclear	5. It is reported that the authors were unable to calculate a response rate.
	3. Response Rate	3. Unclear	1. Retrospective study – non-random sampling method used. 2. Information about prior ADHD diagnosis was gathered via a questionnaire, but it is unclear if/how this was confirmed. Unclear how RD was identified. 3. Response/participation rates not discussed as this was a retrospective study.
Stevenson et al. (2005)	1. Sampling Methods	1. Unclear	1. Sampling method not reported clearly.
	2. Sample Size	2. Unclear	2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
	3. Sample Coverage	3. Unclear	3. Reports that participants with IQ below 70 and those who showed evidence of other conditions were excluded, so it is unclear whether the identified sample was sufficiently covered by the analyses.
	4. Diagnostic Assessment	4. Unclear	4. Unclear who administered diagnostic interviews for ADHD/reading assessments.
	5. Statistical Analysis	5. Unclear	5. The number of participants with ADHD+RD was reported for the purposes of sample characterisation. May not be an accurate estimate of prevalence rates.
	6. Response Rate	6. Unclear	6. Participation/refusal rate not discussed.
Swanson et al. (1999)	1. Sampling Methods	1. Unclear	1. Sampling method not reported clearly.
	2. Sample Size	2. Unclear	2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
	3. Statistical Analysis	3. Unclear	3. The number of participants with ADHD+RD was reported for the purposes of sample characterisation. May not be an accurate estimate of prevalence rates.
	4. Response Rate	4. Unclear	4. Response/participation rate not discussed.
Thomson et al. (2005)	1. Generalisability	1. Unclear	1. Unclear where the sample frame was recruited from.
	2. Sampling Methods	2. Unclear	2. Sampling method unclear – potentially convenience (selection bias).
	3. Sample Size	3. Unclear	3. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.
	4. Sample Coverage	4. Unclear	
	5. Diagnostic Assessment	5. Unclear	

Study	Bias Domain	Rating	Justification
	6. Statistical Analysis 7. Response Rate	6. Unclear 7. Unclear	4. Reports that participants with psychiatric/neurological disorders were excluded, so it is unclear whether the final analyses sufficiently covered the identified sample. 5. Unclear who administered diagnostic assessments. 6. The percentage of participants with ADHD+RD was reported for the purposes of sample characterisation. May not be an accurate estimate of prevalence rates. 7. Response rate not discussed.
Toffalini et al. (2022)	1. Sampling Methods 2. Sample Size 3. Response Rate	1. Unclear 2. Unclear 3. Unclear	1. Convenience sampling used, introduces selection bias. 2. Sample size seems large enough, but there is no evidence of sample size calculation. 3. Majority of identified participants were included in the study, but states that there were some missing data. This is not discussed further, so response rate is unclear.
Toplak et al. (2003)	1. Sampling Methods 2. Sample Size 3. Response Rate	1. Unclear 2. Unclear 3. Unclear	1. Sampling method unclear – most likely used convenience sampling, which introduces selection bias. 2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately. 3. Response/participation rate not discussed.
Tosto et al. (2021)	1. Sampling Methods 2. Sample Size 3. Diagnostic Assessment 4. Statistical Analysis 5. Response Rate	1. Unclear 2. High 3. High 4. Unclear 5. Unclear	1. Sampling method unclear – most likely used convenience sampling, which introduces selection bias. 2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately. 3. It is unclear how prior ADHD and RD diagnoses were identified or confirmed. 4. The percentage of participants with ADHD+RD was reported for the purposes of sample characterisation. May not be an accurate estimate of prevalence rates. 5. Reports some missing responses, but participation rate is unclear.
Vakil et al. (2012)	1. Sampling Methods 2. Sample Size 3. Diagnostic Assessment 4. Response Rate	1. Unclear 2. Unclear 3. Unclear 4. Unclear	1. Sampling method not reported. Looks like opportunity sampling (introduces selection bias). 2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately.

Study	Bias Domain	Rating	Justification
Visser et al. (2020)	1. Sample Coverage	1. Unclear	3. Unclear whether diagnostic assessments were not standardised. Unclear who conducted assessments. 4. Response rate not discussed. 1. It states several reasons for exclusions and that 33.6% of the sample was excluded. Unclear whether the analyses sufficiently cover the identified sample.
Voigt et al. (2017)	1. Sample Size 2. Diagnostic Assessment	1. Unclear 2. Unclear	1. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately. 2. Unclear who administered diagnostic assessments.
Wadsworth et al. (2007)	1. Sample Size 2. Diagnostic Assessment 3. Statistical Analysis	1. Unclear 2. Unclear 3. Unclear	1. States that the sample size of some groups was too small. No sample size calculation reported as this is an ongoing study. 2. Unclear who administered reading assessments/confirmed ADHD. 3. The percentage of participants with a positive history of RD and ADHD symptoms is only reported in the discussion section; this is not the focus of this paper, so may not be appropriate for estimating prevalence rates.
Wassenberg et al. (2010)	1. Generalisability 2. Sampling Methods 3. Sample Size 4. Diagnostic Assessment 5. Statistical Analysis 6. Response Rate	1. Unclear 2. High 3. High 4. Unclear 5. Unclear 6. Unclear	1. Participants recruited from a single clinic. 2. Convenience sampling used, introduces selection bias. 3. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately. 4. RD was not assessed in the study; participants with co-occurring RD were simply not excluded. 5. The number of participants with ADHD+RD was reported for the purposes of sample characterisation. May not be an accurate estimate of prevalence rates. 6. Response rates not discussed.
Westervelt et al. (1998)	1. Sampling Methods 2. Sample Size 3. Diagnostic Assessment 4. Statistical Analysis	1. Unclear 2. High 3. High 4. Unclear	1. Sampling methods not specified. 2. No sample size calculation. Sample size may be insufficient to address prevalence estimates adequately. 3. Prior diagnoses were not assessed or confirmed in this study. 4. The percentage of participants with ADHD+RD was reported for the purposes of sample characterisation. May not be an accurate estimate of prevalence rates.

Study	Bias Domain	Rating	Justification
Willcutt et al. (2000)	1. Sample Size	1. Unclear	1. Reports that the sample size was enough to provide sufficient power to detect differences, but it is unclear whether the sample is large enough to appropriately estimate prevalence as this was not the purpose of the study.
Wisniewska et al. (2007)	1. Generalisability	1. Unclear	1. Not enough information provided about the sample frame.
	2. Sampling Methods	2. Unclear	2. Sampling methods unclear.
	3. Sample Size	3. High	3. Very small sample. No sample size calculation.
	4. Demographic Information	4. Unclear	4. Demographic characteristics not described in detail.
	5. Sample Coverage	5. Unclear	5. Unclear how many participants were initially identified, cannot determine whether the final analyses were conducted with sufficient coverage.
	6. Diagnostic Assessment	6. Unclear	6. No report of how ADHD and RD were identified/diagnosed.
	7. Statistical Analysis	7. Unclear	7. A percentage is reported for the number of children with ADHD+RD, but statistical analyses are not described, so it is unclear whether this is an appropriate estimate of prevalence rates.
	8. Response Bias	8. Unclear	8. Unclear how many people were initially contacted; response rate/refusals not discussed.
Yoshimasu et al. (2010)	1. Diagnostic Assessment	1. Unclear	1. Diagnostic assessments not described in detail in this paper (signposted to previous publications). Unclear who assessed and confirmed diagnoses.

Note. This table corresponds to the **JBI Checklist for Studies Reporting Prevalence Data**. The checklist contains 9 items: 1) Was the sample frame appropriate to address the target population? 2) Were study participants sampled in an appropriate way? 3) Was the sample size adequate? 4) Were the study subjects and the setting described in detail? 5) Was the data analysis conducted with sufficient coverage of the identified sample? 6) Were valid methods used for the identification of the condition? 7) Was the condition measured in a standard, reliable way for all participants? 8) Was there appropriate statistical analysis? 9) Was the response rate adequate, and if not, was the low response rate managed adequately? Items 6 and 7 were merged into the ‘Diagnostic Assessment’ bias domain in the above summary table. Note that this table contains justifications for bias domains rated as unclear or high-risk only. Domains with low risk of bias were not included, but are visualised in Appendix I.

Appendix H. Risk of Bias Assessment Justifications (RQ2)

Table 1. Detailed justifications for studies with unclear or high risk of bias domains (RQ2).

Study	Bias Domain	Rating	Justification
Adams et al. (2001)	1. Inclusion Criteria	1. Unclear	1. Inclusion criteria not described in detail.
	2. Diagnostic Assessment	2. Unclear	2. Standard diagnostic criteria were not used to identify the conditions.
	3. Outcome Measurement	3. Unclear	3. Unclear whether all cognitive measures were standardised. Unclear who administered assessments.
Areces et al. (2018)	1. Inclusion Criteria	1. Unclear	1. Inclusion criteria not described in detail.
	2. Diagnostic Assessment	2. Unclear	2. Prior RD diagnosis was not confirmed in the study.
Caldani et al. (2022)	1. Inclusion Criteria	1. Unclear	1. Inclusion criteria are unclear.
	2. Demographic Information	2. Unclear	2. Study setting not described in detail.
Capano et al. (2008)	1. Confounding Factors	1. Unclear	1. Potential confounding factors not explicitly stated. Does not report whether participants were matched on any characteristics or whether any variables were accounted for in the analyses.
Chadwick et al. (1999)	1. Inclusion Criteria	1. Unclear	1. Inclusion criteria are not clearly defined.
	2. Confounding Factors	2. Unclear	2. Differences in IQ were measured, but not identified as a confounding factor. Confounding factors unclear.
Chan et al. (2008)	1. Inclusion Criteria	1. Unclear	1. Inclusion criteria not reported.
	2. Confounding Factors	2. Unclear	2. Age and IQ controlled for in analyses, but not explicitly identified as confounding factors.
Crippa et al. (2015)	1. Demographic Information	1. Unclear	1. Study setting not described in detail.
De Groot et al. (2017)	1. Diagnostic Assessment	1. Unclear	1. Unclear whether ADHD diagnosis was confirmed via a standardised assessment.

Study	Bias Domain	Rating	Justification
De Jong et al. (2012)	1. Outcome Measurement	1. Unclear	1. Unclear whether all cognitive assessments were standardised. Unclear who administered assessments.
Dewey et al. (2001)	1. Inclusion Criteria	1. Unclear	1. Inclusion criteria not described in detail.
Fernandez-Andres et al. (2021)	1. Outcome Measurement	1. Unclear	1. Cognition was not directly assessed via tasks; the outcome measure was teacher-report, which may not be as reliable.
Gregg et al. (2008)	1. Inclusion Criteria 2. Diagnostic Assessment 3. Confounding Factors	1. Unclear 2. Unclear 3. Unclear	1. Inclusion criteria are clear, but exclusion criteria are not described. 2. Diagnostic assessments are not described in the paper. Unclear whether standard diagnostic criteria were used. 3. Confounding factors were not identified.
Horowitz-Kraus (2016)	1. Inclusion Criteria	1. Unclear	1. Exclusion criteria not clearly defined.
Jakobson et al. (2007)	1. Diagnostic Assessment	1. Unclear	1. Diagnostic assessment of ADHD not specified. Reports that there are no available standardised tests of RD in the country where the study was conducted.
Langer et al. (2019)	1. Diagnostic Assessment	1. Unclear	1. Diagnostic assessments not specified. Diagnostic criteria not specified.
Li et al. (2022)	1. Inclusion Criteria 2. Demographic Information 3. Diagnostic Assessment 4. Outcome Measure	1. Unclear 2. Unclear 3. Unclear 4. Unclear	1. Exclusion criteria unclear. 2. IQ and gender not reported. 3. RD was not assessed via a standardised measure. 4. Unclear if cognitive assessments were standardised.
McGee et al. (2000)	1. Inclusion Criteria	1. Unclear	1. Exclusion criteria unclear.

Study	Bias Domain	Rating	Justification
Miranda et al. (2017)	1. Inclusion Criteria 2. Outcome Measure	1. Unclear 2. Unclear	1. Exclusion criteria unclear. 2. Direct cognitive assessment was not administered; informant report may introduce bias.
Narhi et al. (1995)	1. Inclusion Criteria 2. Diagnostic Assessment	1. Unclear 2. Unclear	1. Exclusion criteria unclear. 2. Unclear if RD measure was standardised. Unclear whether standard diagnostic criteria were used.
Pagirsky et al. (2017)	1. Inclusion Criteria 2. Diagnostic Assessment	1. Unclear 2. Unclear	1. Exclusion criteria unclear. 2. Unclear how ADHD diagnosis was confirmed.
Parke et al. (2020)	1. Diagnostic Assessment	1. Unclear	1. Diagnostic assessments not specified.
Pisecco et al. (2001)	1. Inclusion Criteria	1. Unclear	1. Exclusion criteria not clearly defined.
Poon et al. (2014)	1. Diagnostic Assessment	1. Unclear	1. Diagnostic criteria not specified.
Poon et al. (2021)	1. Diagnostic Assessment	1. Unclear	1. Diagnostic criteria not specified.
Savitz et al. (2003)	1. Inclusion Criteria 2. Diagnostic Assessment	1. Unclear 2. Unclear	1. Exclusion criteria not clearly defined. 2. Diagnostic criteria not specified.
Swanson et al. (1999)	1. Inclusion Criteria	1. Unclear	1. Exclusion criteria not clearly defined.
Tiffin-Richards et al. (2008)	1. Diagnostic Assessment	1. Unclear	1. Diagnostic assessment of RD not specified.
Whipple et al. (2016)	1. Diagnostic Assessment	1. Unclear	1. Diagnostic assessment not specified.

Note. This table corresponds to the **JBIChecklist for Analytical Cross-Sectional Studies**. The checklist contains 8 items:

1) Were the criteria for inclusion on the sample clearly defined? 2) Were the study subjects and setting described in detail? 3) Was the exposure measured in a valid and reliable way? 4) Were objective, standard criteria used for measurement of the condition? 5) Were confounding factors identified? 6) Were strategies to deal with confounding factors stated? 7) Were the outcomes measured in a valid and reliable way? 8) Was appropriate statistical analysis used? Items 3 and 4 were merged into the 'Diagnostic Assessment' bias domain, while items 5 and 6 were merged into the 'Confounding Factors' domain. Note that this table contains justifications for bias domains rated as unclear or high-risk only. Domains with low risk of bias were not included, but are visualised in Appendix J.

Appendix I. Traffic light plot of risk of bias judgments across all domains for each study addressing RQ1.

	Risk of bias								
	D1	D2	D3	D4	D5	D6	D7	D8	D9
Aaron et al. (2002)	-	X	X	+	+	+	+	-	+
Adams et al. (1999)	X	X	-	+	+	-	+	+	+
Aitken et al. (2020)	-	-	-	+	-	-	+	-	+
Altay et al. (2018)	-	X	X	+	+	+	+	+	+
Aro et al. (2022)	+	+	+	+	+	+	+	+	+
Asberg et al. (2010)	X	X	X	+	+	+	+	+	+
Becker et al. (2021)	-	-	X	+	+	+	+	+	+
Biederman et al. (2002)	+	-	-	+	+	+	+	+	+
Bonafina et al. (2000)	X	X	X	+	+	+	+	-	+
Brandenburg et al. (2021)	-	-	X	+	+	+	-	-	+
Brimo et al. (2021)	+	+	+	-	+	+	+	+	+
Capano et al. (2008)	-	X	-	+	+	+	+	+	+
Carlsson et al. (2022)	X	X	X	+	+	+	+	-	+
Carmine Pastura et al. (2009)	-	X	-	+	-	+	+	-	+
Carroll et al. (2005)	+	+	+	+	-	+	+	+	+
Casey et al. (2000)	-	X	X	-	+	+	+	-	+
Cederlof et al. (2017)	+	+	+	+	-	+	+	+	-
Condo et al. (2022)	+	-	X	+	+	-	-	+	-
Crawford et al. (2006)	+	+	-	+	+	+	+	-	+
Czamara et al. (2013)	+	+	+	+	-	-	+	+	-
Darweesh et al. (2020)	-	-	X	+	+	+	+	+	+
Del'Homme et al. (2007)	+	+	-	+	-	+	+	+	-
Doyle et al. (2005)	-	-	X	+	+	+	+	+	+
Dykman et al. (1991)	-	X	X	+	+	+	-	-	+
Efron et al. (2014)	+	+	+	+	X	+	+	+	+
El Sheikh et al. (2016)	+	+	+	+	+	+	-	+	+
Espanol-Martin et al. (2023)	+	+	+	+	+	+	+	+	-
Faraone et al. (1993)	X	X	X	+	-	+	+	+	-
Fortes et al. (2016)	+	+	+	+	-	+	+	+	-
Frances et al. (2023)	+	-	+	+	+	+	+	+	-
Goker et al. (2014)	+	-	+	+	-	-	-	-	-
Goldston et al. (2007)	+	-	-	+	+	+	+	+	+
Halleland et al. (2019)	+	+	-	+	+	+	+	-	+
Hechtman et al. (2005)	-	-	-	-	+	+	-	+	-
Heikkila et al. (2016)	-	-	-	+	+	+	-	-	-
Holland et al. (2012)	+	+	+	+	+	-	-	-	+
Herrera-Gutierrez et al. (2021)	+	-	-	+	-	-	-	-	-
Hinshaw (2002)	+	-	-	+	+	+	+	+	-
Ho et al. (2005)	-	-	-	+	+	+	-	-	-
Huang et al. (2007)	+	-	-	+	-	-	-	-	-
Johnson et al. (2021)	+	-	-	+	+	-	+	-	-
Kadesjo et al. (2001)	+	+	-	+	+	+	+	+	+
Kaplan et al. (2001)	+	+	-	+	-	+	+	-	+
Kita et al. (2020)	+	-	-	-	-	+	+	+	-
Klorman et al. (1999)	+	-	-	+	+	+	-	-	-
Koerner et al. (2021)	+	+	+	+	-	+	-	+	-
Lamminmaki et al. (1995)	-	X	-	+	-	+	-	+	-
Langberg et al. (2010)	+	-	-	+	+	+	-	+	-
Li et al. (2020)	+	-	+	+	+	+	-	-	-

	Risk of bias								
	D1	D2	D3	D4	D5	D6	D7	D8	D9
Little et al. (2010)	+	+	-	+	+	-	-	+	+
Livingston et al. (1990)	-	X	-	+	X	+	+	+	-
Loo et al. (2004)	+	X	-	+	-	+	-	-	-
Luoni et al. (2015)	-	-	-	+	-	+	+	+	+
Marshall et al. (1997)	-	X	-	+	-	+	+	+	-
Martinussen et al. (2015)	+	-	-	+	+	+	+	-	+
Mayes et al. (2006)	+	-	+	+	+	+	+	+	-
Mayes et al. (2020)	-	X	-	+	+	+	+	+	-
McGee et al. (2000)	-	X	-	+	+	+	+	-	-
Miranda et al. (2017)	-	-	X	+	+	+	+	-	-
Molitor et al. (2016)	+	-	-	+	+	+	+	+	-
Nakashima et al. (2012)	X	X	X	+	+	-	+	-	-
Narhi et al. (1995)	X	X	X	+	-	+	+	-	-
Naz et al. (2019)	+	-	-	+	+	+	+	-	-
Nigg (1999)	+	-	X	+	-	+	+	+	-
Nigg et al. (1998)	X	-	-	+	+	+	-	+	+
Pagirsky et al. (2017)	+	-	-	+	+	+	-	-	-
Papaioannou et al. (2016)	+	+	+	+	-	+	-	+	+
Pauc (2005)	-	X	-	+	+	-	+	+	+
Pennington et al. (1993)	X	-	X	+	+	+	+	-	-
Pereira et al. (2020)	-	-	-	+	-	+	-	+	-
Pisecco et al. (2001)	+	+	-	-	+	+	+	-	-
Pritchard et al. (2016)	+	-	-	+	-	+	+	+	-
Punt et al. (2010)	+	-	-	+	-	-	+	+	-
Russell et al. (2015)	+	+	+	+	-	-	+	+	+
Samuelsson et al. (2004)	X	+	-	+	+	-	+	+	+
Sanchez-Moran et al. (2018)	+	-	-	+	-	-	-	-	-
Schachar et al. (2004)	+	-	-	+	+	+	+	+	-
Semrud-Clikeman et al. (1992)	+	-	-	+	+	+	+	+	-
Sesma et al. (2009)	+	-	-	+	+	+	-	-	-
Shanahan et al. (2006)	+	-	-	+	-	+	-	-	+
Sheikhi et al. (2013)	+	-	+	+	+	+	-	+	+
Stack-Cutler et al. (2016)	X	X	-	+	+	X	X	+	-
Stark et al. (2022)	+	-	+	+	+	-	-	+	-
Stevenson et al. (2005)	+	-	-	+	-	+	-	-	-
Swanson et al. (1999)	+	-	-	+	+	+	+	-	-
Thomson et al. (2005)	-	-	-	+	-	+	-	-	-
Toffalini et al. (2022)	+	-	-	+	+	+	+	+	-
Toplak et al. (2003)	+	-	-	+	+	+	+	+	-
Tosto et al. (2021)	+	-	X	+	+	X	X	-	-
Vakil et al. (2012)	+	-	-	+	+	-	-	+	-
Visser et al. (2020)	+	+	+	+	-	+	+	+	+
Voigt et al. (2017)	+	+	-	+	+	+	-	+	+
Wadsworth et al. (2007)	+	+	-	+	+	+	-	-	+
Wassenberg et al. (2010)	-	X	X	+	+	-	+	-	-
Westervelt et al. (1998)	+	-	X	+	+	X	X	-	+
Willcutt et al. (2000)	+	+	-	+	+	+	+	+	+
Wisniewska et al. (2007)	-	-	X	-	-	-	-	-	-
Yoshimasu et al. (2010)	+	+	+	+	+	-	-	+	+

Note. This plot corresponds to the **JBI Checklist for Studies Reporting Prevalence Data**. Red = High Risk of Bias. Yellow = Unclear/Some Concerns. Green = Low Risk of Bias. D1 = Was the sample frame appropriate to address the target population? D2 = Were study participants sampled in an appropriate way? D3 = Was the sample size adequate? D4 = Were the study subjects and the setting described in detail? D5 = Was the data analysis conducted with sufficient coverage of the identified sample? D6 = Were valid methods used for the identification of the condition? D7 = Was the condition measured in a standard, reliable way for all participants? D8 = Was there appropriate statistical analysis? D9 = Was the response rate adequate, and if not, was the low response rate managed adequately? This table was created using the *robvis* web application for visualising risk of bias assessments (McGuinness & Higgins, 2020).

Appendix J. Traffic light plot of risk of bias judgments across all domains for each study addressing RQ2.

