Investigating trait impulsivity: behavioural and neural differences in a non-clinical population

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The aim of this thesis was to conduct a comprehensive investigation of impulsivity, including rapid-response and reward-delay impulsivity dimensions. A behavioural study was conducted to examine the sensitivity of impulsivity measures to differences between low and high impulsivity groups, based on impulsivity questionnaires. Results showed that the proposed measures were sensitive to differences between groups and that combining impulsivity dimensions provided a better predictor of impulsivity level than each dimension alone. We then tested whether a three-factor model of impulsivity, would benefit or not from the inclusion of a psychometric measure of reward-delay. Although results favoured a three-factor model, including the reward-delay psychometric measure did not improve the model fit, and showed that rapid-response and reward-delay impulsivity are two major dimensions which contribute independently to impulsivity. Potential differences in the neural correlates of response inhibition and delay discounting between the two groups were examined using MEG. Results suggested high impulsivity individuals might show an attentional processing deficit, as indicated by smaller M1 components and less alpha suppression in posterior regions in the two tasks. Regarding response inhibition, the M2 component was found to be reduced in individuals scoring high, possibly reflecting less efficiency. The high impulsivity group engaged frontal networks more during the STOP-M3 component only, possibly as a compensatory strategy. Increased preference for immediacy was observed in high impulsivity individuals, as reflected by larger Immediate-M2 amplitudes. Decreased delta and theta band power was observed in high impulsivity individuals, suggesting a possible deficit in frontal pathways involved in motor suppression. Increased delta and theta power were observed in frontal regions in high impulsivity individuals, while beta band power was found to be supressed, suggesting an increased sensitivity towards reward-related cues. The experiments described here illustrated how trait impulsivity relates to differences in the behavioural and neural correlates of cognitive processes.

Keywords: response inhibition, rapid-response, reward-delay, delay discounting, magnetoencephalography.

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LIST OF ABBREVIATIONS

ACC Anterior cingulate cortex

ADHD Attention deficit hyperactivity disorder

ANOVA Analysis of variance

ASPD Antisocial personality disorder

BIS-11 Barratt Impulsiveness Scale

BIS/BAS Behavioural Inhibition System / Behavioural Activation System scales

BSD Bipolar spectrum disorder

CFI Comparative fit index

DDT Delay discounting task

DICS Dynamic imaging of coherent sources

EEG Electroencephalography

EPQ Eysenck Personality Questionnaire

ERF Event related field

ERP Event related potential

FFT Fast Fourier Transform

fMRI Functional magnetic resonance imaging

GNGT Go/No-Go Task

GNGSST Go/No-Go/Stop-Signal Task

HPI Head Position Indicator

ICA Independent component analysis

IFG Inferior frontal gyrus

LCMV Linearly constrained minimum variance

MANCOVA Multivariate analysis of covariance

MCQ Monetary choice questionnaire

MEG Magnetoencephalography

MFG Middle frontal gyrus

MID Monetary incentive delay task

mm Millimetre

MNI Montreal Neurological Institute

MRI magnetic resonance imaging

ms Milliseconds

PCA Principal component analysis

PCC Posterior cingulate cortex

PFC Prefrontal cortex

pre-SMA Pre-supplementary motor area

RI Response inhibition

RMSEA Root mean square error of approximation

RT Response time

s Seconds

SD Standard deviation

SE Standard error

SEM Structural equation modelling

SFG Superior frontal gyrus

SKIP Single key impulsivity paradigm

SMA Supplementary motor area

SPSS Statistical Package for Social Sciences

SST Stop-Signal Task

SZ Schizophrenia

TCIP Two-choice impulsivity paradigm

TLI Tucker-Lewis index

TFR Time-frequency representation

UPPS Urgency, Premeditation, Perseverance, Sensation Seeking Impulsive Behaviour scales

CHAPTER 1: General introduction.

1.1. Introduction.

According to a widely used definition, impulsivity is “a predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions to the impulsive individual or to others” (Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001; p. 1784). Although researchers agree impulsivity is not a uni-dimensional, but a multifaceted construct (Alloy et al., 2009; Dawe & Loxton, 2004; Dawe, Gullo, & Luxton, 2004; MacKillop et al., 2016; Swann, 2010), the classification of the different personality characteristics associated with impulsive behaviour is still not clear (Gullo, Loxton, & Dawe, 2014; Hamilton et al., 2015).