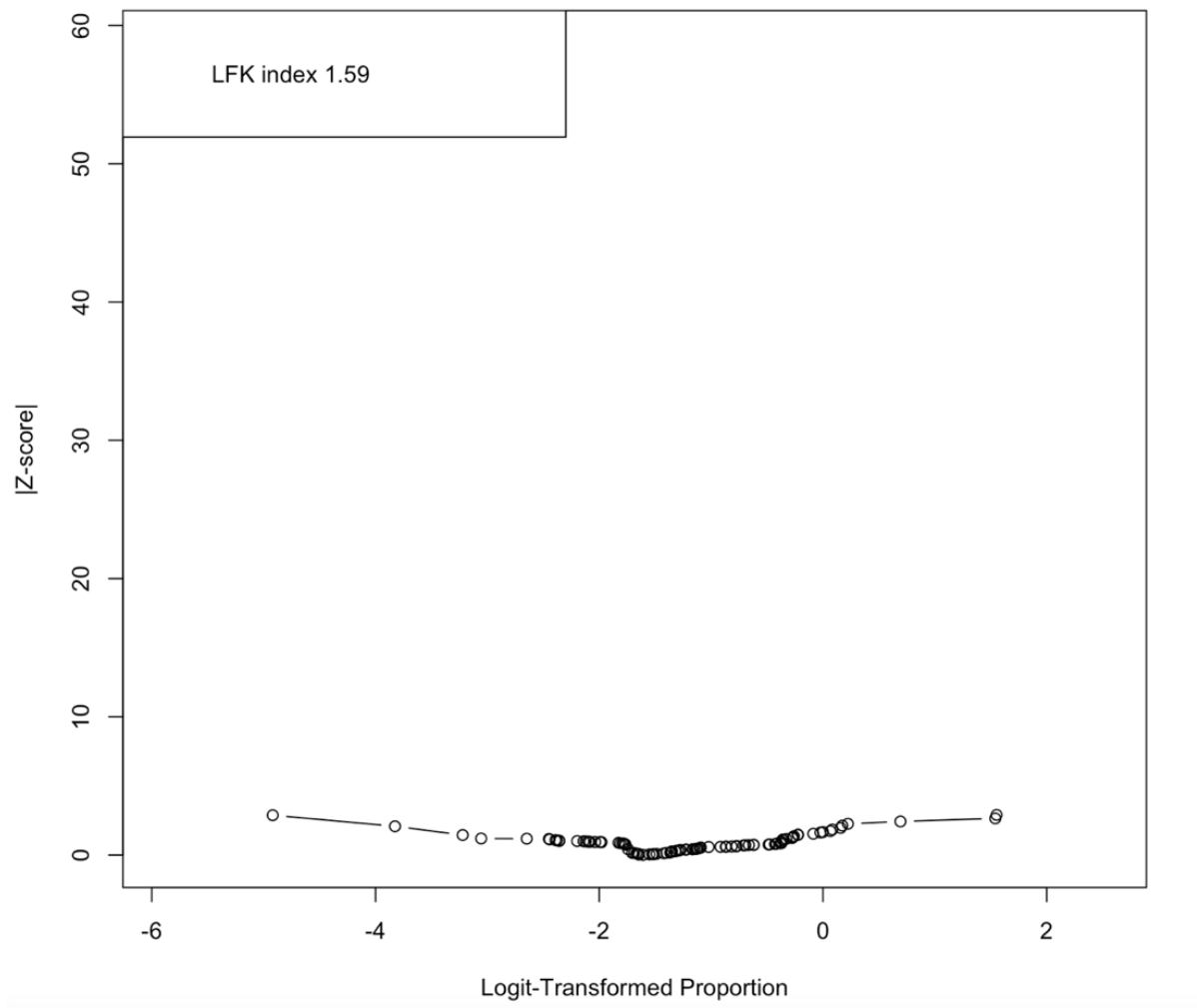
	Risk of bias							
	D1	D2	D3	D4	D5	D6	D7	D8
Adams et al. (2001)	⊖	⊕	⊕	⊖	⊕	⊕	⊖	⊕
Al Dahhan et al. (2022)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Areces et al. (2018)	⊖	⊕	⊖	⊕	⊕	⊕	⊕	⊕
Becker et al. (2021)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Bental et al. (2007)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Caldani et al. (2022)	⊖	⊖	⊕	⊕	⊕	⊕	⊕	⊕
Capano et al. (2008)	⊕	⊕	⊕	⊕	⊖	⊖	⊕	⊕
Chadwick et al. (1999)	⊖	⊕	⊕	⊕	⊖	⊖	⊕	⊕
Chan et al. (2008)	⊖	⊕	⊕	⊕	⊖	⊕	⊕	⊕
Crippa et al. (2015)	⊕	⊖	⊕	⊕	⊕	⊕	⊕	⊕
De Groot et al. (2017)	⊕	⊕	⊖	⊕	⊕	⊕	⊕	⊕
De Jong et al. (2012)	⊕	⊕	⊕	⊕	⊕	⊕	⊖	⊕
De Jong et al. (2009)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Dewey et al. (2001)	⊖	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Fernandez-Andres et al. (2021)	⊕	⊕	⊕	⊕	⊕	⊕	⊖	⊕
Ghelani et al. (2004)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Gooch et al. (2011)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Gregg et al. (2008)	⊖	⊕	⊖	⊖	⊖	⊖	⊕	⊕
Hall et al. (1997)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Ho et al. (2005)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Horowitz-Kraus (2016)	⊖	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Horowitz-Kraus et al. (2019)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Jakobson et al. (2007)	⊕	⊕	⊖	⊕	⊕	⊕	⊕	⊕
Johnson et al. (1999)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Kaplan et al. (2000)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Katz et al. (2011)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Kibby et al. (2008)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Kibby et al. (2021)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Laasonen et al. (2012)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Laasonen et al. (2009)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Langer et al. (2019)	⊕	⊕	⊖	⊖	⊕	⊕	⊕	⊕
Lee (2023)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Li et al. (2022)	⊖	⊖	⊖	⊕	⊕	⊕	⊖	⊕
Maehler et al. (2016)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Mano et al. (2017)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Martinussen et al. (2006)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
McGee et al. (2000)	⊖	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Miranda et al. (2017)	⊖	⊕	⊕	⊕	⊕	⊕	⊖	⊕
Mohl et al. (2018)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕
Moura et al. (2017)	⊕	⊕	⊕	⊕	⊕	⊕	⊕	⊕

Narhi et al. (1995)	-	+	-	-	+	+	+	+
Nigg et al. (1998)	+	+	+	+	+	+	+	+
Pagirsky et al. (2017)	-	+	-	+	+	+	+	+
Parke et al. (2020)	+	+	-	+	+	+	+	+
Pennington et al. (1993)	+	+	+	+	+	+	+	+
Pereira et al. (2020)	+	+	+	+	+	+	+	+
Pisecco et al. (2001)	-	+	+	+	+	+	+	+
Poon et al. (2014)	+	+	+	-	+	+	+	+
Poon et al. (2021)	+	+	+	-	+	+	+	+
Purvis et al. (2000)	+	+	+	+	+	+	+	+
Rucklidge et al. (2002)	+	+	+	+	+	+	+	+
Savitz et al. (2003)	-	+	+	-	+	+	+	+
Seidman et al. (2001)	+	+	+	+	+	+	+	+
Shanahan et al. (2006)	+	+	+	+	+	+	+	+
Shaywitz et al. (1995)	+	+	+	+	+	+	+	+
Shin et al. (2003)	+	+	+	+	+	+	+	+
Stubenrauch et al. (2014)	+	+	+	+	+	+	+	+
Swanson et al. (1999)	-	+	+	+	+	+	+	+
Tannock et al. (2000)	+	+	+	+	+	+	+	+
Tiffin-Richards et al. (2008)	+	+	-	+	+	+	+	+
Toplak et al. (2003)	+	+	+	+	+	+	+	+
Turker et al. (2019)	+	+	+	+	+	+	+	+
Van de Voorde et al. (2010)	+	+	+	+	+	+	+	+
Wang et al. (2018)	+	+	+	+	+	+	+	+
Whipple et al. (2016)	+	+	-	+	+	+	+	+
Willcutt et al. (2001)	+	+	+	+	+	+	+	+
Willcutt et al. (2005)	+	+	+	+	+	+	+	+
Wu et al. (2002)	+	+	+	+	+	+	+	+
Zhang et al. (2022)	+	+	+	+	+	+	+	+

Note. This plot corresponds to the **JBI Checklist for Analytical Cross-Sectional Studies**. Red = High Risk of Bias. Yellow = Unclear/Some Concerns. Green = Low Risk of Bias. D1 = Were the criteria for inclusion on the sample clearly defined? D2 = Were the study subjects and setting described in detail? D3 = Was the exposure measured in a valid and reliable way? D4 = Were objective, standard criteria used for measurement of the condition? D5 = Were confounding factors identified? D6 = Were strategies to deal with confounding factors stated? D7 = Were the outcomes measured in a valid and reliable way? D8 = Was appropriate statistical analysis used? This table was created using the *robvis* web application for visualising risk of bias assessments (McGuinness & Higgins, 2020).

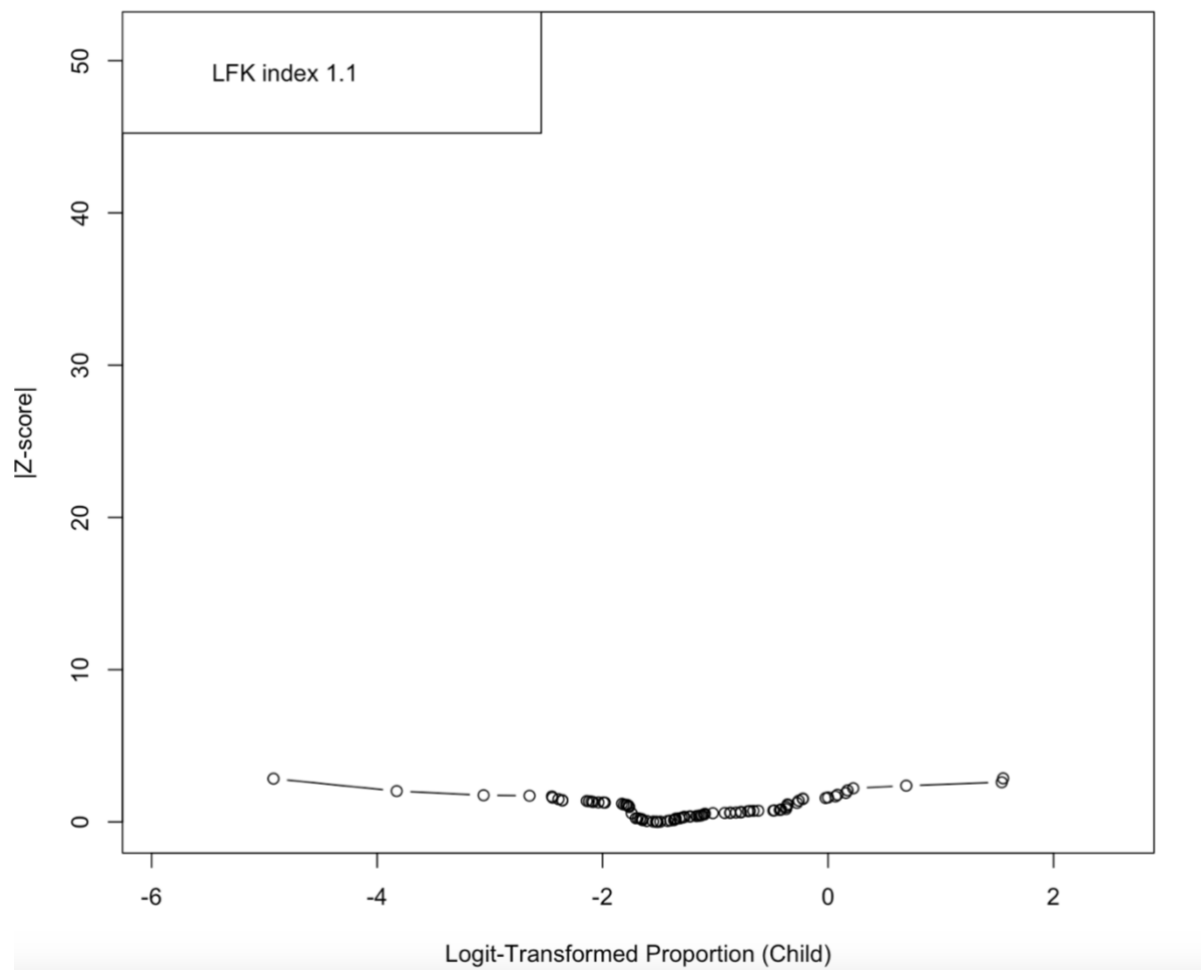
Appendix K. Doi Plots Assessing Reporting Bias

Figure 1. Doi plot for the pooled proportions of ADHD+RD across child and adult studies.



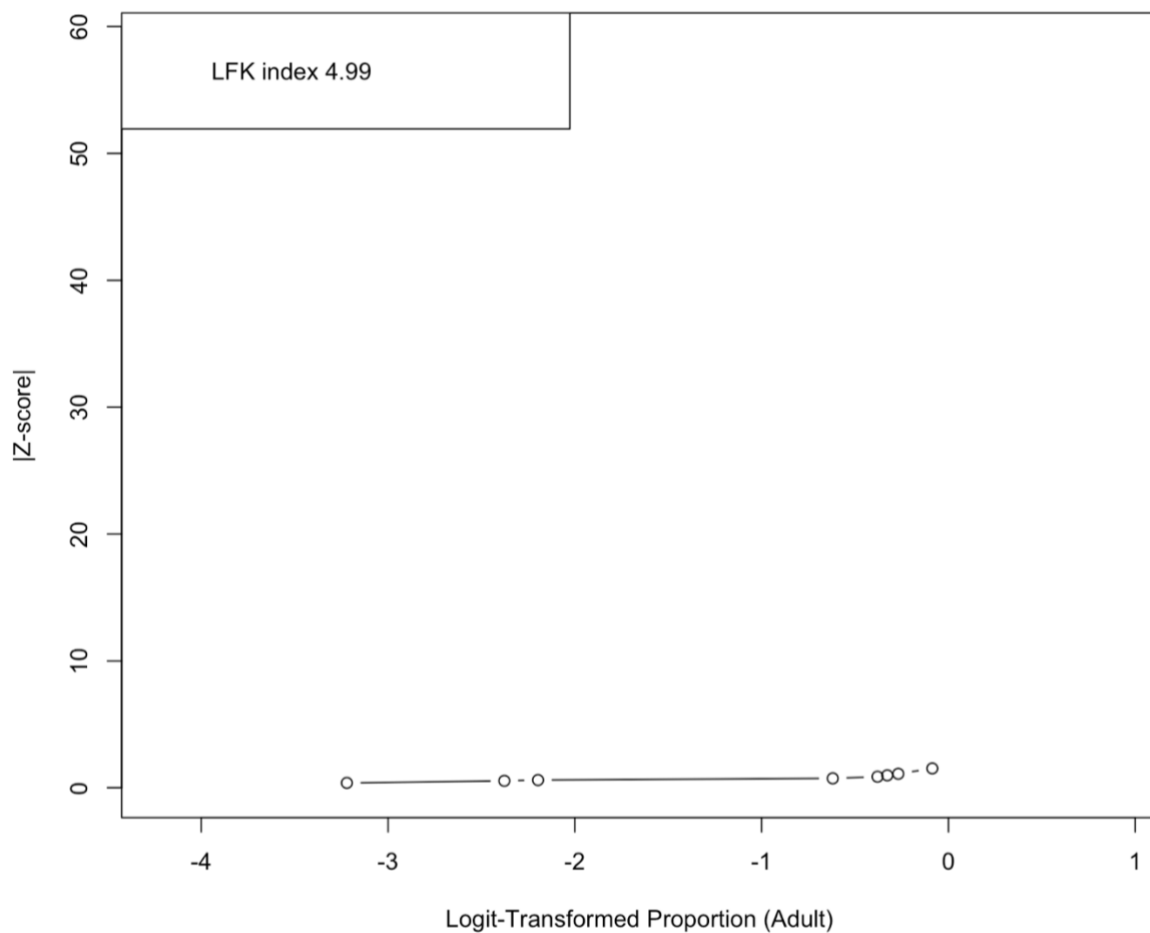
Note. An LFK index outside the range of -1 and +1 should be interpreted as indicating asymmetry and therefore publication bias (Furuya-Kanamori et al., 2018).

Figure 2. Doi plot for the pooled proportions of ADHD+RD for child studies only.



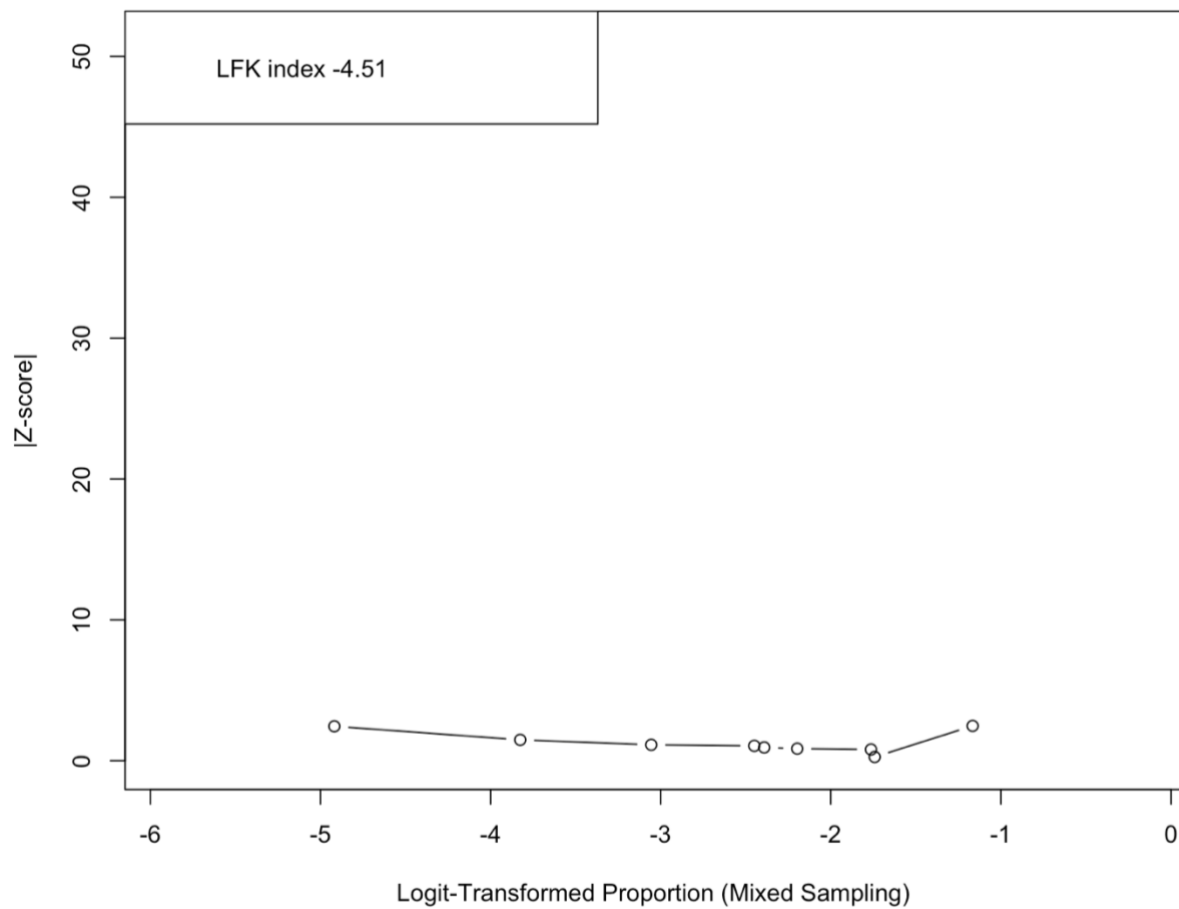
Note. An LFK index outside the range of -1 and +1 should be interpreted as indicating asymmetry and therefore publication bias (Furuya-Kanamori et al., 2018).

Figure 3. Doi plot for the pooled proportions of ADHD+RD for adult studies only.



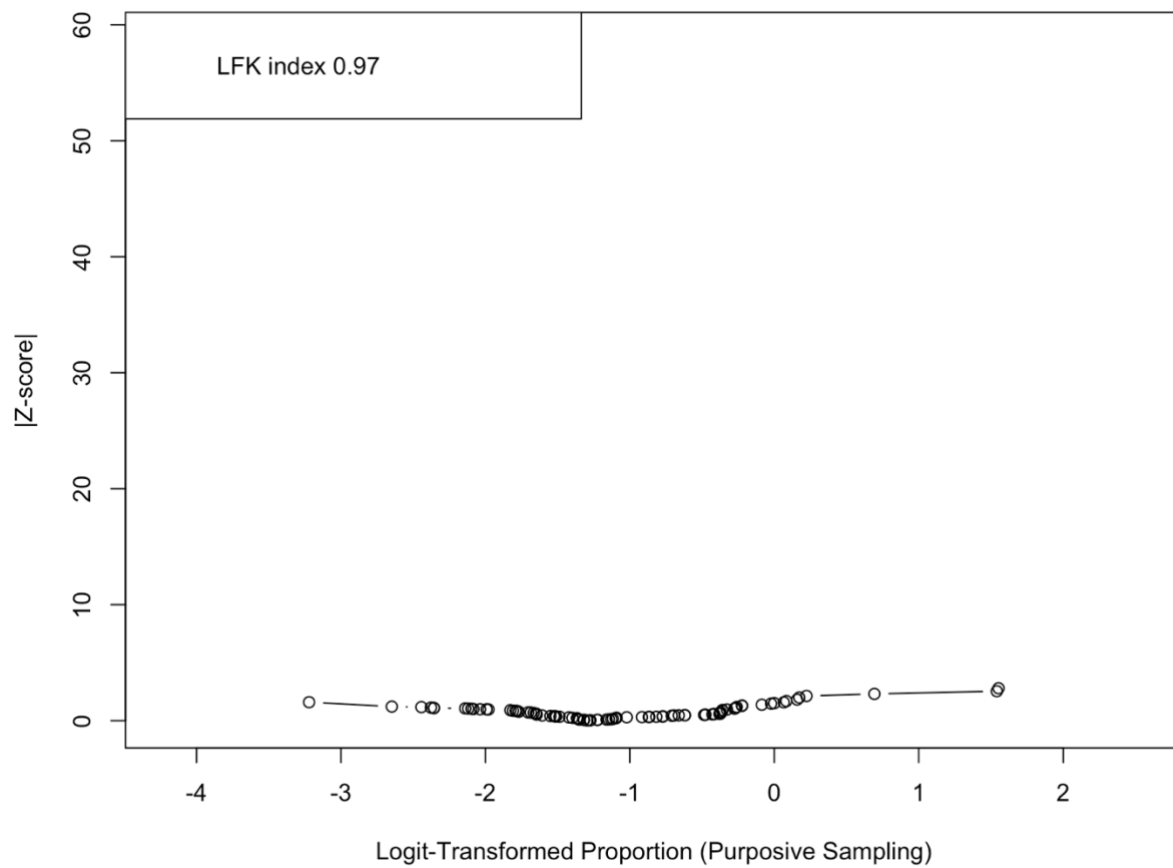
Note. An LFK index outside the range of -1 and +1 should be interpreted as indicating asymmetry and therefore publication bias (Furuya-Kanamori et al., 2018).

Figure 4. Doi plot for the pooled proportions of ADHD+RD for studies employing mixed sampling.



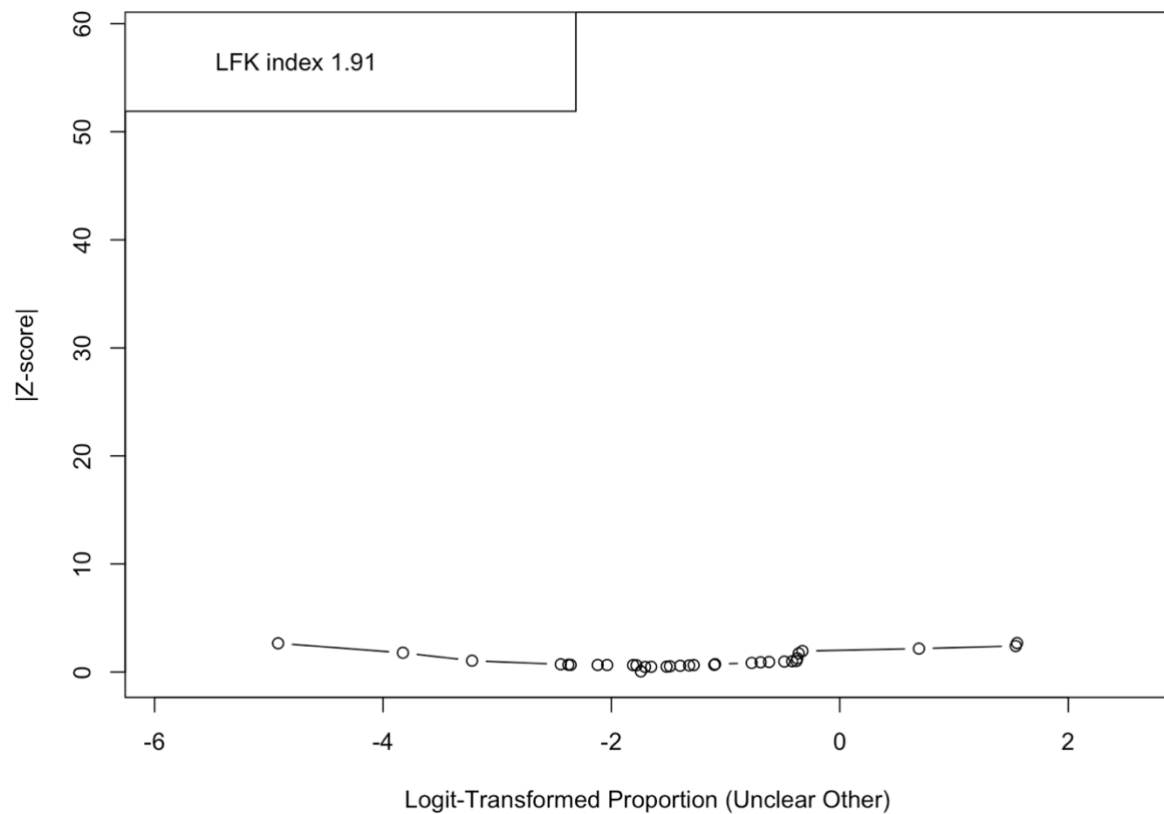
Note. An LFK index outside the range of -1 and +1 should be interpreted as indicating asymmetry and therefore publication bias (Furuya-Kanamori et al., 2018).

Figure 5. Doi plot for the pooled proportions of ADHD+RD for studies employing purposive sampling.



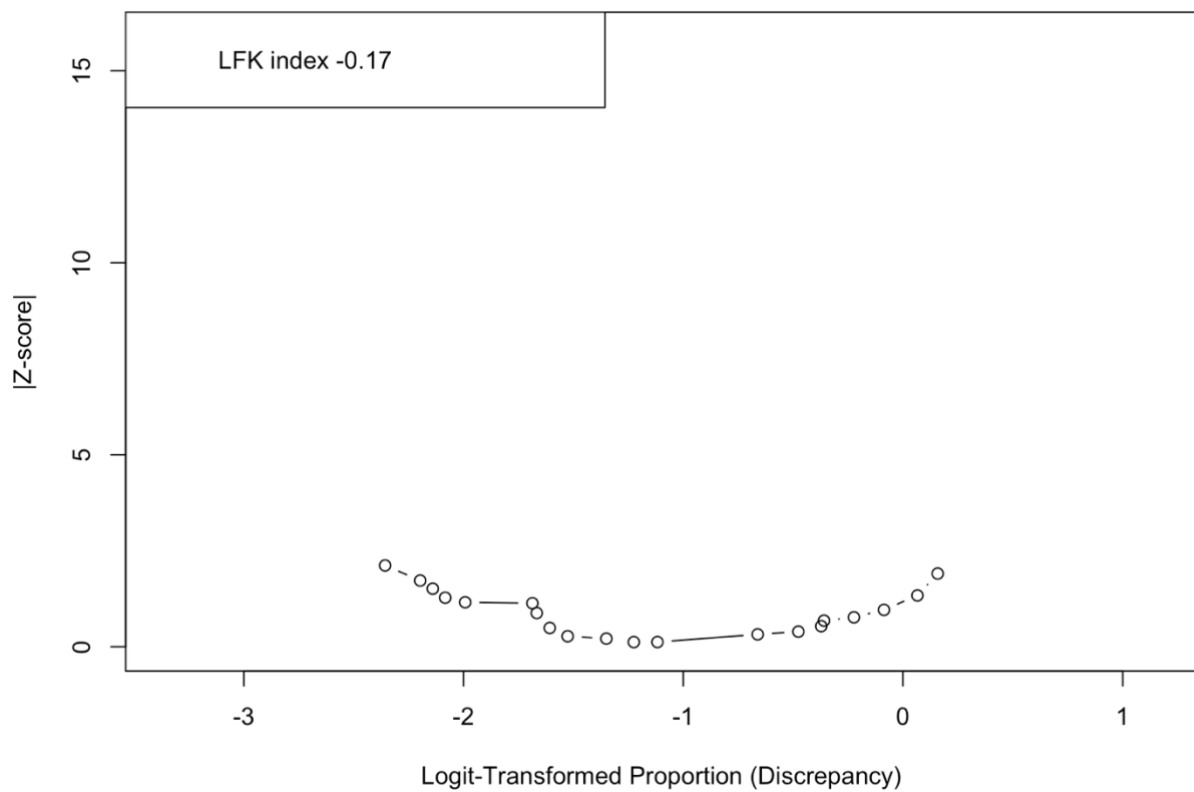
Note. An LFK index outside the range of -1 and +1 should be interpreted as indicating asymmetry and therefore publication bias (Furuya-Kanamori et al., 2018).

Figure 6. Doi plot for the pooled proportions of ADHD+RD for studies using an unclear (or not widely used) model to define RD.



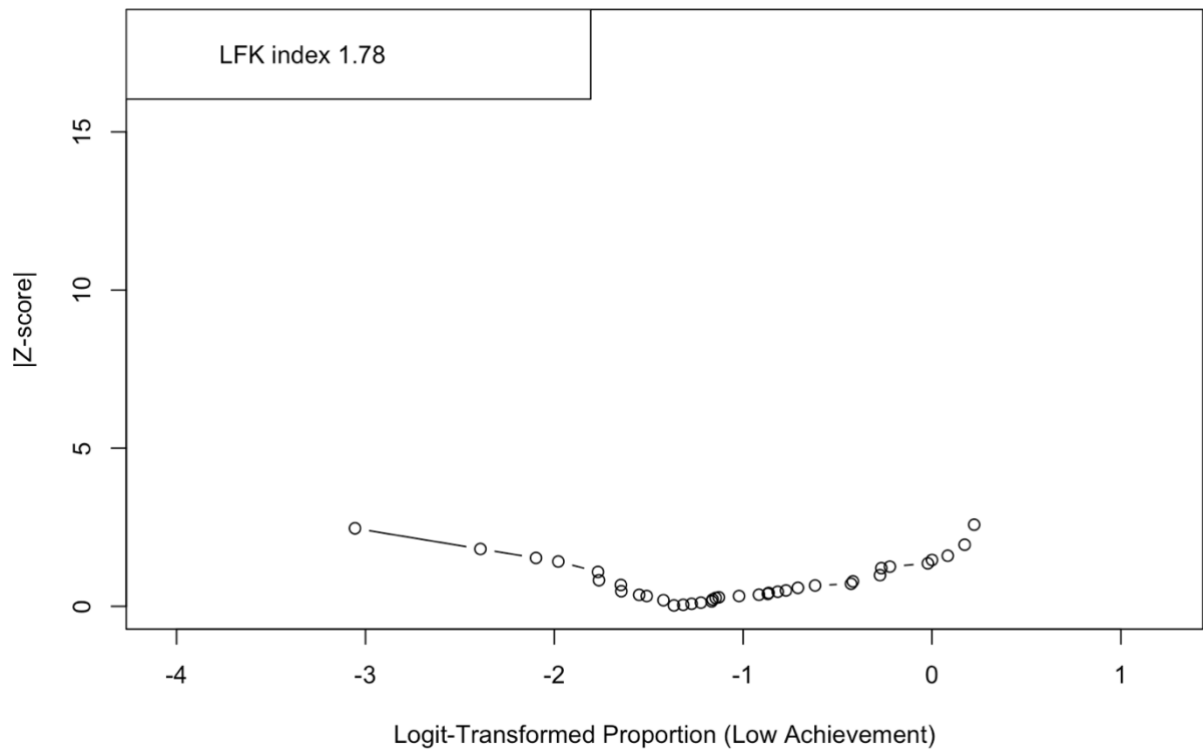
Note. An LFK index outside the range of -1 and +1 should be interpreted as indicating asymmetry and therefore publication bias (Furuya-Kanamori et al., 2018).

Figure 7. Doi plot for the pooled proportions of ADHD+RD for studies using the ‘discrepancy’ model to define RD.



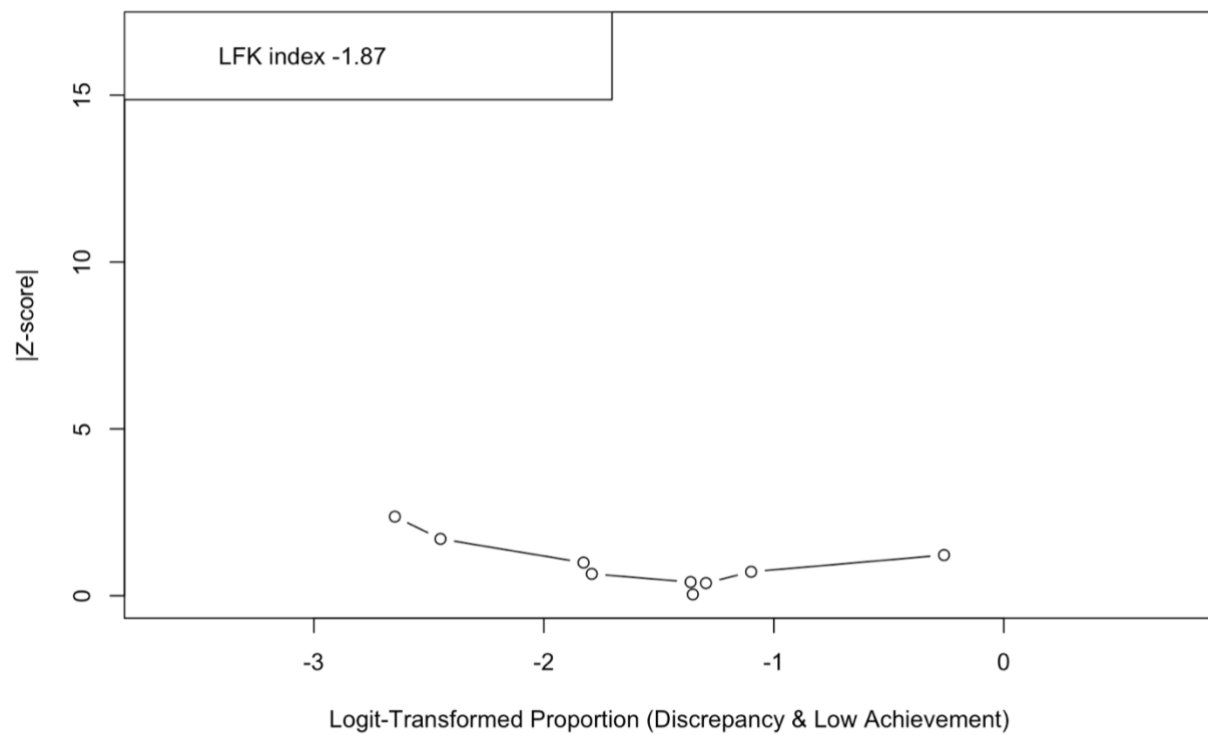
Note. An LFK index outside the range of -1 and +1 should be interpreted as indicating asymmetry and therefore publication bias (Furuya-Kanamori et al., 2018).

Figure 8. Doi plot for the pooled proportions of ADHD+RD for studies using the ‘low achievement’ model to define RD.



Note. An LFK index outside the range of -1 and +1 should be interpreted as indicating asymmetry and therefore publication bias (Furuya-Kanamori et al., 2018).

Figure 9. Doi plot for the pooled proportions of ADHD+RD for studies using a combination of ‘discrepancy’ and ‘low achievement’ models to define RD.



Note. An LFK index outside the range of -1 and +1 should be interpreted as indicating asymmetry and therefore publication bias (Furuya-Kanamori et al., 2018).



PARTICIPANTS NEEDED



FOR RESEARCH INTO NEURODIVERSITY

We are looking for **volunteers** to help us gain a better understanding of **typical and atypical development** and how this may manifest in different individuals.



We aim to **improve** individual wellbeing in the long run through increasing people's access to the **support** they need!

Do you have any difficulties related to **cognitive function** (e.g., attention, learning and/or memory)?

Have you been **referred** to assessment or **support services** for any difficulties?

Are you able to provide informed **consent** on your **own** behalf?

Are you aged between **16** and **50**?



If you have answered **YES** to the above questions, please e-mail **Karin Madericova** at **170096872@aston.ac.uk** to register your interest!

Please scan the above QR code to visit the **ECCaToN website for more information on the project**



Appendix B. Ethical Approval



Aston University
Aston Triangle
Birmingham
B4 7ET
0121 204 3000

Date: 20th December 2022

Karin Madericova
Copy: Prof Joel Talcott
College of Health & Life Sciences

Dear Karin Madericova,

Study title:	Embracing Complexity in the Characterisation and Tracking of Neurodevelopment (ECCaToN)
REC REF:	#HLS21066

Confirmation of Ethical Opinion

On behalf of the Committee, I am pleased to confirm a favourable opinion for the above research on the basis described in the application form, protocol and supporting documentation listed below.

Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

Document	Version	Date
Aston University Research Ethics Application Form	3	20/12/2022
Consent Form	2	15/12/2022
Participant Information Sheet	2	15/12/2022
Debrief Form	2	15/12/2022
Demographic Questionnaire	2	15/12/2022
Letter of Invitation	2	15/12/2022
Protocol	2	15/12/2022
Recruitment poster	1	21/11/2022
Request for Permission	2	15/12/2022
Risk Assessment form	2	15/12/2022
Assessment report template	2	15/12/2022

With the Committee's best wishes for the success of this project.

Yours sincerely,

A handwritten signature in cursive script, appearing to read "C. Stocker".

Dr Claire Stocker
Deputy Chair of the Health & Life Sciences Research Ethics Committee



**‘Embracing Complexity in the Characterisation and Tracking of Neurodevelopment’
(ECCaToN)**

Participant Information Sheet

Invitation

We would like to invite you to take part in a research study forming part of a PhD project for Karin Madericova.

Before you decide if you would like to participate, take time to read the following information carefully and, if you wish, discuss it with others such as your family, friends or colleagues.

Please ask a member of the research team, whose contact details can be found at the end of this information sheet, if there is anything that is not clear or if you would like more information before you make your decision.

What is the purpose of the study?

The purpose of this study is to gain a better understanding of the difficulties experienced by adults who have been referred to assessment or support services for a suspected and/or previously diagnosed developmental condition. Some examples of developmental difficulties that people with a referral may experience include problems with attention, memory or emotional processing. Such difficulties may, in some cases, be associated with various developmental conditions, such as autism or ADHD (Attention Deficit/Hyperactivity Disorder). Currently, the way in which developmental difficulties and conditions are generally understood by professionals (e.g., clinicians or researchers) does not fully take into consideration how experiences may vary across individuals, meaning a lot of people’s needs are often unmet.

We are conducting this study as part of a larger research project, with which we are planning to characterise and track individual wellbeing across development. The overall aim is to promote a shift in the way developmental conditions and difficulties are perceived through embracing how complex and varied they can be. We are hoping that this will allow people with milder difficulties who may not receive a formal diagnosis – but whose daily living is nonetheless impacted by such difficulties – to gain better access to the services they need and potentially receive more individualised support. We are also hoping that people with more complex needs which have previously been difficult to characterise may benefit in the same way. In this first study, we are looking to recruit up to 200 participants from local services to complete a series of cognitive and mental health tests, from which we hope to obtain information on the broad range of individual strengths and difficulties that may characterise developmental disorders. Overall, we would like to gain a better understanding of the nature of various difficulties in adults, the barriers that individuals face as a result of such difficulties, and the best ways in which they can be supported.

Why have I been invited?

You are being invited to participate in this study because you have been referred to assessment or support services for suspected or previously diagnosed difficulties, and/or you have responded to an advert promoting this study.

You may take part in this study if:

- You have developmental difficulties, related to areas such as (but not limited to) learning, language or behaviour (you may or may not have received a formal diagnosis).
- You are an adult between 16 – 50 years of age.
- You are a native English speaker (English does not have to be your first language).
- You have **not** sustained traumatic brain injury/damage.
- You **do not** have a primary sensory impairment (e.g., hearing loss).
- You **do not** have pervasive intellectual disability.
- You have the capacity to provide informed consent **on your own behalf**.

What will happen to me if I take part?

If you decide to take part in the study, you will complete a series of cognitive and mental health measures. These measures are designed to be efficient and therefore should not take longer than 2 hours in total. You will be expected to attend our research unit (at Aston University) on **one** occasion only. You will complete a battery of cognitive tests on an iPad. These tests are designed to measure individual strengths and weaknesses in areas such as attention or memory. Then you will complete a mental health assessment designed to measure people's experiences of things such as low mood, anxiety or emotion regulation skills. This part of the assessment may be completed electronically or on paper.

Please note that none of the tests you will complete have been designed to elicit or cause any psychological distress, and the research team will **not** ask you to disclose any personal information. You will also not be under any time constraints, and you may take breaks in between assessments as needed. Please also note that **none** of the tests you will complete with the research team are within themselves intended to be diagnostic of any specific developmental condition.

If you have been referred to assessment at Aston University, our research and your assessment may take place on the same day – **please note** that these are separate and the tests you complete with us will be used for different purposes. If this applies to you, you may, for example, complete your assessment in the morning, and take part in our research in the afternoon, and so you may spend longer than a total of two hours in our assessment labs.

Please note that if you are attending Aston University for an assessment that you have been referred to, deciding not to take part in our study will not cause problems with your referral. Partaking in our research is completely **voluntary** and separate from any other assessments that you may have been referred to.

You will only be asked to attend and take part in our study if you are happy with the information on this form and give consent to proceed.

Do I have to take part?

No. It is up to you to decide whether or not you wish to take part.

If you do decide to participate, you will be asked to provide informed consent.

You can halt your participation in the research at any time (by telling the researcher or closing down your browser) and any data collected up to that point will not be used.

If you wish to withdraw your responses after participation, you have up to 14 days to do so. You can do this by contacting the research team (please find contact details at the bottom of this form), stating your name. After this point, every effort will be made to withdraw your data if you still wish to do so, but it may be difficult for us to remove data at certain points, depending on the stage of data analyses we are currently in.

You can find the consent form on the following page, where you will see a number of statements and be asked to indicate whether you are happy with each one of them. If there is something you are unsure about, please do not hesitate to ask a member of the research team for further information.

Will my taking part in this study be kept confidential?

Yes. A code will be attached to all the data you provide to maintain anonymity. Analysis of your data will be undertaken using coded data.

If we need to collect personal data (such as your name and contact details) we will only use this for the purposes outlined in this Participant Information Sheet – a) to provide a Debrief Form with further information about the study after you have completed it, b) to ensure that we are linking the correct responses on all our measures to the correct participant, and c) to contact you for potential follow-up research in the future.

Data collected on paper will be stored in a locked filing cabinet in a secure office space at Aston University, occupied by a member of the research team only. All electronic data will be stored in password protected files on BOX, a secure cloud storage system, compliant with Aston University data protection policy procedures, and will be accessed by the research team only.

To ensure the quality of the research Aston University, we may need to access your data to check that the data has been recorded accurately (e.g., for the purposes of audit). If this is required, your personal data will be treated as confidential by the individuals accessing your data.

Please note that we may contact you in the future for potential follow-ups (as mentioned above), which may include the reuse of data for novel ways that have not been established in the protocol of this study. Your personal data (i.e., your name and email address) will be accessed by our research team *only*, and any follow-up studies would be conducted within the same team. If you do not wish to be contacted for potential follow-ups after the completion of this study, please let a member of the research team know and your contact details will be removed.

Please note that your individual data, in *anonymised* format, may be used for other research purposes outside of our research team, with permission from the principal investigator as appropriate. *No data used, shared or published outside of the research team will ever be in any format that could be used to identify any individual.* If you do not wish for your individual anonymised data to be used for research purposes outside of our research team, please let a member of the research team know.

What happens if something is discovered during the study which requires further clinical investigation?

The investigations undertaken during this study are within themselves **not** intended to be diagnostic of any conditions or disorders, and therefore, the potential for incidental findings is very low.

The research team will not hold any information to be able to make any clinical referrals. If you do need any further support, the research team will, with your permission, signpost you to the relevant disability and support unit from which you have been recruited, who will continue to maintain your confidentiality and provide you with further relevant support for the difficulties that you may be experiencing.

If you are a student at Aston University, you may be signposted to:

1. Members of the research team (professionals experienced in cognitive disability).
2. Disability and Support Services at Aston University.
3. The Hub (Aston University Student Support Services – should you wish to discuss the study with someone independent from the research project).
4. Charities dealing with mental health and related issues (e.g., MIND Helpline at 0300 123 3393 or info@mind.org.uk).

If you are a non-Aston participant, you may be signposted to:

1. Members of the research team (professionals experienced in cognitive disability).
2. Disability and Support Services at your relevant organisation/university.
3. Student Support Services at your relevant university.
4. Charities dealing with mental health and related issues (e.g., MIND Helpline at 0300 123 3393 or info@mind.org.uk).

What are the possible benefits of taking part?

Although you may find participation in this research interesting, there may be no direct benefit to you as a result. However, we hope that you will be able to gain some insight into cognitive and emotional processing and learn about your own strengths and weaknesses within these areas of functioning. The information we gain from your results may potentially enable us to refer you to services that can offer relevant support. You will also receive a brief summary report within 7 days of completing the assessment, containing information about your strengths and weaknesses within the assessed cognitive and mental health domains. You may wish to use this report with your support team for identifying areas you may need support with or strategies for improvement. However, **please note** that none of the information that will be provided in this report is intended to be diagnostic of any particular conditions/disorders.

In the long run, we hope that the findings of this research will contribute towards a change in the way that developmental difficulties are currently understood and assessed. This could potentially allow for future advancements in the way people are diagnosed and the support they are able to access on the basis of such diagnoses.

What are the possible risks and burdens of taking part?

The following risks outlined are anticipated to be very small; we do, however, have mitigations in place should these things occur.

We will ask you questions about mood, mental health, and personal behaviours/experiences, which may, for some, cause psychological discomfort. To minimise the risk of this, we ask that you only disclose information you feel comfortable sharing. You also have the right to withdraw at any point during the study should you feel distressed/uncomfortable. If you do,

however, feel distressed after having completed the study, you may wish to speak to a member of the research team, who are all professionals experienced in cognitive disability and have the capacity to offer support (please find contact details at the bottom of this form). If you are a student at Aston University and wish to speak to someone outside of the research team, please contact The Hub (Aston Student Support Services) at thehub@aston.ac.uk or 0121 204 4007. If you are not a student at Aston University and wish to speak to somebody outside of the research team, you may consult your original support services, and/or student services at your relevant university, or ask a member of the research team to signpost you to an external party (e.g., relevant charity).

In some cases, participants may happen to disclose information that is of *significant* concern and may require action on our behalf. Please note, the research team have an *obligation* to report such incidences to external parties. For example, if we are concerned about risk to your safety or the safety of others, we may have to breach confidentiality through making a safeguarding referral. This will be done for matters of real concern *only*, and any information will be shared with relevant services *only*.

If necessary, the safeguarding referral will be made to Birmingham City Council, who have an Adult Social Care Centre dealing with possible abuse/neglect of adults with care and support needs. Alternatively, the research team have the responsibility to dial 999 in cases of real emergency or immediate danger.

Finally, we do not wish to coerce or deceive any individual taking part in our research, and for this reason you had been informed that participation is completely voluntary and given a detailed description of the study.

What will happen to the results of the study?

The results of this study may be published in scientific journals and/or presented at conferences. If the results of the study are published, your identity will remain *anonymous*.

A lay summary of the results of the study can be forwarded to you when the study has been completed. Should you wish to receive a copy, please provide your email address on the consent form, or contact a member of the research team.

We also intend to publish provisional lay summaries of our findings on our project website, where you will be able to see the progress of the study and how the data are being used. Please note that no individual identifiable data will be published, and your identity will remain *anonymous*.

The results of the study may also be shared with other researchers for future studies, with permission from the principal data holder. If individual results are shared for other research purposes not outlined in this study, individual data will be *anonymised and no data will ever be shared in any format that could be used to identify any individual*.

The results of the study will also be used in Karin Madericova's PhD thesis.

Expenses and payments

You will be reimbursed for the time you have taken to take part in our research. This will be in the form of a £10 Amazon voucher for each hour that you will spend with us.

The assessments you complete as part of our research are not intended to take longer than 2 hours in total (including breaks), and therefore, you may receive a maximum of 2 vouchers (worth £20 in total).

Aston students taking part in the study may opt to participate in exchange for course credit OR Amazon vouchers. You may receive up to 24 course credits for your participation (at a standard rate of 1 credit per 5 minutes), as the study should take a total of 120 minutes (2 hours).

Any reimbursements in the form of vouchers will be covered by the allocated project funds (provided by Aston University, from research accounts help by the supervisor investigator).

You may also claim for travel/parking expenses from Aston University. To this end, you will need to complete an 'Expenses Claim Form' and submit it via the University Finance Office.

Who is funding the research?

The project and the doctoral researcher's PhD studentship are being funded by Aston University and UK Research and Innovation (UKRI), both contributing 50% to the sponsorship.

Who is organising this study and how is my data being used?

Aston University is organising this study and acting as data controller for the study. Research data will be used only for the purposes of the study or related uses identified in this Information Sheet or Appendix A.

Who has reviewed the study?