In the 1970’s, Eysenck and Eysenck started debating how impulsivity could be classified within the Big Three (Psychoticism, Extraversion, Neuroticism) framework, and at first, impulsivity was included as a subscale of Extraversion (Eysenck, 1967; Whiteside & Lynam, 2001). Years later, they revisited the original idea and a new factor analysis revealed two factors: Impulsivity, included in Psychoticism, and Venturesomeness, containing risk-taking and sensation seeking items, included in Extraversion (Eysenck & Eysenck, 1985). Subsequent models of personality, such as the four-factor model proposed by Cloninger et al. (1991) and the Alternative Big-Five by Zuckerman, Kuhlman, Thornquist, & Kiers (1991), classified impulsivity as a component of the higher-level Sensation Seeking factor (Zuckerman & Glicksohn, 2016).

These authors tried to classify impulsivity within theoretical frameworks of personality, however, these models have not been widely accepted (Whiteside & Lynam, 2001). Instead, other researchers took a different approach, by aiming to describe impulsivity as a construct itself, which comprises several separated dimensions (Whiteside, Lynam, Miller, & Reynolds, 2005). In recent years, exploratory factor analysis, hypothesis-testing confirmatory factor analysis and Structural Equation Modelling (SEM) methods, for example, have facilitated investigation of the latent structure of impulsivity.

A principal component analysis conducted only on behavioural paradigms of impulsivity by Reynolds and colleagues (2006), identified two dimensions: “impulsive disinhibition’’ comprising the Go/No-Go task (GNGT) and Stop-Signal task (SST), both measuring response inhibition; and ‘‘impulsive decision-making’’, consisting of the Delay-Discounting task, measuring the inability to delay gratification, and the Balloon Analog Risk Task, assessing risk-taking behaviours. In line with these results, a recent study by MacKillop and colleagues (2016), conducted a confirmatory factor analysis on both behavioural and self-report measures of impulsivity and found three latent constructs: impulsive choice, comprising reward-directed tasks which measure the inability to delay reward; impulsive action, consisting

of behavioural tasks measuring response inhibition; and impulsive personality traits, which included self-report measures of impulsivity.

These findings are consistent with the general agreement between researchers, that impulsivity comprises at least two distinct dimensions: rapid-response impulsivity (also referred to as response inhibition or impulsive action) and reward-delay impulsivity or impulsive choice (Alloy et al., 2009; Dawe & Loxton, 2004; Dawe, Gullo, & Luxton, 2004; Swann, Lijffijt, Lane, Steinberg, & Moeller, 2010). The study by MacKillop et al. (2016) seems to partly confirm this hypothesis, the three-factor model provided the best fit to the data, which included: impulsive personality traits, reflecting self-reports of impulsivity, impulsive action, behavioural measures of response inhibition and impulsive choice, assessing delay discounting of rewards. They did not include self-report measures of reward sensitivity, risk-taking or sensation seeking, which as mentioned earlier, authors like Eysenck, Cloninger and Zuckerman, considered sensation seeking to be a fundamental part of impulsive behaviours.

The experiments conducted in this thesis aim to deliver a more comprehensive analysis, in Experiment 2 for example, the hypothesis that a three-factor model of impulsivity was tested, consisting of impulsive action, impulsive choice and impulsive personality traits, would benefit from including a psychometric measure of reward-delay impulsivity and still be differentiated into three latent constructs. Using common factor analysis, the aim was to identify the least number of factors that can explain common variance, this analysis included both behavioural and psychometric measures of rapid-response and reward-delay impulsivity, along with sensation seeking. Then the resulting model was confirmed using a Structural Equation Modelling approach, to help clarifying the classification of the different personality characteristics associated with impulsive behaviour and its dimensionality.

Another problematic matter discussed in previous studies is the relationship between self-report and behavioural measures of impulsivity, as correlational and factorial analyses have reported very inconsistent results regarding their association (e.g., Aichert et al., 2012; Enticott, Ogloff, & Bradshaw, 2006; Horn, Dolan, Elliott, Deakin, & Woodruff, 2003; Keilp, Sackeim, & Mann, 2006; Lijffijt, Bekker, Quik, Bakker, Kenemans, & Verbaten, 2004; Malesza & Ostaszewski, 2016; Reynolds et al., 2006; Rodriguez-Fornells, Lorenzo-Seva, & Andrés-Pueyo, 2002; Spinella, 2004). The question of whether these two types of measures truly assess the same construct is extremely relevant, as both are generally used in behavioural and neuroimaging studies (e.g., Aichert et al., 2012; Dimoska & Johnstone, 2007; Lansbergen et al., 2007; Lijffijt et al., 2004; Wilbertz et al., 2014), and therefore, it will also be investigated here. The experiments conducted in this thesis are intended to be as inclusive as possible, by investigating not only one but several measures, both behavioural and psychometric, of each

dimension of impulsivity, to further elucidate the different and contradictory results reported to date.