This study was given a favorable ethical opinion by the Aston University Research Ethics Committee.

What if I have a concern about my participation in the study?

If you have any concerns about your participation in this study, please speak to the research team and they will do their best to answer your questions. Contact details can be found at the end of this information sheet.

If the research team are unable to address your concerns or you wish to make a complaint about how the study is being conducted you should contact the Aston University Research Integrity Office at research_governance@aston.ac.uk or via the University switchboard on +44 (0)121 204 3000.

Research Team

Principal Researcher:

Name: Karin Madericova

Email: 170096872@aston.ac.uk

Principal Supervisor:

Name: Prof Joel B Talcott

Email: j.b.talcott@aston.ac.uk

Contact Telephone Number: 0121 204 4083

Aston University takes its obligations under data and privacy law seriously and complies with the Data Protection Act 2018 (“DPA”) and the General Data Protection Regulation (EU) 2016/679 as retained in UK law by the Data Protection, Privacy and Electronic Communications (Amendments etc) (EU Exit) Regulations 2019 (“the UK GDPR”). Aston University is the sponsor for this study based in the United Kingdom. We will be using information from you in order to undertake this study. Aston University will process your personal data in order to register you as a participant and to manage your participation in the study. It will process your personal data on the grounds that it is necessary for the performance of a task carried out in the public interest (GDPR Article 6(1)(e)). Aston University may process special categories of data about you which includes details about your health. Aston University will process this data on the grounds that it is necessary for statistical or research purposes (GDPR Article 9(2)(j)). Aston University will keep identifiable information about you for 6 years after the study has finished.

Your rights to access, change or move your information are limited, as we need to manage your information in specific ways in order for the research to be reliable and accurate. If you withdraw from the study, we will keep the information about you that we have already obtained. To safeguard your rights, we will use the minimum personally identifiable information possible.

You can find out more about how we use your information at <https://www.aston.ac.uk/about/statutes-ordinances-regulations/publication-scheme/policies-regulations/data-protection> or by contacting our Data Protection Officer at dp_officer@aston.ac.uk.

If you wish to raise a complaint on how we have handled your personal data, you can contact our Data Protection Officer who will investigate the matter. If you are not satisfied with our response or believe we are processing your personal data in a way that is not lawful you can complain to the Information Commissioner’s Office (ICO).

When you agree to take part in a research study, the information about you may be provided to researchers running other research studies in this organisation and in other organisations. These organisations may be universities, NHS organisations or companies involved in health and care research in this country or abroad.

This information will not identify you and will not be combined with other information in a way that could identify you. The information will only be used for the purpose of research and cannot be used to contact you.

Appendix D. Consent Form



‘Embracing Complexity in the Characterisation and Tracking of Neurodevelopment’ (ECCaToN)

Consent Form

Name of Chief Investigator: Karin Madericova

Please initial boxes

1.	I confirm that I have read and understand the Participant Information Sheet [Version 2, 15/12/2022] for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.	
2.	I understand that my participation is voluntary and that I am free to withdraw at any time during the study, without giving a reason and without my legal rights being affected.	
3.	I understand that I am able to withdraw my data up to 14 days after taking part in the study by contacting the research team, after this time my data will be anonymised, and I will no longer be able to withdraw.	
4.	I agree to my personal data and data relating to me collected during the study being processed as described in the Participant Information Sheet.	
5.	I understand that if during the study I tell the research team something that causes them to have concerns in relation to my health and/or welfare, they may need to breach my confidentiality.	
6.	I agree to my anonymised data being used by research teams for future research.	
7.	I agree to my personal data being processed for the purposes of inviting me to participate in future research projects. I understand that I may opt out of receiving these invitations at any time.	
8.	I agree to take part in this study.	

Name of participant

Date

Signature

Name of Person receiving
consent.

Date

Signature

Appendix E. Debrief Form

‘Embracing Complexity in the Characterisation and Tracking of Neurodevelopment’ (ECCaToN)

Thank you for taking part in our study, which will form a part of a larger project to identify and characterise individual differences in neurodiverse conditions. We are interested in characterising the complexity of the cognitive, affective and emotional processing difficulties underlying these conditions in adults. We have created this specific assessment with the aim of increasing the breadth of cognitive measures used with adults, and to capture the traits of mental health in people with various experiences of neurodevelopmental conditions.

By taking part in our study, you have helped to provide data which will help us to track, predict and evaluate various dimensions and clusters of strengths and difficulties. We will use these data to study neurodevelopment from a broader perspective, moving away from the traditional categorical views and more towards individually tailored approaches.

We will provide you with information about your strengths and difficulties in various areas of functioning (i.e., cognitive and mental health) through a brief summary report of your profile, available within 7 working days of this appointment. If you wish, you can share this report with your support team. *Please note*, however, that this report is independent of and does not inform any official diagnosis or assessment you may have completed or received.

We will publish interim reports on our project website (<https://170096872.wixsite.com/eccaton-project>), updating the progress of the study. Here, you will be able to obtain key information regarding our study, such as the range and number of participants, and descriptions about how the data we have collected are being used. *Please note that no data will ever be published in any format that could be used to identify any individual.*

We may contact you in the future for potential follow ups, which may include the reuse of data for novel ways that have not been established in the protocol of the current study. Please note that your data may be used (in *anonymised* format) for other research purposes, with permission from the principal investigator as appropriate. *No data shared outside of the research team will ever be shared in any format that could be used to identify any individual.* If you are not happy to be contacted and/or do not wish for us to use your data for any purposes not outlined in this study, please let a member of the research team know.

If you have questions regarding any aspect of the study, please feel free to contact a member of the research team using the contact information below. If you are a student at Aston University and you wish to consult with someone independent of this study, you may contact The Hub (at thehub@aston.ac.uk), where you will be referred to an appropriate University Support Service. Alternatively, if you are not an Aston student, you may contact the Disability and Support Services at your relevant organisation, who will have liaised with the research team and have information regarding the study. If you are concerned about your mental health or experiencing any difficulties unrelated to this study, you may wish to contact the MIND Helpline (a mental health charity) at 0300 123 3393 or info@mind.org.uk, or ask a member of the research team to signpost you to other relevant external parties/charities. *Please note* we are unable to provide clinical/GP referrals as none of our assessments have been designed to be diagnostic of any conditions.

Principal Researcher: Karin Madericova (170096872@aston.ac.uk)

Principal Supervisor: Prof Joel B Talcott (j.b.talcott@aston.ac.uk)

Research Assessment Report

Name:

Date of Birth:

Date of Assessment:

Age at Assessment:

The purpose of this report is to provide an account of your cognitive ability across the range of measures we administered. This will be helpful in framing your relative strengths and weaknesses in the domains tested. **In the table below, your data are presented as percentile scores, which indicates that percentage of the population of persons of the same age who would score equal to or lower to the score which you have achieved.**

Please note that the data provided are indicative of your strengths and weaknesses identified within **a research context only** and are independent of any diagnostic or screening process you may have completed or are currently seeking.

NIH Cognitive toolbox

The NIH Cognitive toolbox consists of a number of individually administered, computer-based measures that tap cognitive skills across a range of domains.

Scale	Domain Assessed	Raw Score	Standardised Score	Percentile Score
Flanker Inhibitory Control and Attention Test	Inhibition; ability to deal with environmental stimulation			
Dimensional Change Card Sort Test	Executive function, Goal-oriented behaviour; ability to plan, organise and monitor behaviour			
Picture Sequence Memory Test	Episodic memory; acquisition, storage and			

	retrieval of new information			
List Sorting Working Memory Test	Working memory; the ability to store information in the mind			
Pattern Comparison Processing Speed Test	Processing speed; how much information can be processed within a certain amount of time			
Picture Vocabulary Test	Receptive vocabulary; understanding the meaning of words			
Oral Reading Recognition Test	Decoding skills and crystallised abilities			

The average range for a Standardised Score is 85 – 115, with the average at 100 relative to chronological age.

In accordance with the General Data Protection Regulation (GDPR), this report will be stored securely. The electronic copy will be password protected and will be accessible to the individual, and authorised professionals within Aston University only. The electronic copy will be stored for five years from the date of the testing.

Appendix G. The Extended Strengths and Weaknesses Assessment of Normal Behaviour (E-SWAN; Alexander et al., 2020)

ADHD Rating Scale

For each item, check the column that best describes you over the past six months.	Far below average	Below average	Slightly below average	About average	Slightly above average	Above average	Far above average
1. I give close attention to detail and avoid careless mistakes							
2. I sustain attention on tasks or activities							
3. I listen when spoken to directly							
4. I follow through on instructions and finish my work and chores							
5. I organize tasks and activities							
6. I engage in tasks that require sustained mental effort							
7. I keep track of things necessary for activities (don't lose them)							
8. I ignore extraneous (irrelevant) stimuli (things)							
9. I remember daily activities							
10. I sit still (control movement of my hands/feet or control squirming)							
11. I stay seated (when required by rules or social conventions)							
12. I modulate my motor activity (inhibit inappropriate physical activity, e.g., running)							
13. I keep my noise levels reasonable							
14. I settle down and rest (control constant activity)							
15. I modulate my verbal activity (control excessive talking)							
16. I reflect on questions (control blurting out answers)							

17. I await my turn (stand in line and take turns)							
18. I enter into conversation without interrupting or intruding							

Depression Rating Scale

Individuals differ in their abilities to handle emotions and behave appropriately in several situations. For each item listed below, please rate how well you think you handle emotions and behave compared to other people of your age.

Compared to other people your age, in recent weeks, how well do you:	Far below average	Below average	Slightly below average	About average	Slightly above average	Above average	Far above average
1A. Limit feelings of sadness							
1B. Limit feelings of irritability							
2A. Show interest in performing activities							
2B. Enjoy activities							
3A. Maintain stable weight							
3B. Maintain appropriate appetite							
4. Sleep for an appropriate amount of time							
5. Maintain your typical pace of movement							
6. Have energy to perform activities							
7. Feel self-worth							
8A. Concentrate							
8B. Make decisions							
9. Feel life is worth living							

Panic Disorder Rating Scale

Individuals differ in their abilities to handle emotions and behave appropriately in several situations. For each item listed below, please rate how well you think you handle emotions and behave compared to other people of your age.

Compared to other people your age, most of the time, how well do you:	Far below average	Below average	Slightly below average	About average	Slightly above average	Above average	Far above average
1. Avoid or limit moments of intense fear or discomfort that occur 'out of the blue'							
2. Keep the mind free from worries about experiencing intense fear or discomfort							
3. Return to typical behaviours/activities after experiencing a moment of intense fear or discomfort							

Compared to other people your age, <u>during moments of intense fear or discomfort</u>, how well do you:	Far below average	Below average	Slightly below average	About average	Slightly above average	Above average	Far above average
1. Keep a regular heartbeat							
2. Keep dry and cool							
3. Keep hands steady							
4. Breathe at your typical speed							
5. Breathe without choking sensations							
6. Remain without chest discomfort or pain							
7. Keep a settled stomach							
8. Keep clear-headed							

9. Maintain feelings of a comfortable body temperature							
10. Maintain feelings in your hands or feet							
11. Maintain awareness of yourself							
12. Feel in control							
13. Feel physically safe							

Social Anxiety Rating Scale

Individuals differ in their abilities to handle emotions and behave appropriately in several situations. For each item listed below, please rate how well you think you handle emotions and behave compared to other people of your age.

Compared to other people your age, how well do you:	Far below average	Below average	Slightly below average	About average	Slightly above average	Above average	Far above average
1. Tolerate feelings of anxiety in social situations							
2. Handle fears of appearing anxious in social situations							
3. Stay relaxed in social situations							
4A. Seek out social activities							
4B. Enjoy participating in social activities							
5. Control anxiety in challenging social situations							

Appendix H. The Emotion Regulation Skills Questionnaire (ERSQ; Grant et al., 2018)

2. Dealing with emotions: In the last week ...		not at all	rarely	some- times	often	almost always
1.)	... I was able to consciously pay attention to my feelings.	O ₀	O ₁	O ₂	O ₃	O ₄
2.)	... I could consciously bring about positive feelings.	O ₀	O ₁	O ₂	O ₃	O ₄
3.)	... I understood my emotional reactions.	O ₀	O ₁	O ₂	O ₃	O ₄
4.)	... I could endure my negative feelings.	O ₀	O ₁	O ₂	O ₃	O ₄
5.)	... I was able to accept my negative feelings.	O ₀	O ₁	O ₂	O ₃	O ₄
6.)	... I could have labeled my feelings.	O ₀	O ₁	O ₂	O ₃	O ₄
7.)	... I had a clear physical perception of my feelings.	O ₀	O ₁	O ₂	O ₃	O ₄
8.)	... I did what I wanted to do, even if I had to face negative feelings on the way.	O ₀	O ₁	O ₂	O ₃	O ₄
9.)	... I tried to reassure myself during distressing situations.	O ₀	O ₁	O ₂	O ₃	O ₄
10.)	... I was able to influence my negative feelings.	O ₀	O ₁	O ₂	O ₃	O ₄
11.)	... I knew what my feelings meant.	O ₀	O ₁	O ₂	O ₃	O ₄
12.)	... I could focus on my negative emotions if necessary.	O ₀	O ₁	O ₂	O ₃	O ₄
13.)	... I knew what emotions I was feeling in the moment.	O ₀	O ₁	O ₂	O ₃	O ₄
14.)	... I consciously noticed when my body reacted towards emotionally charged situations in a particular way.	O ₀	O ₁	O ₂	O ₃	O ₄
15.)	... I tried to cheer myself up in emotionally distressing situations.	O ₀	O ₁	O ₂	O ₃	O ₄
16.)	... I did what I intended to do despite my negative feelings.	O ₀	O ₁	O ₂	O ₃	O ₄
17.)	... I was OK with my feelings, even if they were negative .	O	O ₁	O ₂	O ₃	O ₄
18.)	... I was certain that I would be able to tolerate even intense negative feelings.	O ₀	O ₁	O ₂	O ₃	O ₄
19.)	... I was able to experience my feelings consciously.	O ₀	O ₁	O ₂	O ₃	O ₄
20.)	... I was aware of why I felt the way I felt.	O ₀	O ₁	O ₂	O ₃	O ₄
21.)	... I knew that I was able to influence my feelings.	O ₀	O ₁	O ₂	O ₃	O ₄
22.)	... I pursued goals that were important to me, even if I thought that doing so would trigger or intensify negative feelings.	O ₀	O ₁	O ₂	O ₃	O ₄
23.)	... I was able to experience my negative feelings without immediately trying to fight them off.	O ₀	O ₁	O ₂	O ₃	O ₄
24.)	... my physical sensations were a good indication of how I was feeling.	O ₀	O ₁	O ₂	O ₃	O ₄
25.)	... I was clear about what emotions I was experiencing.	O ₀	O ₁	O ₂	O ₃	O ₄
26.)	... I could tolerate my negative feelings.	O ₀	O ₁	O ₂	O ₃	O ₄
27.)	... I supported myself in emotionally distressing situations.	O ₀	O ₁	O ₂	O ₃	O ₄

Appendix I. The Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997)

Strengths and Difficulties Questionnaire

S¹⁷⁺

For each item, please mark the box for Not True, Somewhat True or Certainly True. It would help us if you answered all items as best you can even if you are not absolutely certain. Please give your answers on the basis of how things have been for you over the last six months.

Your Name

Male/Female

Date of Birth.....

	Not True	Somewhat True	Certainly True
I try to be nice to other people. I care about their feelings	☐	☐	☐
I am restless, I find it hard to sit down for long	☐	☐	☐
I get a lot of headaches, stomach-aches or sickness	☐	☐	☐
I usually share with others, for example food or drink	☐	☐	☐
I get very angry and often lose my temper	☐	☐	☐
I would rather be alone than with other people	☐	☐	☐
I am generally willing to do what other people want	☐	☐	☐
I worry a lot	☐	☐	☐
I am helpful if someone is hurt, upset or feeling ill	☐	☐	☐
I am constantly fidgeting or squirming	☐	☐	☐
I have at least one good friend	☐	☐	☐
I fight a lot. I can make other people do what I want	☐	☐	☐
I am often unhappy, depressed or tearful	☐	☐	☐
Other people generally like me	☐	☐	☐
I am easily distracted, I find it difficult to concentrate	☐	☐	☐
I am nervous in new situations. I easily lose confidence	☐	☐	☐
I am kind to children	☐	☐	☐
I am often accused of lying or cheating	☐	☐	☐
Other people pick on me or bully me	☐	☐	☐
I often offer to help others (family members, friends, colleagues)	☐	☐	☐
I think before I do things	☐	☐	☐
I take things that are not mine from home, work or elsewhere	☐	☐	☐
I get along better with older people than with people of my own age	☐	☐	☐
I have many fears, I am easily scared	☐	☐	☐
I finish the work I'm doing. My attention is good	☐	☐	☐

Do you have any other comments or concerns?

Overall, do you think that you have difficulties in one or more of the following areas:
emotions, concentration, behaviour or being able to get along with other people?

No	Yes- minor difficulties	Yes- definite difficulties	Yes- severe difficulties
☐	☐	☐	☐

If you have answered "Yes", please answer the following questions about these difficulties:

- How long have these difficulties been present?

Less than a month	1-5 months	6-12 months	Over a year
☐	☐	☐	☐

- Do the difficulties upset or distress you?

Not at all	Only a little	Quite a lot	A great deal
☐	☐	☐	☐

- Do the difficulties interfere with your everyday life in the following areas?

	Not at all	Only a little	Quite a lot	A great deal
getting along with the people you are closest to (e.g. family, partner)	☐	☐	☐	☐
making and keeping friends	☐	☐	☐	☐
work or study	☐	☐	☐	☐
hobbies, sports or other leisure activities	☐	☐	☐	☐

- Do the difficulties make it harder for those around you (family, friends, etc.)?

Not at all	Only a little	Quite a lot	A great deal
☐	☐	☐	☐

Your Signature

Today's Date

Thank you very much for your help

© Robert Goodman, 2009

Supplementary Materials 3 (Chapters 4-5: Transdiagnostic Study Results)

Appendix A. Descriptive Statistics and Preliminary Analyses (Chapters 4-5)

Overall Descriptive Statistics (Full Sample)

Table 1. Mean standard scores across cognitive tasks for the whole sample (n = 175).

Task	<i>M</i>	<i>SD</i>
Flanker Inhibitory Control And Attention Test	82.7	17.7
Dimensional Change Card Sort Test	86.9	20.5
Picture Sequence Memory Test	100.0	14.9
List Sorting Working Memory Test	103.1	15.0
Pattern Comparison Processing Speed Test	97.9	23.5
Picture Vocabulary Test	96.8	13.6
Oral Reading Recognition Test	120.3	15.3
Fluid Cognition Composite	91.1	18.8
Crystallised Cognition Composite	109.3	13.2
Cognitive Function Composite	100.1	15.5

Note. *M* = Mean. *SD* = Standard Deviation. Age-corrected standard scores were generated via the NIH Toolbox; this score compares individual performance to people of the same age within the NIH Toolbox normative sample (based on the population of the United States). A score of 100 indicates that the participant performed at the population average level for their age. Scores 15 points above or below 100 indicate that the individual's performance was 1 standard deviation (SD) away from the average.

Table 2. Mean scores across mental health measures for the whole sample ($n = 175$).

Measure	<i>M</i>	<i>SD</i>
ERSQ		
Awareness	2.6	0.7
Sensations	2.5	0.8
Clarity	2.5	0.9
Understanding	2.5	0.9
Acceptance	2.3	0.8
Tolerance	2.5	0.8
Confrontation	2.4	0.8
Self-Support	2.6	0.8
Modification	2.2	0.8
Total Score	2.5	0.6
SDQ		
Emotional Difficulties	5.6	2.5
Conduct Problems	2.4	1.6
Hyperactivity	6.1	2.2
Peer Problems	3.0	1.7
Prosocial Skills	8.6	1.5
Externalising	8.5	3.0
Internalising	8.7	3.6
Total Difficulties	17.1	5.3
Impact	3.1	2.5
E-SWAN		
ADHD	4.0	1.0
Depression	4.3	1.0
Panic Disorder	4.3	1.1
Social Anxiety	4.3	1.3

Note. *M* = Mean. *SD* = Standard Deviation. *ADHD* = Attention Deficit Hyperactivity Disorder. The Emotion Regulation Skills Questionnaire (ERSQ) is a 5-point scale ranging from 0 – ‘Not At All’ to 4 – ‘Almost Always’. A higher score indicates better emotion regulation skills. The Strengths and Difficulties Questionnaire (SDQ) is a 3-point scale ranging from 0 – ‘Not True’ to 2 – ‘Certainly True’. The Emotional Difficulties, Conduct Problems, Hyperactivity, Peer Problems and Prosocial subscales each consist of 5 items and are scored out of 10. The Externalising scale is the sum of the Conduct and Hyperactivity scales, while the Internalising scale is the sum of the Emotional and Peer Problems subscales. The Total Difficulties scale is the sum of all subscales except the Prosocial scale. The Impact score represents overall distress and impairment as a result of any difficulties experienced. A higher score on any subscale except the Prosocial scale represents a higher level of self-reported difficulties. The Extended Strengths and Weaknesses Assessment of Normal Behaviour (E-SWAN) is a 7-point scale. The E-SWAN was reverse scored from 7 – ‘Far Below Average’ to 1 – ‘Far Above Average’. A score of 4 or above represents above average weaknesses in each area assessed, i.e., a higher score is indicative of more difficulties experienced.

Preliminary Analyses (Broad Diagnostic Groupings)

Table 3. Mean scores and one-way ANOVA results for group differences on cognitive tasks across broad diagnostic groupings ($n = 175$).

	ND ($n = 49$)	Suspected ND ($n = 108$)	No ND ($n = 18$)			
Task	$M (SD)$	$M (SD)$	$M (SD)$	F	$df2$	p
Flanker Inhibitory Control and Attention Test	83.2 (20.4)	82.1 (17.2)	84.9 (12.2)	0.38	51	.686
Dimensional Change Card Sort Test	85.8 (21.6)	87.3 (20.8)	87.6 (15.5)	0.10	50	.902
Picture Sequence Memory Test	102.1 (12.8)	99.5 (15.8)	96.8 (14.7)	1.14	46	.327
List Sorting Working Memory Test	106.5 (16.1)	101.9 (14.3)	101.2 (15.3)	1.62	44	.209
Pattern Comparison Processing Speed Test	96.6 (27.0)	98.4 (21.6)	98.3 (25.2)	0.08	42	.922
Picture Vocabulary Test	97.8 (15.3)	96.3 (13.0)	97.2 (12.8)	0.17	45	.844
Oral Reading Recognition Test	120.4 (16.3)	120.6 (14.8)	117.9 (16.0)	0.22	44	.804
Fluid Cognition Composite Score	92.2 (20.9)	90.6 (18.2)	90.7 (16.9)	0.12	45	.891
Crystallised Cognition Composite Score	109.8 (14.3)	109.3 (12.7)	108.1 (13.4)	0.11	44	.897
Cognitive Function Composite Score	100.9 (17.8)	99.9 (14.7)	99.1 (14.2)	0.11	45	.900

Note. M = Mean. SD = Standard Deviation. df = Degrees of Freedom ($df1 = 2$ across all tasks). ND = Neurodiverse Diagnosis. For more information on the NIH Toolbox, please refer to Table 1.

Table 4. Mean scores and one-way ANOVA results for group differences on mental health measures across broad diagnostic groupings ($n = 175$).

	ND ($n = 49$)	Suspected ND ($n = 108$)	No ND ($n = 18$)			
Measure	$M (SD)$	$M (SD)$	$M (SD)$	F	$df2$	p
ERSQ						
Awareness	2.5 (0.8)	2.6 (0.7)	3.0 (0.6)	4.42	47	.017
Sensations	2.5 (0.8)	2.4 (0.8)	2.9 (0.8)	2.70	45	.078
Clarity	2.6 (0.8)	2.4 (0.9)	2.7 (0.7)	1.25	48	.296
Understanding	2.6 (0.9)	2.5 (0.9)	2.8 (0.7)	1.50	47	.234
Acceptance	2.3 (0.8)	2.3 (0.8)	2.7 (0.7)	3.42	47	.041
Tolerance	2.4 (0.8)	2.5 (0.8)	2.6 (0.6)	0.79	51	.461
Confrontation	2.2 (0.8)	2.4 (0.8)	2.9 (0.6)	8.27	52	<.001
Self-Support	2.5 (0.7)	2.6 (0.8)	2.8 (0.8)	0.98	45	.383
Modification	2.2 (0.9)	2.2 (0.8)	2.5 (0.7)	0.98	46	.385
Total Score	2.4 (0.6)	2.4 (0.5)	2.8 (0.5)	3.53	45	.038
SDQ						
Emotional Difficulties	6.0 (2.7)	5.7 (2.4)	4.2 (2.5)	3.41	44	.042
Conduct Problems	2.3 (1.6)	2.5 (1.6)	2.1 (1.3)	0.57	48	.570
Hyperactivity	6.5 (2.1)	6.2 (2.1)	4.2 (2.6)	5.88	43	.006
Peer Problems	3.3 (1.8)	3.0 (1.7)	2.6 (1.5)	1.22	46	.304
Prosocial Skills	8.4 (1.4)	8.6 (1.6)	9.1 (0.9)	3.21	57	.048
Externalising	8.8 (2.9)	8.7 (2.9)	6.3 (3.3)	4.13	43	.023
Internalising	9.3 (3.9)	8.7 (3.4)	6.8 (3.4)	3.23	45	.049
Total Difficulties	18.1 (5.4)	17.3 (5.0)	13.2 (5.3)	5.72	44	.006
Impact	3.6 (2.5)	3.2 (2.4)	1.1 (1.9)	10.48	49	<.001
E-SWAN						
ADHD	4.4 (0.9)	3.9 (0.9)	3.1 (0.9)	13.78	44	<.001
Depression	4.5 (0.9)	4.3 (1.0)	3.3 (1.2)	6.61	43	.003
Panic Disorder	4.5 (1.1)	4.4 (0.9)	3.4 (1.3)	5.35	41	.009
Social Anxiety	4.4 (1.3)	4.4 (1.2)	3.7 (1.4)	2.00	43	.147

Note. M = Mean. SD = Standard Deviation. df = Degrees of Freedom ($df1 = 2$ across all subscales). ND = Neurodiverse Diagnosis. $ADHD$ = Attention Deficit Hyperactivity Disorder. ERSQ = Emotion Regulation Skills Questionnaire. SDQ = Strengths and Difficulties Questionnaire. E-SWAN = Extended Strengths and Weaknesses Assessment of Normal Behaviour. Significant group differences are highlighted in bold. For more information on the mental health measures, please refer to Table 2.

A one-way analysis of variance (ANOVA) revealed no significant differences overall across the three diagnostic groups on any of the cognitive tasks (see Table 3). However, significant between-group differences were found on three of the Emotion Regulation Skills Questionnaire (ERSQ) subscales (awareness, acceptance and confrontation), as well as the total emotion regulation score (as shown in Table 4). Post-hoc comparisons using the Games-Howell test indicated that the Neurodiverse Diagnosis (ND) group displayed significantly lower Emotional Awareness than the No ND group ($t(39.8) = -2.66, p = .029$), while the Suspected ND group

also scored significantly lower than the No ND group on the same subscale ($t(25.5) = -2.85, p = .023$), with a mean difference of -0.47 and -0.44, respectively. The Suspected ND group also reported significantly lower Acceptance scores than the No ND group ($t(25.4) = -2.63, p = .037$), with a mean difference of -0.46. Further, post-hoc results revealed that the ND group scored significantly lower than the No ND group on the Confrontation subscale ($t(44.1) = -3.98, p < .001$), with the Suspected ND group also displaying significantly lower Confrontation than the No ND group ($t(31.3) = -3.29, p = .007$), with a mean difference of -0.71 and -0.52, respectively. Finally, the Suspected ND group displayed a significantly lower total score than the No ND group ($t(23.6) = -2.61, p = .039$), with a mean difference of -0.35, indicating weaker overall emotion regulation skills.

Further, a one-way ANOVA revealed significant group differences on seven subscales of the Strengths and Difficulties Questionnaire (SDQ) – Emotional Difficulties, Hyperactivity, Prosocial Skills, Externalising and Internalising Problems, Total Difficulties and Impact of Difficulties (see Table 4). Post-hoc analyses showed that the ND group displayed significantly more self-reported Emotional Difficulties than the No ND group ($t(32.6) = 2.56, p = .039$), as well as reporting significantly more Hyperactive traits ($t(25.8) = 3.43, p = .006$), with a mean difference of 1.78 and 2.31, respectively. The Suspected ND group also displayed significantly more Hyperactivity than the No ND group ($t(21) = 3.12, p = .014$), with a mean difference of 1.98.

Interestingly, the Games-Howell post-hoc test did not reveal any significant group differences on the Prosocial subscale; however, the difference between the ND and No ND group approached significance, with the ND group reporting relatively fewer Prosocial skills ($t(45.8) = -2.38, p = .055$), with a mean difference of -0.68. Additionally, the ND ($t(27) = 2.77, p = .026$) and Suspected ND ($t(21.5) = 2.78, p = .029$) groups reported significantly more Externalising Problems than the No ND group, with a mean difference of 2.46 and 2.32, respectively. The ND group also displayed significantly more Internalising Problems than the No ND group ($t(34.5) = 2.54, p = .041$), with a mean difference of 2.45. Post-hoc comparisons also indicated significant group differences in Total Difficulties, with the ND ($t(30.5) = 3.33, p = .006$), and Suspected ND ($t(22.3) = 3.08, p = .014$) groups reporting significantly more difficulties than the No ND group, with a mean difference of 4.91 and 4.16, respectively. Finally, post-hoc analyses revealed significant group differences on the Impact of Difficulties subscale, with the ND group ($t(41) = 4.34, p < .001$) and Suspected ND group ($t(27.1) = 4.11,$

$p < .001$) reporting significantly higher impact than the No ND group, with a mean difference of 2.48 and 2.05.

Lastly, a one-way ANOVA also indicated significant group differences on the ADHD, Depression and Panic Disorder subscales of the Extended Strengths and Weaknesses Assessment of Normal Behaviour (E-SWAN) questionnaire (see Table 4). Post-hoc comparisons revealed that the ND group reported significantly more ADHD symptoms than the Suspected ND group ($t(89.1) = 3.26, p = .004$) and the No ND group ($t(30.9) = 5.18, p < .001$), with a mean difference of 0.52 and 1.33, respectively. To this end, the Suspected ND group also scored significantly higher than the No ND group on the ADHD subscale ($t(22.7) = 3.43, p = .006$), with a mean difference of 0.81, indicating more self-reported ADHD symptoms. Further, post-hoc results showed that the ND group ($t(23.8) = 3.66, p = .003$) and Suspected ND group ($t(20.8) = 3.24, p = .011$) reported significantly more Depression symptoms than the No ND group, with a mean difference of 1.15 and 0.98, respectively. Finally, significant group differences were also observed in Panic Disorder scores, with the ND group ($t(26.6) = 3.26, p = .008$) and the Suspected ND group ($t(20.2) = 3.03, p = .017$) reporting significantly more symptoms than the No ND group, with a mean difference of 1.10 and 0.95, respectively.

Preliminary Cluster Analyses

A *k*-means cluster analysis was conducted to identify distinct data-driven groups based on strengths and weaknesses in cognitive and mental health functioning across measures. For the purposes of this analysis, the E-SWAN and several subscales of the SDQ (Emotional Difficulties, Conduct Problems, Hyperactivity, Peer Problems and Impact) were reverse scored to ensure that all scores were on the same scale. Therefore, in the following results, higher scores indicate more adaptive functioning (or fewer difficulties) across all measures, with the aim to simplify visual inspection of comparisons between clusters. Further, any composite scores – i.e., the Fluid Cognition, Crystallised Cognition and Cognitive Function composites of the NIH Cognitive Toolbox, along with the Total Score of the ERSQ, and the Total Difficulties, Externalising and Internalising subscales of the SDQ – were excluded from the analysis. This is because the inclusion of composite scores could result in the misleading increase of homogeneity between and/or within clusters due to the inherent correlation between individual measures and their respective composite variables. All data were standardised (i.e.,

converted to *z*-scores based on the sample's mean and standard deviation) when entered into the cluster analysis.

Clusters Derived from Cognitive Data

The initial cluster analysis was performed across the entire sample using cognitive variables only, without assumptions based on any diagnostic groupings. Here, a three-cluster structure was suggested by *k*-means clustering as the optimal solution using the gap statistic. The clusters consisted of 68, 51 and 56 participants, respectively. As can be seen in Table 5, individuals with a formal ND were most frequently placed in Cluster 1, (capturing 42.9% of all individuals with a formal ND), followed by Cluster 2 (30.6%) and Cluster 3 (26.5%). Participants with a suspected ND also most frequently appeared in Cluster 1 (representing 37% of all individuals with suspected ND), followed by Cluster 3 (34.3%) and Cluster 2 (28.7%). Finally, participants with no ND were most frequent in Cluster 1 (representing 38.9% of all individuals with no ND), followed by Cluster 3 (33.3%) and Cluster 2 (27.8%). Overlaps were observed across all three clusters, as no diagnostic group (i.e., ND, Suspected ND or No ND) was fully represented by a single cluster, and no individual cluster was fully composed of a single diagnostic group. The prevalence of diagnostic groups within individual clusters (derived from cognitive data only) is presented in Table 5.

Further, regarding the sensitivity of individual clusters in capturing primary diagnosis type, Cluster 2 captured the highest proportion of individuals with ADHD (55% of all individuals with this diagnosis), followed by Cluster 1 (25%) and Cluster 3 (20%). Autistic individuals were most frequently captured by Cluster 1 (57.1%), followed by Cluster 3 (28.6%) and Cluster 2 (14.3%). Individuals with Dyslexia were most frequently found in Cluster 1 (capturing 42.9% of participants with this diagnosis), followed by Cluster 3 (35.7%) and Cluster 2 (21.4%). Finally, individuals with Intellectual Disability were captured by Clusters 1 and 3 only, with 2/3 of these participants being placed in Cluster 1. These results indicate a high overlap in the distribution of primary diagnosis type across all clusters. The remaining (less prevalent) types of primary diagnoses are presented in Table 5.

Table 5. Frequency of diagnostic groups and types of primary diagnoses within individual clusters derived from cognitive data only ($n = 175$).

	Cluster 1 ($n = 68$)	Cluster 2 ($n = 51$)	Cluster 3 ($n = 56$)
Group	n (%)	n (%)	n (%)
Diagnostic Group			
ND	21 (30.9)	15 (29.4)	13 (23.2)
Suspected ND	40 (58.8)	31 (60.8)	37 (66.1)
No ND	7 (10.3)	5 (9.8)	6 (10.7)
Type of Diagnosis			
n	21	15	13
ADHD	5 (23.8)	11 (73.3)	4 (30.8)
Autism	4 (19.0)	1 (6.7)	2 (15.4)
Dyslexia	6 (28.6)	3 (20.0)	5 (38.5)
Dyspraxia	1 (4.8)	-	-
ID	2 (9.5)	-	1 (7.7)
SLI	1 (4.8)	-	-
TS	1 (4.8)	-	-
PPD	-	-	1 (7.7)
SLD	1 (4.8)	-	-

Note. *ND* = Neurodiverse Diagnosis. *ADHD* = Attention Deficit Hyperactivity Disorder. *ID* = Intellectual Disability. *SLI* = Specific Language Impairment. *TS* = Tourette Syndrome. *PPD* = Perceptual Processing Difficulty. *SLD* = Specific Learning Difficulty (but not specified). The frequency of primary diagnosis type was calculated for individual clusters from the total number of participants with a formal ND found within each cluster.

ANOVA & Post-Hoc Results for Clusters Derived from Cognitive Data

Table 6. Mean standard scores and one-way ANOVA results for group differences on cognitive tasks for clusters derived from cognitive data only ($n = 175$).

	Cluster 1 ($n = 68$)	Cluster 2 ($n = 51$)	Cluster 3 ($n = 56$)			
Task	$M (SD)$	$M (SD)$	$M (SD)$	F	$df2$	p
Flanker Inhibitory Control and Attention Test	69.2 (11.5)	97.3 (15.2)	85.9 (13.6)	67.7	105	<.001
Dimensional Change Card Sort Test	67.3 (9.8)	104.6 (14.7)	94.6 (13.7)	159.7	101	<.001
Picture Sequence Memory Test	96.2 (14.6)	108.4 (15.2)	96.8 (11.8)	11.8	110	<.001
List Sorting Working Memory Test	101.0 (14.0)	114.7 (10.9)	95.1 (13.0)	38.9	114	<.001
Pattern Comparison Processing Speed Test	76.9 (16.5)	109.1 (17.5)	113.1 (15.3)	92.7	110	<.001
Picture Vocabulary Test	94.1 (11.7)	109.0 (11.8)	89.1 (9.3)	46.5	110	<.001
Oral Reading Recognition Test	117.9 (13.2)	131.7 (11.9)	112.7 (14.5)	31.5	112	<.001

Note. M = Mean. SD = Standard Deviation. df = Degrees of Freedom ($df1 = 2$ across all tasks).

A one-way ANOVA revealed significant group differences between clusters on all cognitive tasks (as shown in Table 6). Post-hoc comparisons using the Games-Howell test indicated that Cluster 1 scored significantly lower than Cluster 2 ($t(89.8) = -11.0, p < .001$) and Cluster 3 ($t(108) = -7.30, p < .001$) on the Flanker Inhibitory Control and Attention Test, with a mean difference of -28.1 and -16.7, respectively; while Cluster 2 scored significantly higher than Cluster 3 on this task ($t(101) = 4.08, p < .001$), with a mean difference of 11.4. Similar results were observed for the Dimensional Change Card Sort Test, with Cluster 1 scoring significantly lower than Cluster 2 ($t(82.1) = -15.8, p < .001$) and Cluster 3 ($t(96.7) = -12.50, p < .001$), with a mean difference of -37.4 and -27.3, respectively; while Cluster 2 displayed significantly higher scores than Cluster 3 on this task ($t(102.4) = 3.66, p = .001$), with a mean difference of 10.1. Further, significant group differences in performance were also found on the Picture

Sequence Memory Test, with Cluster 1 scoring significantly lower than Cluster 2 ($t(105) = -4.40, p < .001$), with a mean difference of -12.2, and Cluster 2 scoring significantly higher than Cluster 3 ($t(94.5) = 4.38, p < .001$), with a mean difference of 11.59.

Further, post-hoc analyses also indicated significant differences on the List Sorting Working Memory Test, with Cluster 1 displaying significantly lower scores than Cluster 2 ($t(117) = -5.99, p < .001$) and significantly higher scores than Cluster 3 ($t(120) = 2.45, p = .041$), with a mean difference of -13.7 and 5.94, respectively. To this end, Cluster 2 also scored significantly higher than Cluster 3 on this task ($t(104) = 8.49, p < .001$), with a mean difference of 19.61. Additionally, Cluster 1 scored significantly lower than Cluster 2 ($t(104) = -10.2, p < .001$) and Cluster 3 ($t(120.2) = -12.66, p < .001$) on the Pattern Comparison Processing Speed Test, with a mean difference of -32.2 and -36.23, respectively. Significant differences were also found on the Picture Vocabulary Test, with Cluster 1 displaying significantly lower scores than Cluster 2 ($t(107) = -6.82, p < .001$), and significantly higher scores than Cluster 3 ($t(121.9) = 2.64, p = .025$), with a mean difference of -14.9 and 4.98, respectively. Cluster 2 also scored significantly higher than Cluster 3 on this task ($t(94.7) = 9.59, p < .001$), with a mean difference of 19.85. Finally, post-hoc comparisons indicated that Cluster 1 displayed significantly lower scores than Cluster 2 ($t(113) = -5.98, p < .001$), with a mean difference of -13.8; while Cluster 2 scored significantly higher than Cluster 3 on this task ($t(104) = 7.44, p < .001$), with a mean difference of 19.01.

Clusters Derived from Cognitive and Mental Health Data

A *k*-means cluster analysis was conducted with the addition of 19 mental health variables from the ERSQ, SDQ and E-SWAN. This analysis aimed to explore whether mental health dimensions can help to further explain the variability in data-driven transdiagnostic groups, and whether mental health functioning will affect cluster structure and/or membership. Again, a three-cluster structure was suggested as the optimal solution using the gap statistic. To this end, the clusters consisted of 55, 64 and 56 participants, respectively. As can be seen in Table 7, individuals with a formal ND were most frequently placed in Cluster 1 (capturing 38.8% of all individuals with a formal ND), followed by Cluster 2 (36.7%) and Cluster 3 (24.5%). Participants with a suspected ND most frequently appeared in Cluster 2 (representing 38% of all individuals with a suspected ND), followed by Cluster 1 (32.4%) and Cluster 3 (29.6%). Finally, participants with no ND were most frequent in Cluster 3 (representing 66.7% of all

individuals with no ND), followed by Cluster 2 (27.8%) and Cluster 1 (5.6%). Overlaps were observed across all three clusters, as no diagnostic group (i.e., ND, Suspected ND or No ND) was fully represented by a single cluster, and no individual cluster was fully composed of a single diagnostic group. The prevalence of diagnostic groups within individual clusters is presented in Table 7.

Further, Cluster 2 captured the highest proportion of individuals with ADHD (50% of all individuals with this diagnosis), followed by Cluster 1 (30%) and Cluster 3 (20%). Autistic individuals were most frequently captured by Cluster 1 (71.4%), followed by Clusters 2 and 3, both representing 14.3% of all participants with this diagnosis, respectively. Individuals with Dyslexia were equally represented by Cluster 2 and Cluster 3 (both capturing 35.7% of individuals with this diagnosis), with Cluster 1 representing the remaining 28.6% of dyslexic participants. Interestingly, individuals with Intellectual Disability were captured by Clusters 1 and 2 only, with 2/3 of these participants being placed in Cluster 1. These results indicate a high between-cluster overlap in the distribution of primary diagnosis type across all three clusters. The remaining (less prevalent) types of primary diagnoses are presented in Table 7.

Table 7. Frequency of diagnostic groups and types of primary diagnoses within individual clusters derived from cognitive and mental health data ($n = 175$).

	Cluster 1 ($n = 55$)	Cluster 2 ($n = 64$)	Cluster 3 ($n = 56$)
Group	n (%)	n (%)	n (%)
Diagnostic Group			
ND	19 (34.5)	18 (28.1)	12 (21.4)
Suspected ND	35 (63.6)	41 (64.1)	32 (57.1)
No ND	1 (1.8)	5 (7.8)	12 (21.4)
Type of Diagnosis			
<i>N</i>	19 (100)	18 (100)	12 (100)
ADHD	6 (31.6)	10 (55.6)	4 (33.3)
Autism	5 (26.3)	1 (5.6)	1 (8.3)
Dyslexia	4 (21.1)	5 (27.8)	5 (41.7)
Dyspraxia	-	1 (5.6)	-
ID	2 (10.5)	1 (5.6)	-
SLI	1 (5.3)	-	-
TS	1 (5.3)	-	-
PPD	-	-	1 (8.3)
SLD	-	-	1 (8.3)

Note. *ND* = Neurodiverse Diagnosis. *ADHD* = Attention Deficit Hyperactivity Disorder. *ID* = Intellectual Disability. *SLI* = Specific Language Impairment. *TS* = Tourette Syndrome. *PPD* = Perceptual Processing Difficulty. *SLD* = Specific Learning Difficulty (but not specified). The frequency of primary diagnosis type was calculated for individual clusters from the total number of participants with a formal ND found within each cluster.

ANOVA & Post-Hoc Results for Clusters Derived from Cognitive and Mental Health Data

Table 8. Mean standard scores and one-way ANOVA results for group differences on cognitive tasks for clusters derived from cognitive and mental health data ($n = 175$).

	Cluster 1 ($n = 55$)	Cluster 2 ($n = 64$)	Cluster 3 ($n = 56$)			
Task	$M (SD)$	$M (SD)$	$M (SD)$	F	$df2$	p
Flanker Inhibitory Control and Attention Test	69.0 (11.7)	92.5 (15.3)	84.9 (16.7)	47.89	112	<.001
Dimensional Change Card Sort Test	71.0 (15.1)	100.8 (15.7)	86.7 (18.5)	55.07	112	<.001
Picture Sequence Memory Test	98.2 (15.4)	106.1 (14.8)	94.7 (11.9)	11.12	113	<.001
List Sorting Working Memory Test	100.9 (14.9)	107.9 (14.9)	99.8 (14.0)	5.39	114	.006
Pattern Comparison Processing Speed Test	81.7 (22.6)	108.9 (17.2)	101.2 (22.1)	26.55	109	<.001
Picture Vocabulary Test	93.6 (12.2)	105.0 (13.3)	90.7 (10.6)	22.72	114	<.001
Oral Reading Recognition Test	121.6 (14.9)	121.6 (16.0)	117.5 (14.7)	1.44	114	.242

Note. M = Mean. SD = Standard Deviation. df = Degrees of Freedom ($df1 = 2$ across all tasks).

A one-way ANOVA revealed significant group differences between the three clusters on all cognitive measures, except the Oral Reading Recognition Test (as shown in Table 8). Post-hoc comparisons using the Games-Howell test indicated that Cluster 1 scored significantly lower than Cluster 2 ($t(115) = -9.48, p < .001$), and Cluster 3 ($t(98.5) = -5.83, p < .001$) on the Flanker Inhibitory Control and Attention Test, with a mean difference of -23.5 and -15.95, respectively. Cluster 2 also displayed significantly higher scores than Cluster 3 on this task ($t(112.5) = 2.58, p = .030$), with a mean difference of 7.59. Similar results were observed for the Dimensional Change Card Sort Test, with Cluster 1 scoring significantly lower than Cluster 2 ($t(115) = -10.5, p < .001$) and Cluster 3 ($t(106) = -4.91, p < .001$), with a mean difference of -29.8 and -15.8, respectively. Cluster 2 also scored significantly higher than Cluster 3 on this task ($t(108) = 4.44, p < .001$), with a mean difference of 14.0.

Further, Cluster 1 displayed significantly lower scores than Cluster 2 on the Picture Sequence Memory Test ($t(113) = -2.87, p = .014$), with a mean difference of -7.96; while Cluster 2 scored significantly higher than Cluster 3 on this test ($t(117) = 4.69, p < .001$), with a mean difference of 11.45. Post-hoc analyses revealed similar results for the List Sorting Working Memory Test, with Cluster 1 displaying significantly lower scores than Cluster 2 ($t(114) = -2.53, p = .034$), with a mean difference of -6.93; and Cluster 2 scoring significantly higher than Cluster 3 ($t(117) = 3.06, p = .008$), with a mean difference of 8.07. Significant group differences were also observed on the Pattern Comparison Processing Speed Test, with Cluster 1 scoring significantly lower than Cluster 2 ($t(99.9) = -7.30, p < .001$) and Cluster 3 ($t(109) = -4.59, p < .001$), with a mean difference of -27.2 and -19.47, respectively. Finally, post-hoc analyses revealed that Cluster 1 scored significantly lower than Cluster 2 on the Picture Vocabulary Test ($t(116) = -4.90, p < .001$), with a mean difference of -11.5; while Cluster 2 scored significantly higher than Cluster 3 on this task ($t(117) = 6.57, p < .001$), with a mean difference of 14.29.

A one-way ANOVA also revealed significant group differences between the three clusters on the ERSQ, SDQ and E-SWAN (as shown in Table 9). Post-hoc comparisons using the Games-Howell test indicated that Cluster 1 ($t(104) = -5.78, p < .001$) and Cluster 2 ($t(114) = -4.29, p < .001$) showed significantly fewer Emotional Awareness skills than Cluster 3, with a mean difference of -0.67 and -0.51. Similar results were observed on the Sensations subscale, with Cluster 1 ($t(102) = -4.15, p < .001$) and Cluster 2 ($t(117) = -5.61, p < .001$) both scoring significantly lower than Cluster 3, with a mean difference of -0.56 and -0.71, respectively. Post-hoc analyses also revealed that Cluster 1 ($t(100) = -5.73, p < .001$) and Cluster 2 ($t(115) = -5.55, p < .001$) displayed significantly less Emotional Clarity than Cluster 3, with a mean difference of -0.81 and -0.75. Additionally, Cluster 1 reported significantly lower Emotional Understanding than Cluster 3 ($t(106) = -5.86, p < .001$), with a mean difference of -0.80; while Cluster 2 also displayed significantly lower Understanding scores than Cluster 3 ($t(116) = -5.47, p < .001$), with a mean difference of -0.76. Further, Cluster 1 reported significantly lower Acceptance of emotions than Cluster 2 ($t(117) = -3.59, p = .001$) and Cluster 3 ($t(109) = -6.90, p < .001$), with a mean difference of -0.46 and -0.87, respectively. To this end, Cluster 2 also displayed significantly lower Acceptance scores than Cluster 3 ($t(118) = -3.15, p = .006$), with a mean difference of -0.40.