As previously mentioned, impulsivity is known to characterise several mental illnesses such as ADHD; Nigg, 2001), drug addiction (Jentsch & Taylor, 1999; Bari & Robbins, 2013), obsessive compulsive disorder (OCD; Chamberlain et al., 2007), schizophrenia (SZ; Reddy et al., 2014) and especially, bipolar spectrum disorders (BSD; Strakowski & Fleck, 2009). Yet, what may be less clear is whether each of the two dimensions of impulsivity also characterise certain mental illnesses or not. Interestingly, both rapid-response and reward-delay impulsivity have been reported in in bipolar patients (Strakowski et al, 2010; Swann, Lijffijt, Lane, Steinberg, & Moeller, 2009; Swann, 2010) and drug abusers (Bari & Robbins, 2013). On the one hand, rapid-response impulsivity alone, as assessed by an impairment in behavioural inhibition, has been found to predict alcohol- and drug-related use in an adolescent sample (Nigg et al., 2006). On the other hand, elevated reward sensitivity has been associated with a 6-times greater probability of being diagnosed with BSDs compared to those with moderate sensitivity among a sample of students (Alloy et al., 2006). Furthermore, Rubio et al. (2008) reported that in adults the inability to delay reward was an indicator of early alcohol consumption, whereas poor performance in response inhibition tasks was an indicator of consumption maintenance and of risk for alcoholic dependence. These findings, in addition to results from the previous clinical studies mentioned earlier, could suggest that both reward-delay and rapid-response impulsivity, represent shared vulnerabilities to some mental illnesses.

The main objective of this thesis is to examine the neural mechanisms of rapid-response and reward-delay impulsivity separately, in individuals scoring high and low on impulsivity questionnaires. Despite this being the main objective, it is also crucial to ensure that all the measures used for this purpose are the most precise and specific ones. Accordingly, the measures used in the design of the neuroimaging study, such as the questionnaires that will classify participants into high or low impulsivity scorers, or the behavioural paradigms used to examine the neural correlates of each process, were carefully examined for suitability.

For this purpose, the behavioural measures of impulsivity will be tested in an undergraduate student sample in Experiment 1. The aim of the behavioural study was to compare rapid-response and reward-delay task measures of impulsivity in low-scoring and high-scoring individuals, as measured by self-reports, to examine whether these measures are sensitive enough to pick up the differences between them. In this experiment, correlational analyses were also conducted to investigate the association between behavioural paradigms and self-reports, expecting this will provide a better assessment of impulsivity as a multifaceted

construct. A factor-analytic approach was conducted to clarify whether a three-factor model of impulsivity would benefit from including a psychometric measure of reward-delay impulsivity, and still be differentiated into three latent constructs. This experiment included relevant, well-validated and widely used behavioural and self-report measures of impulsivity.

Finally, any behavioural differences found during the behavioural study will be examined further by testing the same tasks using a neuroimaging technique, magnetoencephalography (MEG). Specifically, potential differences in neural mechanisms between high and low impulsivity individuals, as measured by self-reports, during the response inhibition and delay discounting tasks tested previously will be investigated. I believe the literature review and experiments conducted will provide new evidence and clarify previous debates on impulsivity as a multidimensional concept and as potential link to risk for psychopathology.

1.2. Impulsivity

1.2.1. Impulsive personality traits.

The rationale to classify impulsivity measures in this thesis follows the structure of the hypothesis-testing confirmatory factor analysis by MacKillop et al. (2016). One of the three factors of this model was labelled 'Impulsive Personality Traits' and included the subscales of two self-report measures of impulsivity, the Barratt Impulsiveness Scale (BIS-11, Patton, Stanford, & Barratt, 1995) and the UPPS Impulsivity Scale (Whiteside & Lynam), as indicators. The other two factors, "Impulsive Choice" and "Impulsive Action", comprised behavioural measures of reward-delay impulsivity and response inhibition, respectively. This section will describe some of the most widely used self-report measures of impulsivity, how they were designed, what they intend to assess, and a review of results from clinical studies.