Post-hoc comparisons also indicated significant differences in Tolerance of emotions, with Cluster 1 reporting significantly lower Tolerance than Cluster 2 ($t(115) = -4.76, p < .001$) and Cluster 3 ($t(106) = -6.15, p < .001$), with a mean difference of -0.67 and -0.83, respectively. Significant differences were also observed on the Confrontation subscale, with Cluster 1 ($t(109) = -7.14, p < .001$) and Cluster 2 ($t(118) = -7.76, p < .001$) displaying significantly lower scores than Cluster 3, with a mean difference of -0.92 and -1.00. Similarly, Cluster 1 ($t(102) = -9.24, p < .001$) and Cluster 2 ($t(111) = -6.82, p < .001$) reported significantly fewer Self-Support skills than Cluster 3, with a mean difference of -1.08 and -0.83, respectively. Finally, Cluster 1 displayed significantly fewer Emotion Modification skills than Cluster 2 ($t(117) = -3.43, p = .002$) and Cluster 3 ($t(106) = -10.25, p < .001$), with a mean difference of -0.44 and -1.20, respectively.

Table 9. Mean scores and one-way ANOVA results for group differences on mental health measures for clusters derived from cognitive and mental health data ($n = 175$).

	Cluster 1 ($n = 55$)	Cluster 2 ($n = 64$)	Cluster 3 ($n = 56$)			
Measure	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	<i>F</i>	<i>df2</i>	<i>p</i>
ERSQ						
Awareness	2.3 (0.7)	2.5 (0.8)	3.0 (0.5)	19.15	113	<.001
Sensations	2.4 (0.8)	2.2 (0.8)	2.9 (0.6)	17.82	113	<.001
Clarity	2.2 (0.8)	2.3 (0.8)	3.0 (0.6)	23.00	113	<.001
Understanding	2.3 (0.8)	2.3 (0.9)	3.1 (0.7)	22.63	114	<.001
Acceptance	1.9 (0.7)	2.3 (0.7)	2.7 (0.7)	23.72	114	<.001
Tolerance	2.0 (0.8)	2.6 (0.8)	2.8 (0.7)	20.10	114	<.001
Confrontation	2.2 (0.7)	2.1 (0.7)	3.1 (0.7)	37.58	114	<.001
Self-Support	2.1 (0.7)	2.4 (0.8)	3.2 (0.5)	49.55	113	<.001
Modification	1.7 (0.7)	2.1 (0.7)	2.9 (0.6)	55.18	114	<.001
SDQ						
Emotional Difficulties	2.5 (1.8)	4.3 (2.1)	6.3 (2.2)	51.41	114	<.001
Conduct Problems	7.2 (1.8)	7.6 (1.6)	8.2 (1.3)	6.88	112	.002
Hyperactivity	3.2 (2.1)	3.2 (2.0)	5.5 (1.9)	26.70	113	<.001
Peer Problems	6.1 (1.7)	7.2 (1.7)	7.6 (1.4)	12.60	114	<.001
Prosocial Skills	8.1 (1.8)	8.5 (1.3)	9.1 (1.1)	7.43	109	<.001
Impact	5.7 (2.5)	6.6 (2.2)	8.6 (1.7)	29.85	111	<.001
E-SWAN						
ADHD	3.7 (0.9)	3.8 (0.8)	4.7 (0.9)	22.79	111	<.001
Depression	3.1 (0.8)	3.5 (0.6)	4.7 (0.9)	52.24	107	<.001
Panic Disorder	3.0 (0.8)	3.6 (0.8)	4.4 (1.1)	29.45	109	<.001
Social Anxiety	2.9 (1.0)	3.6 (1.2)	4.5 (1.2)	28.03	114	<.001

Note. *M* = Mean. *SD* = Standard Deviation. *df* = Degrees of Freedom (*df* = 2 across all subscales). *ADHD* = Attention Deficit Hyperactivity Disorder. *ERSQ* = Emotion Regulation Skills Questionnaire. *SDQ* = Strengths and Difficulties Questionnaire. *E-SWAN* = Extended Strengths and Weaknesses Assessment of Normal Behaviour. Emotional *D* = Emotional Difficulties. Conduct *P* = Conduct Problems. Higher scores on each mental health measure now indicate more adaptive functioning (i.e.,

fewer difficulties). For instance, a score of 4 or above on the E-SWAN no longer represents above average weaknesses, but instead represents strengths.

For the following results, the scores were reversed to ensure all measures were on the same scale. Therefore, lower scores on the subsequent measures signify more difficulties. A one-way ANOVA revealed significant group differences on the SDQ (as shown in Table 9). Post-hoc analyses using the Games-Howell test indicated that Cluster 1 displayed more Emotional Difficulties than Cluster 2 ($t(117) = -5.04, p < .001$) and Cluster 3 ($t(106) = -10.14, p < .001$), with a mean difference of -1.81 and -3.79. To this end, Cluster 2 also reported more Emotional Difficulties than Cluster 3 ($t(116) = -5.06, p < .001$), with a mean difference of -1.99. Further, Cluster 1 ($t(98.4) = -3.51, p = .002$) and Cluster 2 ($t(117.5) = -2.49, p < .038$) reported significantly more Conduct Problems than Cluster 3, with a mean difference of -1.03 and -0.65, respectively. Similar results were observed for the Hyperactivity subscale, with Cluster 1 ($t(108) = -6.17, p < .001$) and Cluster 2 ($t(117) = -6.44, p < .001$) both reporting significantly more signs of Hyperactivity than Cluster 3, with a mean difference of -2.32 and -2.26. Additionally, Cluster 1 reported significantly more Peer Problems than Cluster 2 ($t(115) = -3.57, p = .002$) and Cluster 3 ($t(106) = -4.93, p < .001$), with a mean difference of -1.11 and -1.46, respectively. Post-hoc analyses also revealed significant group differences on the Prosocial scale, with Cluster 1 ($t(88.4) = -3.45, p = .002$) and Cluster 2 ($t(117.3) = -2.80, p = .016$) displaying significantly fewer Prosocial skills than Cluster 3, with a mean difference of -0.98 and -0.62. Finally, Cluster 1 ($t(95.2) = -7.05, p < .001$) and Cluster 2 ($t(116.5) = -5.53, p < .001$) reported a significantly higher Impact of Difficulties than Cluster 3, with a mean difference of -2.90 and -1.99, respectively.

Furthermore, a one-way ANOVA also revealed significant group differences on the E-SWAN subscales (see Table 9). Post-hoc Games-Howell analyses showed that Cluster 1 ($t(109) = -6.05, p < .001$) and Cluster 2 ($t(107) = -5.93, p < .001$) both reported more ADHD symptoms than Cluster 3, with a mean difference of -1.06 and -0.94, respectively. Significant differences were also observed on the Depression subscale, with Cluster 1 reporting significantly more symptoms of Depression than Cluster 2 ($t(99.9) = -2.94, p = .011$) and Cluster 3 ($t(107.4) = -9.86, p < .001$), with a mean difference of -0.39 and -1.61; while Cluster 2 also displayed more symptoms of Depression than Cluster 3 ($t(93.2) = -8.47, p < .001$), with a mean difference of -1.22. Similarly, Cluster 1 reported significantly more symptoms of Panic Disorder than Cluster 2 ($t(113) = -4.06, p < .001$) and Cluster 3 ($t(99.2) = -7.66, p < .001$), with a mean difference of

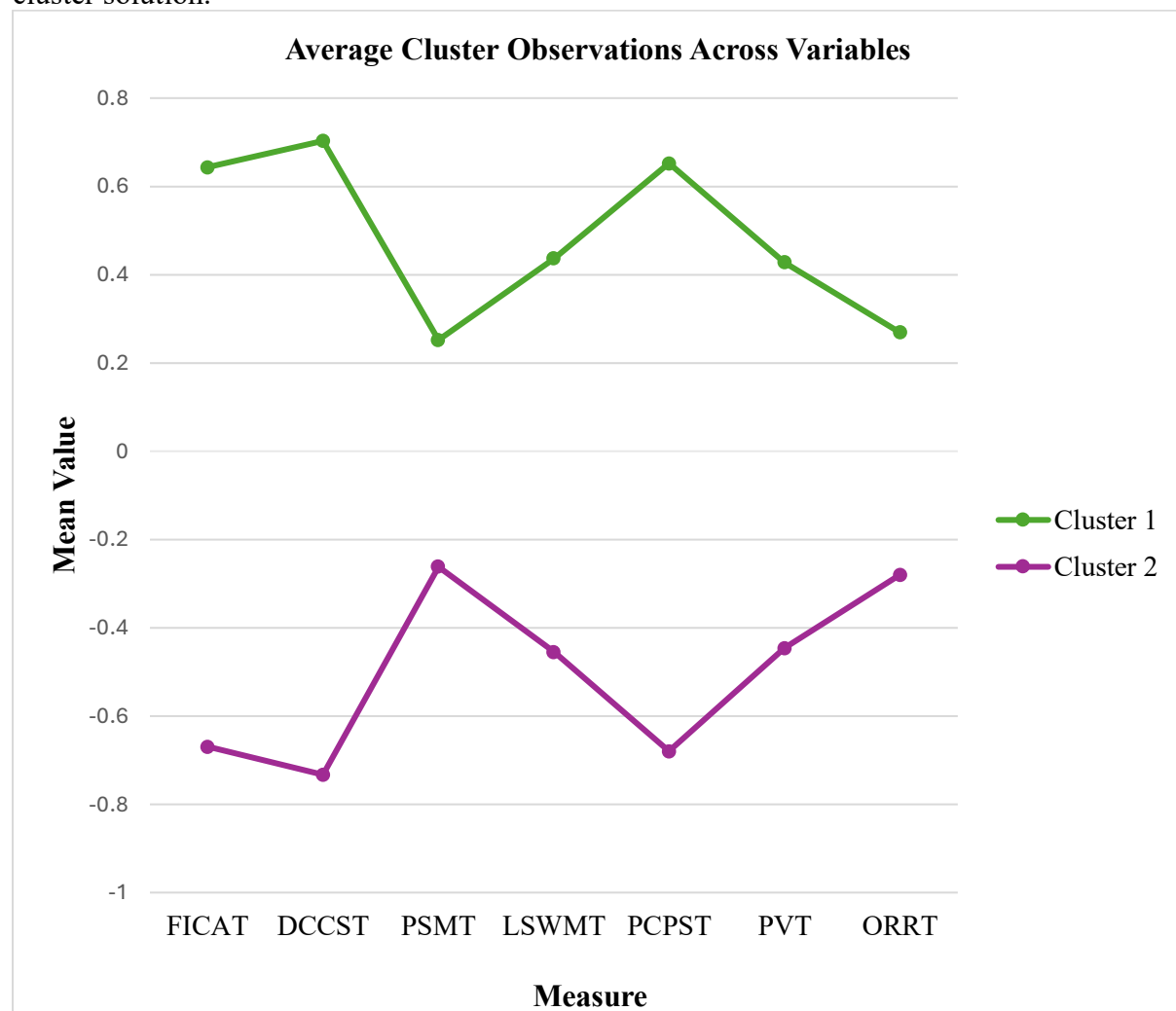
-0.58 and -1.41, respectively. To this end, Cluster 2 also displayed significantly more symptoms of Panic Disorder than Cluster 3 ($t(94.8) = -4.67, p < .001$), with a mean difference of -0.83. Finally, Cluster 1 reported significantly more symptoms of Social Anxiety than Cluster 2 ($t(117) = -3.47, p = .002$) and Cluster 3 ($t(108) = -7.51, p < .001$), with a mean difference of -0.70 and -1.56, respectively; while Cluster 2 also displayed significantly more symptoms of Social Anxiety than Cluster 3 ($t(116) = -4.00, p < .001$), with a mean difference of -0.86.

Cluster Analysis by Primary Diagnostic Label (Cognitive Data)

A separate *k*-means cluster analysis was performed on the Neurodiverse Diagnosis (ND) group, with the aim to explore whether data-driven groups map directly onto formal diagnostic labels based on cognitive dimensions. A two-cluster structure was suggested by the gap statistic as the optimal solution. However, three-cluster and six-cluster solutions were also explored as there were clear inflections at these points using the elbow method. Average cluster observations across cognitive measures for the different cluster solutions are shown in Figures 1, 2 and 3. Given the ambiguity of the elbow method and the underlying structure of the dataset, a cluster silhouette analysis was conducted in SPSS (Version 29.0.2.0; IBM SPSS Statistics, 2023) to help determine the optimal cluster solution.

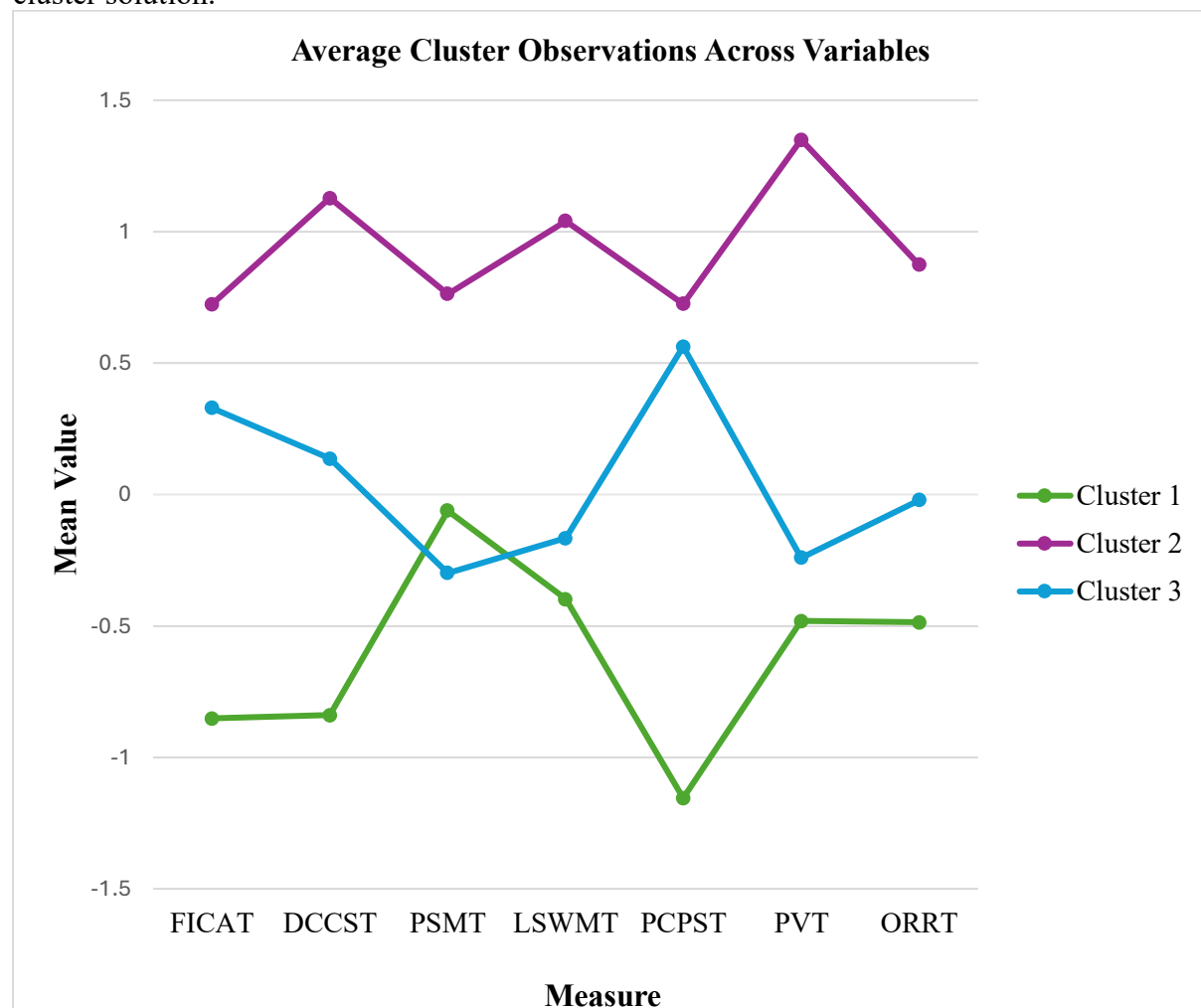
As can be seen in Table 10, the 2-cluster solution was reasonable but only moderate in quality, given the negative minimum value in Cluster 2, which suggests there may be some misclassification or overlap between the clusters. However, the maximum values were similar across both clusters within this solution, suggesting that the most cohesive points were reasonably well-grouped. Further, the 3-cluster solution seemed to improve the overall clustering cohesion compared to the initial 2-cluster solution, with Cluster 1 showing a high mean silhouette score. However, Cluster 3 within this solution had a relatively low mean score and a negative minimum value, indicating poor grouping of some points within this cluster. Finally, the 6-cluster structure did not appear to be an effective solution overall. Although Cluster 3 within this solution was well-defined (i.e., highest mean silhouette score and highest minimum score), there was a large number of low silhouette scores in the remaining clusters (especially Clusters 1 and 6), suggesting that this solution was likely overfitting the data. Therefore, the 3-cluster structure was chosen as the optimal solution given its higher average silhouette score compared to the 2-cluster solution suggested by the gap statistic, offering a good balance between cluster cohesion and separation.

Figure 1. A line graph showing average observations across cognitive variables for a two-cluster solution.



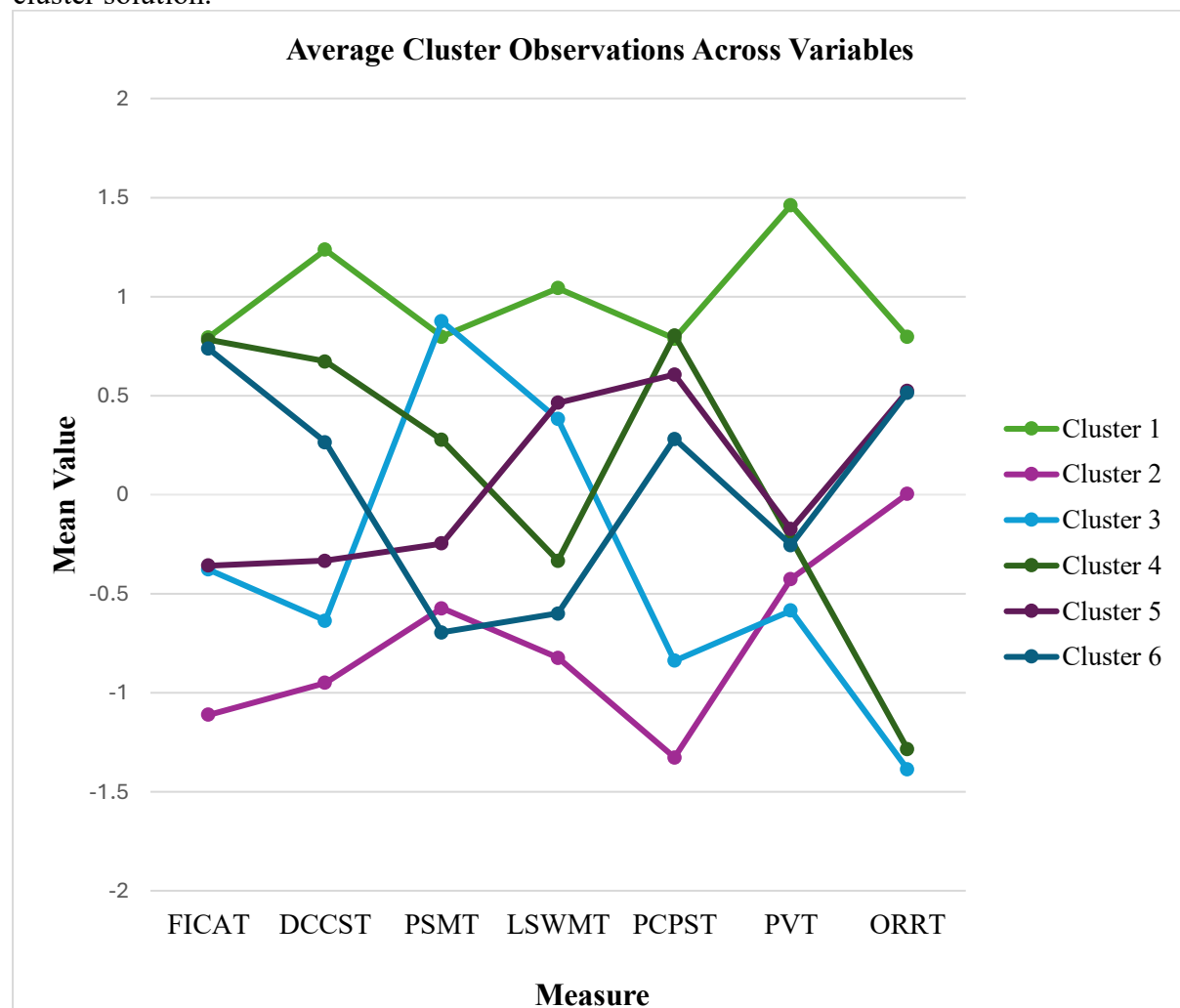
Note. *FICAT* = Flanker Inhibitory Control and Attention Test. *DCCST* = Dimensional Change Cart Sort Test. *PSMT* = Picture Sequence Memory Test. *LSWMT* = List Sorting Working Memory Test. *PCPST* = Pattern Comparison Processing Speed Test. *PVT* = Picture Vocabulary Test. *ORRT* = Oral Reading Recognition Test.

Figure 2. A line graph showing average observations across cognitive variables for a three-cluster solution.



Note. *FICAT* = Flanker Inhibitory Control and Attention Test. *DCCST* = Dimensional Change Cart Sort Test. *PSMT* = Picture Sequence Memory Test. *LSWMT* = List Sorting Working Memory Test. *PCPST* = Pattern Comparison Processing Speed Test. *PVT* = Picture Vocabulary Test. *ORRT* = Oral Reading Recognition Test.

Figure 3. A line graph showing average observations across cognitive variables for a six-cluster solution.



Note. *FICAT* = Flanker Inhibitory Control and Attention Test. *DCCST* = Dimensional Change Cart Sort Test. *PSMT* = Picture Sequence Memory Test. *LSWMT* = List Sorting Working Memory Test. *PCPST* = Pattern Comparison Processing Speed Test. *PVT* = Picture Vocabulary Test. *ORRT* = Oral Reading Recognition Test.

Table 10. Cluster silhouette statistics for the three cluster solutions derived from cognitive data for the ND group only ($n = 49$).

Cluster Solution	Case Count	Mean	Minimum	Maximum
2-Cluster Solution				
Cluster 1	25	.312	.062	.491
Cluster 2	24	.296	-.072	.495
3-Cluster Solution				
Cluster 1	17	.384	.117	.520
Cluster 2	10	.328	.143	.438
Cluster 3	22	.179	-.025	.372
6-Cluster Solution				
Cluster 1	9	.285	.011	.430
Cluster 2	11	.306	.131	.465
Cluster 3	6	.384	.359	.422
Cluster 4	6	.337	.203	.472
Cluster 5	9	.302	.187	.390
Cluster 6	8	.124	.013	.240

Note. ND = Neurodiverse Diagnosis.

The three clusters consisted of 17, 10 and 22 participants, respectively. The cluster membership of individuals with different primary diagnostic labels was explored (as shown in Table 11). As expected, given the heterogeneity and frequent co-occurrence of NDs, the diagnostic categories did not directly map onto data-driven clusters. For instance, the most prevalent diagnoses within this dataset (ADHD, autism and dyslexia) were captured by all three clusters. Cluster 1 captured 20% of all individuals with a primary diagnosis of ADHD within this dataset, with Cluster 2 representing 35%, and Cluster 3 capturing the biggest proportion of participants with ADHD (45%). Further, the majority of autistic individuals were represented by Cluster 1 (57.1%), followed by Cluster 3 (28.6%), and Cluster 2 (14.3%). Finally, Cluster 3 captured the biggest proportion of participants with Dyslexia (57.1%), followed by Cluster 1 (28.6%) and Cluster 2 (14.3%). Interestingly, the only individual with a primary diagnosis of Dyspraxia was captured by the cluster with the highest proportion of participants with ADHD and Dyslexia (Cluster 3).

Table 11. Cluster membership of individuals with different types of primary NDs based on cognitive data only ($n = 49$).

	Cluster 1 ($n = 17$)	Cluster 2 ($n = 10$)	Cluster 3 ($n = 22$)
Diagnosis	n (%)	n (%)	n (%)
ADHD	4 (23.5)	7 (70)	9 (40.9)
Autism	4 (23.5)	1 (10)	2 (9.1)
Dyslexia	4 (23.5)	2 (20)	8 (36.4)
Dyspraxia	-	-	1 (4.5)
ID	2 (11.8)	-	1 (4.5)
SLI	1 (5.9)	-	-
TS	1 (5.9)	-	-
PPD	-	-	1 (4.5)
SLD	1 (5.9)	-	-

Note. ND = Neurodiverse Diagnosis. ADHD = Attention Deficit Hyperactivity Disorder. ID = Intellectual Disability. SLI = Specific Language Impairment. TS = Tourette Syndrome. PPD = Perceptual Processing Difficulty. SLD = Specific Learning Difficulty (not specified). The percentages indicate the proportion of individuals with a given diagnostic label within each cluster.

A one-way ANOVA revealed significant group differences between the clusters on all cognitive measures (see Table 12). Post-hoc comparisons using the Games-Howell test indicated that Cluster 1 scored significantly lower than Cluster 2 ($t(16.2) = -5.79, p < .001$) and Cluster 3 ($t(36.2) = -4.86, p < .001$) on the Flanker Inhibitory Control and Attention Test, with a mean difference of -32.2 and -24.13, respectively. Similar results were observed for the Dimensional Change Card Sort Test, with Cluster 1 displaying significantly lower scores than Cluster 2 ($t(12.1) = -6.29, p < .001$) and Cluster 3 ($t(36.2) = -4.95, p < .001$), with a mean difference of -42.6 and -21.1; while Cluster 2 scored significantly higher than Cluster 3 ($t(14.6) = 3.01, p = .023$), with a mean difference of 21.5. Additionally, Cluster 2 also scored significantly higher than Cluster 3 on the Picture Sequence Memory Test ($t(15.5) = 3.36, p = .011$), with a mean difference of 13.58. Post-hoc analyses also revealed significant differences on the List Sorting Working Memory Test, with Cluster 1 displaying significantly lower scores than Cluster 2 ($t(21.9) = -5.43, p < .001$), with a mean difference of -23.2, and Cluster 2 scoring significantly higher than Cluster 3 ($t(29.7) = 5.43, p < .001$), with a mean difference of 19.44. Significant differences were also found on the Pattern Comparison Processing Speed Test, with Cluster 1 scoring significantly lower than Cluster 2 ($t(22.2) = -10.9, p < .001$) and Cluster 3 ($t(36.9) = -9.74, p < .001$), with a mean difference of -50.7 and -46.35, respectively. Cluster 1 also displayed significantly lower scores than Cluster 2 on the Picture Vocabulary Test ($t(19.9) = -5.61, p < .001$), with a mean difference of -28.0; while Cluster 2 scored significantly higher than Cluster 3 on this task ($t(13.6) = 5.63, p < .001$), with a mean difference of 24.31. Finally, Cluster 1 scored significantly lower than Cluster 2 on the Oral Reading Recognition Test

($t(19.6) = -4.35, p < .001$), with a mean difference of -22.1; while Cluster 2 displayed significantly higher scores than Cluster 3 on this task ($t(22.1) = 2.77, p = .029$), with a mean difference of 14.55.

Table 12. Mean scores and one-way ANOVA results for group differences across cognitive tasks for clusters derived from formal ND groups ($n = 49$).

	Cluster 1 ($n = 17$)	Cluster 2 ($n = 10$)	Cluster 3 ($n = 22$)			
Task	$M (SD)$	$M (SD)$	$M (SD)$	F	$df2$	p
Flanker Inhibitory Control and Attention Test	65.8 (12.3)	98.0 (14.8)	90.0 (18.6)	21.21	24.3	<.001
Dimensional Change Card Sort Test	67.6 (10.6)	110.2 (19.8)	88.7 (16.0)	25.54	21.6	<.001
Picture Sequence Memory Test	101.4 (14.9)	111.9 (11.0)	98.3 (9.6)	5.47	22.6	.012
List Sorting Working Memory Test	100.1 (15.9)	123.3 (5.7)	103.9 (14.5)	23.75	29.8	<.001
Pattern Comparison Processing Speed Test	65.5 (13.2)	116.2 (10.8)	111.8 (16.5)	71.37	27.1	<.001
Picture Vocabulary Test	90.4 (13.0)	118.4 (12.2)	94.1 (9.0)	18.07	21.5	<.001
Oral Reading Recognition Test	112.5 (13.1)	134.6 (12.6)	120.0 (16.1)	9.24	25.5	<.001

Note. ND = Neurodiverse Diagnosis. M = Mean. SD = Standard Deviation. df = Degrees of Freedom ($df1 = 2$ across all tasks).

Cluster Analysis by Primary Diagnostic Label (Cognitive and Mental Health Data)

A *k*-means cluster analysis was conducted on the ND group with the addition of 19 mental health variables, with the aim to explore whether the optimal cluster solution and/or cluster membership of different diagnostic categories shifts as a result of interactions between cognitive and mental health dimensions. Similarly to the first analysis using cognitive variables only, the gap statistic suggested a 2-cluster structure as the optimal solution, while the elbow method indicated two inflection points at the 3-cluster and 6-cluster solutions. Again, a cluster silhouette analysis was performed to assess the quality of these clusters and to help determine the optimal cluster solution. As can be seen in Table 13, the 2-cluster solution had very low mean silhouette scores and negative minimum values for both clusters, suggesting that these clusters may be weakly defined. The 3-cluster solution may also not be optimal due to the negative mean score of Cluster 2 as well as the negative minimum values for all clusters, suggesting poor cluster cohesion and misclassification of several cases. Interestingly, the 6-cluster solution appeared to be the optimal choice, as Cluster 5 stood out with the highest mean silhouette score across all solutions, with Clusters 2 and 3 also showing relatively high mean scores, indicating some well-defined clusters within this solution. Despite some negative mean silhouette scores and negative minimum values in Clusters 1, 4 and 6, the high-quality clusters balanced out the worst cases, making this solution likely to yield the most distinct groups.

However, upon closer inspection of individual clusters in the 6-cluster solution, it was found that the cluster with the highest silhouette score (Cluster 5) was comprised of 2 participants only. This is generally not statistically meaningful, as it is likely that such small sizes do not represent a true pattern in data, but may instead be a result of noise or outliers. Additionally, the other two clusters with high mean silhouette scores (Clusters 2 and 3) also had a relatively small number of participants. This is an issue as small clusters can be highly sensitive to slight changes in data and are unlikely to provide meaningful insights. Therefore, taking into consideration the silhouette analysis results and the composition of individual clusters within each cluster solution, the 2-cluster structure suggested by the gap statistic was decided upon as the optimal solution. This is because it appeared to be the most balanced in terms of cluster quality compared to the 3-cluster solution, with the 2-cluster solution having a higher overall silhouette score (suggesting more distinct clusters). Thus, the results suggest that the interaction between cognitive and mental health variables affected the data-driven grouping of primary diagnostic labels within this dataset.

Table 13. Cluster silhouette statistics for the three cluster solutions derived from cognitive and mental health data for the ND group only ($n = 49$).

Cluster Solution	Case Count	Mean	Minimum	Maximum
2-Cluster Solution				
Cluster 1	20	.011	-.083	.081
Cluster 2	29	.074	-.007	.158
3-Cluster Solution				
Cluster 1	20	.065	-.118	.193
Cluster 2	19	-.086	-.252	-.007
Cluster 3	10	.128	-.192	.304
6-Cluster Solution				
Cluster 1	11	-.086	-.244	.093
Cluster 2	7	.335	.100	.464
Cluster 3	5	.344	.236	.425
Cluster 4	12	-.044	-.233	.102
Cluster 5	2	.471	.366	.575
Cluster 6	12	-.040	-.403	.179

Note. ND = Neurodiverse Diagnosis.

As can be seen in Table 14, individuals with ADHD and Dyslexia were most frequently captured by Cluster 2, with 55% of all individuals with ADHD and 64.3% of all dyslexic participants being represented by this cluster. Further, 57.1% of all autistic individuals were captured by Cluster 1. Again, there was an overlap in the distribution of primary NDs between the two clusters, and no cluster was fully represented by a single diagnostic label. The cluster membership of less prevalent types of diagnoses can be seen in Table 14.

Table 14. Cluster membership of individuals with different types of primary NDs based on cognitive data and mental health data ($n = 49$).

	Cluster 1 ($n = 20$)	Cluster 2 ($n = 29$)
Diagnosis	n (%)	n (%)
ADHD	9 (45.0)	11 (37.9)
Autism	4 (20.0)	3 (10.3)
Dyslexia	5 (25.0)	9 (31.0)
Dyspraxia	-	1 (3.4)
ID	1 (5.0)	2 (6.9)
SLI	1 (5.0)	-
TS	-	1 (3.4)
PPD	-	1 (3.4)
SLD	-	1 (3.4)

Note. ND = Neurodiverse Diagnosis. ADHD = Attention Deficit Hyperactivity Disorder. ID = Intellectual Disability. SLI = Specific Language Impairment. TS = Tourette Syndrome. PPD = Perceptual Processing Difficulty. SLD = Specific Learning Difficulty (not specified). The percentages indicate the proportion of individuals with a given diagnostic label within each cluster.

A one-way ANOVA revealed no significant differences between the two clusters on any of the cognitive measures (see Table 15). However, significant group differences were found on several ERSQ subscales (see Table 16). Post-hoc comparisons using the Games-Howell test indicated that participants in Cluster 1 reported significantly fewer Emotional Awareness skills compared to Cluster 2 ($t(37.3) = -2.72, p = .010$), with a mean difference of -0.59. Similar results were observed on the Tolerance subscale, with Cluster 1 displaying significantly lower scores than Cluster 2 ($t(33.9) = -3.03, p = .005$), with a mean difference of -0.72. Additionally, Cluster 1 also scored significantly lower than Cluster 2 on Confrontation ($t(34.5) = -3.44, p = .002$), with a mean difference of -0.77. Post-hoc analyses also revealed significant differences on the Self-Support subscale, with Cluster 1 displaying significantly lower scores than Cluster 2 ($t(43.2) = -2.92, p = .006$), with a mean difference of -0.58. Finally, Cluster 1 also scored significantly lower on the Modification subscale ($t(41.6) = -5.58, p < .001$), with a mean difference of -1.14.

A one-way ANOVA also revealed significant group differences on the SDQ (see Table 16). Post-hoc comparisons indicated that Cluster 1 displayed significantly more Emotional Difficulties than Cluster 2 ($t(46.0) = -7.57, p < .001$), with a mean difference of -3.80; as well as reporting significantly more symptoms of Hyperactivity ($t(44.1) = -2.98, p = .005$), with a mean difference of -1.64. Similar results were observed for the Peer Problems subscale, with Cluster 1 reporting significantly more problems than Cluster 2 ($t(35.2) = -4.65, p < .001$), with a mean difference of -2.05. Additionally, post-hoc analyses also indicated that Cluster 1 reported a significantly higher Impact of difficulties than Cluster 2 ($t(44.5) = -8.29, p < .001$), with a mean difference of -3.90.

Significant differences were also observed on the E-SWAN subscales (see Table 16), with post-hoc comparisons indicating that Cluster 1 displayed significantly more ADHD symptoms than Cluster 2 ($t(40.5) = -2.38, p = .022$), with a mean difference of -0.63. Similarly, Cluster 1 reported significantly more symptoms of Depression than Cluster 2 ($t(46.1) = -7.89, p < .001$), with a mean difference of -1.31; as well as reporting significantly more symptoms of Panic Disorder ($t(38.1) = -3.68, p < .001$), with a mean difference of -1.05. Finally, Cluster 1 also displayed significantly more signs of Social Anxiety compared to Cluster 2 ($t(46.4) = -5.41, p < .001$), with a mean difference of -1.57.

Note that these analyses are only exploratory, with the aim to investigate whether formal diagnostic labels map directly onto data-driven groups. The number of observations per each variable here is not sufficient, given that the number of individuals with a formal diagnosis was limited within this dataset, as the majority of individuals fell in the ‘Suspected ND’ group, and thus were not classified into a specific diagnostic category. As a result, the exploratory clusters based on ND group data may not be as meaningful. This is reflected in the generally low silhouette scores in the optimal cluster solutions, with all values being closer to 0 than 1. Therefore, and any significant differences reported should be interpreted with caution.

Table 15. Mean standard scores and one-way ANOVA results for group differences on cognitive tasks for ND clusters derived from cognitive and mental health data ($n = 49$).

	Cluster 1 ($n = 20$)	Cluster 2 ($n = 29$)			
Task	$M (SD)$	$M (SD)$	F	$df2$	p
Flanker Inhibitory Control and Attention Test	79.2 (20.2)	86.0 (20.5)	1.33	41.4	.256
Dimensional Change Card Sort Test	86.9 (23.4)	85.0 (20.8)	0.09	37.8	.771
Picture Sequence Memory Test	102.0 (13.2)	102.2 (12.8)	0.00	40.2	.949
List Sorting Working Memory Test	110.1 (17.7)	104.1 (14.7)	1.53	35.9	.224
Pattern Comparison Processing Speed Test	95.3 (28.7)	97.6 (26.2)	0.08	38.6	.773
Picture Vocabulary Test	103.2 (17.4)	94.1 (12.6)	3.99	32.4	.054
Oral Reading Recognition Test	123.1 (15.1)	118.5 (17.0)	0.98	43.9	.327

Note. ND = Neurodiverse Diagnosis. M = Mean. SD = Standard Deviation. df = Degrees of Freedom ($df1 = 1$ across all tasks).

Table 16. Mean scores and one-way ANOVA results for group differences on mental health measures for ND clusters derived from cognitive and mental health data ($n = 49$).

	Cluster 1 ($n = 20$)	Cluster 2 ($n = 29$)			
Measure	$M (SD)$	$M (SD)$	F	$df2$	p
ERSQ					
Awareness	2.5 (0.7)	2.6 (0.8)	0.20	43.4	.658
Sensations	2.5 (0.9)	2.5 (0.7)	0.00	33.4	.959
Clarity	2.4 (0.9)	2.7 (0.8)	1.88	39.1	.179
Understanding	2.4 (0.8)	2.8 (0.9)	2.81	41.7	.101
Acceptance	2.0 (0.8)	2.6 (0.7)	7.42	37.3	.010
Tolerance	1.9 (0.9)	2.7 (0.7)	9.18	33.9	.005
Confrontation	1.8 (0.8)	2.5 (0.7)	11.86	34.5	.002
Self-Support	2.2 (0.7)	2.7 (0.7)	8.53	43.2	.006
Modification	1.5 (0.7)	2.6 (0.7)	31.17	41.6	<.001
SDQ					
Emotional Difficulties	1.8 (1.3)	5.6 (2.2)	57.24	46.0	<.001
Conduct Problems	7.3 (1.3)	8.0 (1.8)	2.78	47.0	.102
Hyperactivity	2.5 (1.8)	4.1 (2.0)	8.86	44.1	.005
Peer Problems	5.5 (1.6)	7.6 (1.3)	21.61	35.2	<.001
Prosocial Skills	8.4 (1.4)	8.5 (1.4)	0.01	40.2	.904
Impact	4.1 (1.5)	8.0 (1.8)	68.77	44.5	<.001
E-SWAN					
ADHD	3.2 (0.9)	3.8 (0.9)	5.66	40.5	.022
Depression	2.8 (0.5)	4.1 (0.6)	62.26	46.1	<.001
Panic Disorder	2.9 (1.0)	3.9 (0.9)	13.55	38.1	<.001
Social Anxiety	2.7 (0.8)	4.2 (1.3)	29.31	46.4	<.001

Note. M = Mean. SD = Standard Deviation. df = Degrees of Freedom ($df1 = 1$ across all subscales). *ADHD* = Attention Deficit Hyperactivity Disorder. *ERSQ* = Emotion Regulation Skills Questionnaire. *SDQ* = Strengths and Difficulties Questionnaire. *E-SWAN* = Extended Strengths and Weaknesses Assessment of Normal Behaviour. Emotional D = Emotional Difficulties. Conduct P = Conduct Problems. Higher scores on each mental health measure indicate more adaptive functioning (or fewer difficulties). For instance, a score of 4 or above on the E-SWAN no longer represents above average weaknesses, but instead represents strengths.

Appendix B. Comparison Analyses (Chapter 4)

Principal Component Analysis (PCA) of Cognitive Variables

The cognitive variables generated via the NIH Toolbox were retained as separate dimensions for the purposes of subsequent cluster analyses. This is because each of the 7 standardised measures were designed to tap a different cognitive domain, in comparison with the 19 mental health variables which displayed higher correlations (see Table 1), and needed to be reduced to fewer non-redundant dimensions. Although Bartlett's Test of Sphericity was significant in a PCA of cognitive variables ($\chi^2(21, N = 175) = 280, p < .001$), suggesting that dimensionality reduction was appropriate, the variables loaded onto two components only (see Table 2). The component loadings were reasonable; however, the components failed to adequately represent the distinct cognitive domains assessed by each measure. Although cognitive processes often show significant overlap and any given task cannot assess a single cognitive domain in isolation, reducing the assessed variables into fewer components would compromise the capacity to make meaningful distinctions between such cognitive abilities. Therefore, retaining separate cognitive variables in subsequent analyses allowed for a more accurate reflection of individual profiles.

Table 1. Spearman's rank-order correlation coefficient matrix for all cognitive and mental health variables (n = 175).

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26
1. FICAT	-																									
2. DCCST	0.69***	-																								
3. PSMT	0.17*	0.31***	-																							
4. LSWMT	0.25**	0.22**	0.26***	-																						
5. PCPST	0.45***	0.56***	0.22**	0.13	-																					
6. PVT	0.28***	0.30***	0.24**	0.31***	0.14	-																				
7. ORRT	0.12	0.09	0.08	0.31***	0.05	0.31***	-																			
8. Awareness	0.23**	0.21**	0.01	0.03	0.14	0.08	0.11	-																		
9. Sensations	0.08	0.01	-0.01	-0.01	0.07	-0.02	0.06	0.49***	-																	
10. Clarity	0.10	0.08	-0.01	0.05	0.10	-0.02	0.07	0.54***	0.60***	-																
11. Understanding	0.10	0.08	-0.03	0.04	0.17*	-0.08	0.05	0.57***	0.53***	0.74***	-															
12. Acceptance	0.17*	0.11	0.01	0.02	0.09	0.01	0.10	0.41***	0.19*	0.28***	0.33***	-														
13. Tolerance	0.17*	0.10	-0.00	0.07	0.13	0.07	0.11	0.26***	0.18*	0.25***	0.32***	0.60***	-													
14. Confrontation	0.13	0.01	-0.17*	-0.09	0.03	-0.15*	-0.14	0.34***	0.33***	0.25***	0.29***	0.43***	0.35***	-												
15. Self-Support	0.13	0.06	-0.05	-0.09	0.08	-0.08	-0.05	0.33***	0.41***	0.33***	0.36***	0.31***	0.30***	0.48***	-											
16. Modification	0.11	0.04	-0.15*	0.01	0.07	-0.15	-0.03	0.30***	0.28***	0.38***	0.41***	0.41***	0.38***	0.43***	0.50***	-										
17. Emotional D.	0.24**	0.17*	-0.08	0.00	0.16*	-0.06	0.02	0.22**	0.11	0.25***	0.29***	0.36***	0.26***	0.21**	0.38***	0.52***	-									
18. Conduct P.	0.07	0.03	0.06	0.01	0.13	-0.00	-0.08	0.11	0.09	0.19*	0.18*	0.12	0.07	0.08	0.08	0.18*	0.18*	-								
19. Hyperactivity	0.07	0.04	-0.04	-0.05	-0.02	-0.22**	-0.13	0.24*	0.20**	0.27***	0.19*	0.20**	0.06	0.29***	0.23**	0.24**	0.35***	0.21**	-							
20. Peer Problems	0.13	0.08	-0.08	-0.11	0.13	0.03	-0.00	0.18*	0.06	0.08	0.16*	0.10	0.13	0.06	0.15	0.32***	0.42***	0.17*	0.02	-						
21. Prosocial	-0.00	0.03	-0.02	0.09	0.09	-0.04	-0.13	0.09	0.20**	0.09	0.03	0.04	-0.03	0.26***	0.32***	0.26***	0.06	0.29***	0.16*	0.16*	-					
22. Impact	0.03	-0.00	-0.11	-0.15	-0.05	-0.19*	-0.20**	0.10	0.11	0.11	0.17*	0.34***	0.19*	0.37***	0.29***	0.47***	0.56***	0.21**	0.46***	0.28***	0.20**	-				
23. ADHD	0.08	0.06	-0.13	-0.18*	-0.01	-0.19*	-0.17*	0.28***	0.25**	0.26***	0.16*	0.19*	0.10	0.28***	0.29***	0.33***	0.34***	0.13	0.64***	0.09	0.17*	0.44***	-			
24. Depression	0.10	0.02	-0.20**	-0.16*	0.08	-0.28***	-0.15*	0.21**	0.23**	0.29***	0.31***	0.38***	0.30***	0.34***	0.40***	0.56***	0.68***	0.11	0.43***	0.32***	0.12	0.67***	0.51***	-		
25. Panic D.	0.15	0.11	-0.14	-0.06	0.09	-0.17*	-0.10	0.21**	0.07	0.23**	0.24**	0.28***	0.33***	0.30***	0.28***	0.40***	0.61***	0.09	0.30***	0.22**	-0.08	0.49***	0.43***	0.64***	-	
26. Social Anxiety	0.11	0.08	-0.14	0.05	0.06	-0.11	-0.07	0.16*	0.05	0.19*	0.25***	0.22**	0.26***	0.26***	0.33***	0.43***	0.63***	-0.02	0.21**	0.37***	0.01	0.44***	0.27***	0.58***	0.52***	-

Note. * $p < .05$, ** $p < .01$, *** $p < .001$. *FICAT* = Flanker Inhibitory Control and Attention Test. *DCCST* = Dimensional Change Card Sort Test. *PSMT* = Picture Sequence Memory Test. *LSWMT* = List Sorting Working Memory Test. *PCPST* = Pattern Comparison Processing Speed Test. *PVT* = Picture Vocabulary Test. *ORRT* = Oral Reading Recognition Test. Given that mental health and cognitive data may not follow a normal distribution, Spearman's rank order correlation was chosen to construct the correlation matrix, as it is less sensitive to outliers and non-normally distributed data, and better suited for capturing monotonic relationships compared to Pearson's correlation. However, the results did not differ significantly from Pearson's r , suggesting that non-normality did not substantially affect the results.

Table 2. Component loadings of cognitive variables in principal component analysis.

Variable	Component	
	1	2
Flanker Inhibitory Control and Attention Test	0.771	-0.313
Dimensional Change Card Sort Test	0.819	-0.364
Picture Sequence Memory Test	0.475	0.166
List Sorting Working Memory Test	0.539	0.505
Pattern Comparison Processing Speed Test	0.643	-0.466
Picture Vocabulary Test	0.588	0.428
Oral Reading Recognition Test	0.365	0.647

Note. ‘None’ rotation was used. Strong correlations are highlighted in bold.

PCA of Cognitive and Mental Health Variables Combined

In order to explore the overall structure of the dataset without the assumption that cognition and mental health are separate, all variables were simultaneously entered into a principal component analysis (PCA), with the aim to cross-compare differences in cluster solutions and the dimensions that distinguish between them. Results of Bartlett's Test of Sphericity were significant ($\chi^2(325, N = 175) = 1958, p < .001$), indicating that PCA was appropriate. The analysis was conducted with no rotation in order to maximise the amount of variance explained by each component. A minimum factor loading of 0.5 was applied to highlight variables with only moderate to strong correlations to a given component. The optimal number of components to retain was determined using parallel analysis, which suggested a 5-component solution. The component loadings are presented in Table 3. Together, the first five components explained 57.0% of the total variance, with PC1, PC2 and PC3 accounting for majority of the variance (45.2%) in the dataset (see Table 4).