1.2.1.1. The Eysenck Personality Questionnaire

The Eysenck Personality Questionnaire (EPQ; Eysenck & Eysenck, 1975), was one of the first self-report measures to assess impulsive behaviours. Clinical studies have found high scores on Psychoticism to be associated with alcohol and/or drug abuse (Eysenck & Eysenck, 1977; Sher, Bartholow, & Wood, 2000; Zuckerman, 1993), gamblers to show higher scores than non-gamblers (Sharma, Markon, & Clark, 2014), and to even serve as a predictor of substance use disorders in high scoring individuals compared to those scoring low on this subscale (Sher et al., 2000).

1.2.1.2. The Barratt Impulsiveness Scale

The Barratt Impulsiveness Scale (BIS-11, Patton, Stanford, & Barratt, 1995), is possibly the most frequently used impulsivity questionnaire in research and clinical studies (Stanford et al., 2009). Although it has gone through several versions, it was originally designed to: "(1) to describe impulsiveness in normal persons, (2) to arrive at the role of impulsiveness in psychopathology, and (3) to develop a personality framework within which impulsiveness as a personality trait could be related to other traits" (Barratt et al., 1994, p. 63). The BIS-11 is a 30-item questionnaire measuring the personality and behavioural construct of impulsivity. This version was designed based on a principal component analysis (PCA) conducted on the previous version's (BIS-10) data, which showed three components: Motor Impulsiveness, Non-Planning Impulsiveness and Attentional Impulsiveness.

Significantly higher scores on the BIS-11 have been reported in adults with substance abuse compared to controls (Lane, Moeller, Steinberg, Buzby, & Kosten, 2007; Bond, Verheyden, Wingrove, & Curran, 2004), in adults diagnosed with ADHD compared to adults with no history of psychopathology (Malloy-Diniz, Fuentes, Leite, Correa, & Bechara, 2007), in pathological gamblers with history of ADHD compared to gamblers with no previous history (Rodriguez-Jimenez et al., 2006), in adults with history of suicidal attempts compared to those with no previous history (Dougherty et al., 2004), in adult bipolar patients compared to those diagnosed with unipolar depression (Peluso et al., 2007; Swann et al., 2008), and even in unaffected relatives of bipolar patients compared to healthy controls (Sanches et al., 2014).

1.2.1.3. Other questionnaires and the UPPS Impulsivity Scale

Although the BIS-11 is a widely used scale to measure impulsivity, Whiteside and Lynam (2001) suggested that the BIS-11 and similar self-report measures to assess impulsivity, might not be sensitive enough to gauge all aspects of impulsivity, and therefore, they intended to design a more complete psychometric measure. These authors based their hypotheses on the well-established Five-Factor Model of personality (FFM) by Costa & McCrae (1990) and conducted an extensive exploratory factor analysis on nine frequently used self-report measures of impulsivity: EASI-III Impulsivity Scales (Buss & Plomin, 1975), Dickman's Functional and Dysfunctional Impulsivity Scales (Dickman, 1990), Barratt Impulsiveness Scale-11 (BIS-11; Patton, Stanford, & Barratt, 1995), I-7 Impulsiveness Scale (I-7; Eysenck, Pearson, Easting, & Allsopp, 1985), Personality Research Form Impulsivity Scale (PRF; Jackson, 1967), Multidimensional Personality Questionnaire Control Scale (MPQ; Tellegen, 1985), Temperament and Character Inventory (TCI; Cloninger et al., 1991), Sensation Seeking Scale (SSS; Zuckerman 1994), and the Revised NEO Personality Inventory (NEO-PI-R; Costa & McCrae, 1992). The exploratory factor analysis identified four factors which

constitute the UPPS Impulsivity Scale: Urgency, (lack of) Premeditation, (lack of) Perseverance, and Sensation Seeking; the authors suggested these four factors to be four different personality traits which lead to analogous behaviours (Whiteside & Lynam, 2001).

Some of these subscales, specifically Urgency, (lack of) Premeditation and Sensation Seeking, have been found to differentiate adults diagnosed with borderline personality disorder, pathological gambling and alcohol abuse, from a control group (Whiteside & Lynam, 2005). The Urgency subscale has also been found to influence drinking problems and binge eating, adults with higher scores on this subscale being found to be more likely to engage in this type of behaviours (Smith et al., 2007; Stojek et al., 2014); while Sensation Seeking has been found to influence the frequency of engaging in these behaviours, specifically (Smith et al., 2007). Sensation Seeking has also been reported to be significantly associated with alcohol consumption (Lynam & Miller 2004; Murphy & MacKillop, 2012).