The component loadings table indicated that PC1 was primarily associated with variables relating to emotional difficulties and specific mental health conditions, while PC2 was strongly associated with cognitive abilities. Further, PC3 was negatively correlated with variables relating to emotional clarity, PC4 displayed moderate to strong relationships with prosocial skills and conduct problems, while PC5 was associated with hyperactivity. Considering the highest loadings onto each component, PC1 could be labelled as the '*Emotional Difficulties*' dimension, PC2 could be characterised as the '*Cognitive Abilities*' dimension, PC3 could be defined as '*Emotional Clarity*', PC4 could be described as the '*Social Behaviour*' dimension, and PC5 could be labelled as '*Hyperactive Behaviour*'. Together, these results suggest that dimensions relating to emotional difficulties, cognitive abilities and emotional clarity explained the largest amount of variance in the dataset.

Table 3. Component loadings of cognitive and mental health variables in PCA.

Variable	Component				
	1	2	3	4	5
Cognition					
FICAT	0.233	0.661	0.363	0.003	0.183
DCCST	0.142	0.719	0.347	-0.019	0.291
PSMT	-0.128	0.474	0.029	-0.049	0.243
LSWMT	-0.059	0.555	0.021	0.001	-0.173
PCPST	0.193	0.577	0.230	0.100	0.244
PVT	-0.200	0.633	0.020	0.135	-0.151
ORRT	-0.094	0.448	-0.102	-0.176	-0.390
ERSQ					
Awareness	0.523	0.315	-0.462	-0.137	0.164
Sensations	0.488	0.104	-0.617	-0.004	0.131
Clarity	0.603	0.193	-0.558	-0.092	0.051
Understanding	0.620	0.174	-0.515	-0.108	-0.034
Acceptance	0.588	0.185	-0.022	-0.102	-0.237
Tolerance	0.516	0.239	0.002	-0.113	-0.341
Confrontation	0.604	-0.108	-0.118	0.075	-0.045
Self-Support	0.619	-0.000	-0.153	0.169	-0.145
Modification	0.736	-0.033	-0.037	0.212	-0.257
SDQ					
Emotional Difficulties	0.707	0.009	0.393	-0.056	-0.113
Conduct Problems	0.247	0.037	-0.032	0.554	0.312
Hyperactivity	0.523	-0.186	0.061	-0.141	0.537
Peer Problems	0.379	0.021	0.201	0.438	-0.257
Prosocial Skills	0.277	-0.018	-0.091	0.802	0.040
Impact	0.651	-0.276	0.318	0.138	0.017
E-SWAN					
ADHD	0.608	-0.206	0.080	-0.159	0.452
Depression	0.800	-0.239	0.245	-0.135	0.000
Panic Disorder	0.701	-0.115	0.293	-0.326	-0.003
Social Anxiety	0.635	-0.051	0.339	-0.113	-0.299

Note. 'None' rotation was used. *PCA* = Principal Component Analysis. *FICAT* = Flanker Inhibitory Control and Attention Test. *DCCST* = Dimensional Change Card Sort Test. *PSMT* = Picture Sequence Memory Test. *LSWMT* = List Sorting Working Memory Test. *PCPST* = Pattern Comparison Processing Speed Test. *PVT* = Picture Vocabulary Test. *ORRT* = Oral Reading Recognition Test. Strong correlations (above 0.5) are highlighted in bold.

Table 4. Percentage of variance explained by each principal component in PCA.

Component	SS Loadings	% of Variance	Cumulative %
1	6.73	25.87	25.9
2	2.93	11.27	37.1
3	2.10	8.08	45.2
4	1.55	5.96	51.2
5	1.51	5.81	57.0

Note. *SS* = Sum of Squares. *PCA* = Principal Component Analysis. The *SS* loading is the sum of squared loadings for all variables contributing to a given component. This indicates how much of the variance in the original dataset is accounted for by that component, based on the contributions of each variable. An *SS* loading close to 1 indicates that the component explains a larger amount of variance in the data.

Comparison of PCA and Exploratory Factor Analysis (EFA) Models for Cognitive and Mental Health Variables Combined

The PCA results were compared with an exploratory factor analysis (EFA) of the same variables, using Principal Axis Factoring with no rotation. The Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy was 0.83 and Bartlett's Test of Sphericity was significant ($\chi^2(325, N = 175) = 1958, p < .001$), indicating that the data were factorable and appropriate for EFA. Similarly to the PCA, a minimum factor loading of 0.5 was applied to highlight variables with only moderate or strong correlations to a given factor. The optimal number of factors retained was determined using parallel analysis, which suggested a 6-factor solution (presented in Table 5). The six factors cumulatively explained 51.6% of variance in the dataset (see Table 6), which was lower than the percentage of variance accounted for by the five components derived from the PCA.

Similarly to PC1, the factor loadings table indicated that Factor 1 was primarily associated with variables relating to emotional difficulties and specific mental health conditions, while Factor 2 was also almost identical to PC2, displaying strongest correlations with cognitive variables. Further, Factor 3 was also mainly associated with variables relating to emotional clarity (same as PC3), while comparably to PC4, prosocial skills loaded very strongly onto Factor 4. Interestingly, Factor 5 and Factor 6 did not display any loadings in the > 0.5 range, but both showed weaker associations (loadings between 0.3 and 0.5) with variables relating to acceptance and tolerance of emotions, as well as ADHD (hyperactivity and peer problems), similarly to the 'Hyperactivity' principal component. These results suggest that EFA may have over extracted factors due to minor correlations (i.e., statistical noise), with the last two factors

accounting for minimal variance in the dataset (see Table 6). This suggests that PCA was more appropriate for dimensionality reduction in preparation for subsequent cluster analysis, given that it has yielded fewer and more interpretable non-redundant components.

Table 5. Factor loadings of cognitive and mental health variables in EFA.

Variable	Factor					
	1	2	3	4	5	6
Cognition						
FICAT	0.226	0.629	0.328	0.007	0.152	-0.012
DCCST	0.142	0.733	0.343	-0.022	0.261	0.007
PSMT	-0.115	0.377	0.026	-0.029	0.105	0.128
LSWMT	-0.052	0.438	0.019	0.038	-0.113	0.002
PCPST	0.180	0.504	0.179	0.053	0.151	-0.036
PVT	-0.185	0.537	0.022	0.097	-0.092	-0.039
ORRT	-0.084	0.351	-0.063	-0.083	-0.233	-0.050
ERSQ						
Awareness	0.502	0.295	-0.391	-0.138	0.131	-0.033
Sensations	0.468	0.101	-0.528	-0.027	0.130	-0.076
Clarity	0.598	0.196	-0.538	-0.125	0.039	-0.158
Understanding	0.614	0.177	-0.496	-0.144	-0.055	-0.201
Acceptance	0.573	0.171	-0.013	-0.023	-0.284	0.431
Tolerance	0.500	0.220	0.006	-0.029	-0.376	0.390
Confrontation	0.569	-0.095	-0.092	0.111	-0.033	0.280
Self-Support	0.581	-0.003	-0.120	0.172	-0.079	0.045
Modification	0.714	-0.039	-0.041	0.220	-0.190	-0.064
SDQ						
Emotional Difficulties	0.696	-0.004	0.365	-0.043	-0.088	-0.242
Conduct Problems	0.223	0.027	-0.037	0.284	0.205	0.012
Hyperactivity	0.501	-0.176	0.055	-0.142	0.443	0.166
Peer Problems	0.352	0.014	0.140	0.285	-0.115	-0.302
Prosocial Skills	0.273	-0.023	-0.106	0.814	0.155	0.031
Impact	0.626	-0.267	0.271	0.125	0.034	0.050
E-SWAN						
ADHD	0.590	-0.198	0.076	-0.159	0.410	0.116
Depression	0.797	-0.250	0.240	-0.111	0.006	-0.031
Panic Disorder	0.684	-0.116	0.271	-0.276	-0.028	-0.026
Social Anxiety	0.619	-0.061	0.303	-0.062	-0.244	-0.285

Note. ‘Principal axis factoring’ extraction method was used in combination with a ‘none’ rotation. *EFA* = Exploratory Factor Analysis. *FICAT* = Flanker Inhibitory Control and Attention Test. *DCCST* = Dimensional Change Card Sort Test. *PSMT* = Picture Sequence Memory Test. *LSWMT* = List Sorting Working Memory Test. *PCPST* = Pattern Comparison Processing Speed Test. *PVT* = Picture Vocabulary Test. *ORRT* = Oral Reading Recognition Test. Strong correlations (above 0.5) are highlighted in bold.

Table 6. Percentage of variance explained by each factor in EFA.

Factor	SS Loadings	% of Variance	Cumulative %
1	6.33	24.33	24.3
2	2.41	9.26	33.6
3	1.73	6.64	40.2
4	1.15	4.42	44.7
5	1.02	3.92	48.6
6	0.79	3.05	51.6

Note. *SS* = Sum of Squares. *EFA* = Exploratory Factor Analysis. The *SS* loading is the sum of squared loadings for all variables contributing to a given component. This indicates how much of the variance in the original dataset is accounted for by that component, based on the contributions of each variable. An *SS* loading close to 1 indicates that the component explains a larger amount of variance in the data.

Comparison of PCA and EFA Models for Mental Health Variables Only

Both Principal Component Analysis (PCA) and Exploratory Factor Analysis (EFA) were conducted to examine the structure of the mental health variables. While EFA is traditionally more suitable for identifying latent constructs by modelling shared variance, PCA was ultimately chosen as the preferred method for dimensionality reduction due to providing fewer non-redundant variables appropriate for subsequent cluster analysis. Both EFA and PCA were conducted in jamovi (Version 2.3.28; The jamovi project, 2023).

The EFA was performed on 19 mental health variables, using Principal Axis Factoring with no rotation. The Bartlett's Test of Sphericity was significant ($\chi^2(171, N = 175) = 1563, p < .001$), suggesting that an EFA was appropriate. The Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy was 0.85, indicating that the data were factorable. A minimum factor loading of 0.5 was applied in order to highlight variables with only moderate or strong correlations to a given factor. The optimal number of factors to retain was determined using parallel analysis, which suggested a 5-factor solution. The factor loadings of each variable are presented in Table 7. Together, the first five factors explained 56.9% of the total variance in the dataset (see Table 8), which is lower than the percentage of variance explained by the four components derived from the PCA. Similarly to PC1, the factor loadings table indicated that Factor 1 was primarily associated with variables relating to emotion regulation difficulties, hyperactivity, impact of difficulties and specific mental health conditions. Factor 2 was also similar to PC2, displaying stronger correlations with variables relating to emotion regulation only. Further, Factor 3 was very strongly associated with Prosocial Skills (similar to PC3), while Factor 4 was primarily

related to Hyperactivity (similar to PC4). Importantly, no variables displayed loadings above 0.5 for Factor 5, with Acceptance showing the highest loading of 0.408.

Whilst the EFA produced four factors which closely resembled the dimensions of mental health yielded by PCA, the additional fifth factor lacked interpretability. This factor accounted for minimal variance within the dataset (see Table 8), and did not correspond to any distinct mental health domain, suggesting that EFA may have over extracted factors due to weaker correlations (i.e., statistical noise). Given that the purpose of these analyses was to reduce data redundancy in preparation for cluster analysis (as opposed to confirming latent constructs), the results suggest that PCA was more effective at capturing the key dimensions of variance.

Table 7. Factor loadings of each mental health variable.

Variable	Factor				
	1	2	3	4	5
ERSQ					
Awareness	0.502	0.459	-0.135	0.082	-0.016
Sensations	0.476	0.520	-0.016	0.145	-0.062
Clarity	0.608	0.566	-0.120	0.045	-0.160
Understanding	0.623	0.533	-0.147	-0.069	-0.215
Acceptance	0.574	0.054	-0.023	-0.323	0.408
Tolerance	0.503	0.057	-0.044	-0.439	0.375
Confrontation	0.567	0.050	0.120	-0.032	0.278
Self-Support	0.584	0.099	0.180	-0.078	0.037
Modification	0.716	0.003	0.221	-0.157	-0.074
SDQ					
Emotional Difficulties	0.688	-0.357	-0.053	-0.078	-0.247
Conduct Problems	0.222	0.040	0.274	0.184	0.013
Hyperactivity	0.507	-0.130	-0.133	0.530	0.192
Peer Problems	0.348	-0.132	0.266	-0.119	-0.294
Prosocial Skills	0.275	0.079	0.823	0.152	0.026
Impact	0.628	-0.370	0.126	0.087	0.035
E-SWAN					
ADHD	0.592	-0.150	-0.152	0.473	0.148
Depression	0.790	-0.320	-0.105	0.057	-0.029
Panic Disorder	0.676	-0.295	-0.281	-0.024	-0.020
Social Anxiety	0.618	-0.327	-0.077	-0.214	-0.304

Note. *ERSQ* = Emotion Regulation Skills Questionnaire. *SDQ* = Strengths and Difficulties Questionnaire. *E-SWAN* = Extended Strengths and Weaknesses Assessment of Normal Behaviour. *ADHD* = Attention Deficit Hyperactivity Disorder. Strong correlations (above 0.5) are highlighted in bold.

Table 8. Percentage of variance explained by each factor.

Factor	SS Loadings	% of Variance	Cumulative %
1	6.18	32.52	32.5
2	1.73	9.10	41.6
3	1.13	5.95	47.6
4	1.00	5.28	52.8
5	0.77	4.05	56.9

Note. SS = Sum of Squares. The SS loading is the sum of squared loadings for all variables contributing to a given factor. This indicates how much of the variance in the original dataset is accounted for by that factor, based on the contributions of each variable. An SS loading close to 1 indicates that the factor explains a larger amount of variance in the data.

A *k*-means cluster analysis was also performed on the five principal components identified via PCA (for comparison with the model included in the main text). The gap statistic and elbow method suggested that a 1-cluster solution was optimal for this dataset. This implies that reducing the whole dataset to only 5 components has likely compromised the variability that would otherwise allow for clear cluster separation evident in the model included in the main text (i.e., three well-separated clusters). These results provide further evidence that dimensionality reduction is not appropriate for cognitive variables in this dataset as retaining separate variables representing distinct cognitive processes allows for more meaningful group separation. Further, since applying clustering to principal components yielded from combined mental health and cognitive variables was not appropriate, the results also provide further rationale for treating mental health and cognition as separate concepts, despite being interrelated.

EFA of Cognitive Variables (For Information Purposes)

Note that an EFA was also performed on cognitive variables for comparison with the PCA model. Whilst the variable loadings were quite high for the first factor in the EFA, they were relatively low (all below 0.5) for the second factor (see Table 9). The factor structure was similar to that of the PCA (see Table 2). Nonetheless, separate cognitive variables were retained for subsequent analyses; therefore, this comparison was for transparency/information purposes only.

Table 9. Factor loadings of cognitive variables in exploratory factor analysis.

Variable	Factor	
	1	2
Flanker Inhibitory Control and Attention Test	0.722	-0.168
Dimensional Change Card Sort Test	0.879	-0.310
Picture Sequence Memory Test	0.350	0.139
List Sorting Working Memory Test	0.429	0.432
Pattern Comparison Processing Speed Test	0.561	-0.252
Picture Vocabulary Test	0.477	0.378
Oral Reading Recognition Test	0.270	0.448

Note. ‘Principal axis factoring’ extraction method was used in combination with a ‘none’ rotation. Strong correlations (above 0.5) are highlighted in bold.

Multinomial Logistic Regression (PCA-Based Clusters)

To investigate the underlying structure of the dataset and gain further insight into the dimensions that define each cluster, a multinomial logistic regression was conducted on clusters derived from mental health principal components and cognitive variables. The four mental health principal components and seven cognitive variables were entered into the analysis separately, as the model did not perform well when all variables were entered simultaneously. The reference group for all analyses was Cluster 1.

When cognitive variables were entered into the analysis, the overall model was statistically significant ($\chi^2(14) = 316, p < .001$) and explained almost 88% (Nagelkerke R^2) of the variance in clusters. Results of the Omnibus Likelihood Ratio Tests for individual predictors indicated that all cognitive variables, apart from the Picture Sequence Memory Test, were significant predictors of cluster membership (see Table 10). Multinomial logistic regression results showed that performance on the Picture Vocabulary Test significantly predicted cluster membership ($\beta = 1.91, p = .009, OR = 6.74, 95\% CI [1.63, 27.90]$), with higher scores

increasing the likelihood of being in Cluster 2 (versus Cluster 1). Further, higher scores on the Flanker Inhibitory Control and Attention Test ($\beta = -3.42, p = .012, OR = 0.03, 95\% CI [0.002, 0.47]$) and the Dimensional Change Card Sort Test ($\beta = -3.78, p < .001, OR = 0.02, 95\% CI [0.003, 0.17]$) significantly decreased the odds of being placed in Cluster 2. Similarly, individuals with higher scores on the Pattern Comparison Processing Speed Test were also significantly less likely to be members of Cluster 2 ($\beta = -3.90, p < 0.001, OR = 0.02, 95\% CI [0.002, 0.20]$). Finally, higher scores on the Picture Vocabulary Test ($\beta = 3.24, p < .001, OR = 25.45, 95\% CI [4.80, 134.93]$), Oral Reading Recognition Test ($\beta = 3.19, p < .001, OR = 24.36, 95\% CI [4.71, 125.93]$) and List Sorting Working Memory Test ($\beta = 1.61, p = 0.005, OR = 5.02, 95\% CI [1.62, 15.51]$) significantly predicted higher odds of being placed in Cluster 3 (compared to Cluster 1).

When mental health principal components were entered into the analysis, the overall model was also statistically significant ($\chi^2(8) = 47.4, p < .001$), and explained around 17% (Nagelkerke R^2) of the variance in clusters. While this is only a moderate proportion, the model still provided useful information, given the complexity of the underlying dimensions contributing to cluster membership. Results of the Omnibus Likelihood Ratio Tests for individual predictors indicated that all mental health principal components, apart from the Social Skills dimension, significantly predicted cluster membership (see Table 11). Multinomial logistic regression results revealed that individuals with higher scores on the Emotional Difficulties dimension (i.e., less emotional difficulties) were significantly less likely to be members of Cluster 2 compared to Cluster 1 ($\beta = -0.79, p < .001, OR = 0.45, 95\% CI [0.29, 0.70]$), whilst higher self-reported Emotional Clarity significantly increased the odds of being placed in Cluster 2 ($\beta = 0.60, p = .006, OR = 1.82, 95\% CI [1.18, 2.79]$). Further, higher scores on the Emotional Difficulties dimension (i.e., less emotional difficulties) significantly decreased the likelihood of Cluster 3 membership compared to Cluster 1 ($\beta = -0.92, p < .001, OR = 0.40, 95\% CI [0.24, 0.65]$). There was also a significant effect of Hyperactive Behaviour, with higher scores (i.e., less hyperactivity) significantly decreasing the odds of being placed in Cluster 3 ($\beta = -0.87, p < .001, OR = 0.42, 95\% CI [0.26, 0.67]$). Finally, individuals with higher Emotional Clarity were significantly more likely to be captured by Cluster 3 compared to Cluster 1 ($\beta = 0.76, p = .002, OR = 2.14, 95\% CI [1.34, 3.42]$).

Table 10. Omnibus Likelihood Ratio Tests for cognitive variables in PCA-based clusters.

Predictor	χ^2	df	p
Flanker Inhibitory Control and Attention Test	12.00	2	.002
Dimensional Change Card Sort Test	40.22	2	<.001
Picture Sequence Memory Test	5.95	2	.051
List Sorting Working Memory Test	11.16	2	.004
Pattern Comparison Processing Speed Test	35.62	2	<.001
Picture Vocabulary Test	34.73	2	<.001
Oral Reading Recognition Test	38.47	2	<.001

Note. χ^2 = Wald chi-square statistic. df = degrees of freedom. All scores were generated via the NIH Cognition Toolbox.

Table 11. Omnibus Likelihood Ratio Tests for mental health principal components.

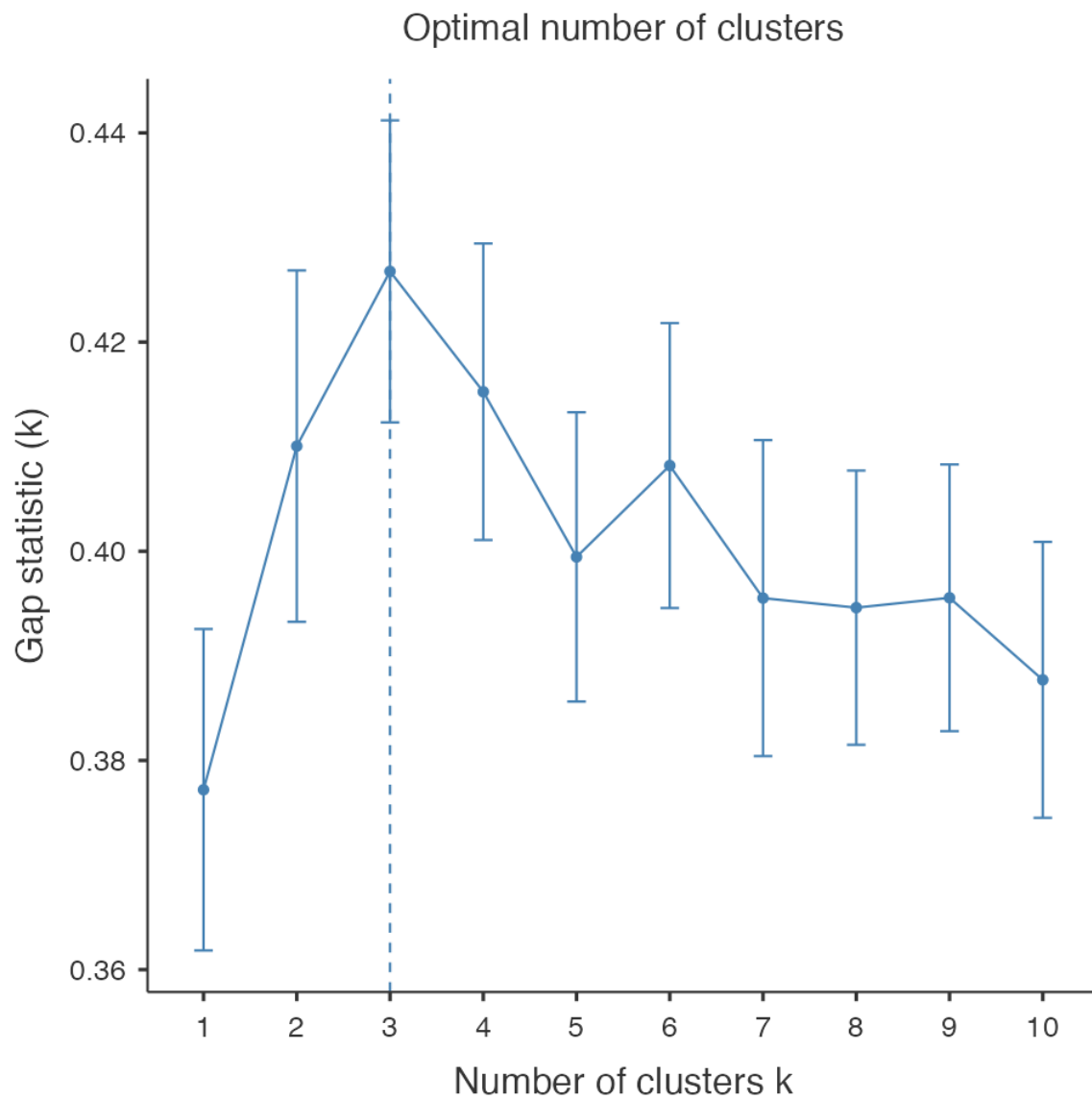
Predictor	χ^2	df	p
Emotional Difficulties	19.69	2	< .001
Emotional Clarity	11.95	2	.003
Social Skills	0.99	2	.608
Hyperactive Behaviour	15.93	2	< .001

Note. χ^2 = Wald chi-square statistic. df = degrees of freedom. Mental health principal components were derived from a combination of 19 mental health variables assessed via the Emotion Regulation Skills Questionnaire, Strengths and Difficulties Questionnaire and the Extended Strengths and Weaknesses Assessment of Normal Behaviour.

These data have been presented for transparency and comparison with results of the LDA presented in the main text of the thesis. The results of the multinomial logistic regression were very similar to that of LDA. However, LDA ultimately provided more interpretable information about the underlying structure of the dataset, and performed well even when both cognitive and mental health variables were simultaneously entered into the analysis.

Appendix C. Additional Figures

Figure 1. Optimal number of clusters (derived from mental health components and cognitive variables).



Note. A three-cluster structure was suggested by the gap statistic and elbow method as the optimal solution.