1.2.1.4. The Behavioural Inhibition System/Behavioural Activation System

Although several measures have been designed to assess impulsivity and more specifically, rapid-response impulsivity, such as the BIS-11 and Eysenck's Impulsiveness Scale (Eysenck & Eysenck, 1991), there are not as many intended to specifically assess reward-delay impulsivity. In this regard, Carver and White (1994) designed the Behavioural Inhibition System/Behavioural Activation System (BIS/BAS) scales to measure reward sensitivity (Dawe & Loxton, 2004), based on the reinforcement sensitivity theory (RST) of personality (Gray & McNaughton, 2000), which proposes the existence of three different systems: (a) the fight-flight-freeze system (FFFS), suggested to be responsible for behaviours towards aversive stimuli, (b) the behavioural approach system (BAS), responsible for behaviours towards appetitive/positive stimuli, and (c) the behavioural inhibition system (BIS) which mediates goal conflicts between and within the two other systems (FFFS-BAS, FFFS-FFFS, BAS-BAS) (Alloy et al., 2009; Duek et al., 2014).

A principal-component analysis of the BIS/BAS scales found four components: Behavioural Inhibition, Reward Responsiveness, Drive, and Fun Seeking (Carver & White, 1994). However, other factor analyses on this scale reported the BAS Fun Seeking subscale to load on both reward-delay and rapid-response impulsivity factors, and to be associated with rapid-response more than with reward-delay impulsivity (Caseras et al., 2003; Zelenski & Larsen, 1999). In clinical studies, bipolar patients have been reported to show higher scores than controls on the BIS/BAS (Alloy et al., 2008, 2009; Urosevic et al., 2008), while elevated scores have also been associated with a 6-times greater probability of being diagnosed with bipolar spectrum disorders compared to those with moderate BAS sensitivity among a sample of students (Alloy et al., 2006).

The BAS/reward hypersensitivity model proposes that a hypersensitive BAS can lead to an exaggerated approach towards reward and goal cues (Molz et al., 2013), and that BAS hyperactivity may result in impulsive decision-making (Mason, O'Sullivan, Blackburn, Bentall, & El-Deredy, 2012). When a reward cue activates the hyperreactive BAS, anticipation of this reward may be responsible for generating an impulsive "state" (Bari & Robbins, 2013) which influences decision-making. BAS hypersensitivity towards rewards may result in an inability to delay gratification and, thus, impulsive decision-making.

1.2.1.5. Other questionnaires of reward-delay impulsivity

Also designed to measure reward sensitivity is the Sensitivity to Reward (SR) subscale from the Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ; Torrubia, Avila, Molto, & Caseras, 2001). Studies using the SPSRQ and BIS/BAS scales in females have reported an influence of reward sensitivity on problematic drinking and dysfunctional eating (Dawe & Loxton, 2004).

Although MacKillop and colleagues' study (2016) found self-reports to load only on one factor, this could be explained by the absence of questionnaires assessing reward-delay impulsivity in the analyses. Studies conducting factor analyses on both types of self-report measures, rapid-response and reward-delay impulsivity, have confirmed that psychometric measures of rapid-response impulsivity, such as the BIS-11 (Patton, Stanford, & Barratt, 1995), the Eysenck Impulsiveness Scale (Eysenck & Eysenck, 1991), the Novelty Seeking Scale (Cloninger Przybeck, Svrakic, & Wetzel, 1994), and the Sensation-Seeking Scales (Zuckerman, 1994) correlate with each other and load on the same factor; while measures of reward sensitivity, the BIS/BAS Scales (Carver & White, 1994) and the Sensitivity to Reward scale (Torrubia et al., 2001) load on a different factor which correlates with the rapid-response impulsivity one (Alloy et al., 2009; Dawe, Gullo, & Loxton, 2004).

This distinction is consistent with results from factor analyses on behavioural tasks only, which have distinguished between response inhibition and reward-delay paradigms (e.g., Dougherty et al., 2003; Reynolds et al., 2006). The following subchapters will explain in detail how the two dimensions of impulsivity are assessed using laboratory behavioural tasks, will summarise the results found when these measures are used in clinical and neuroimaging studies and will explain the possible relationships between these paradigms and the self-report measures described here.

1.2.2. Rapid-response impulsivity.

The concept of rapid-response impulsivity refers to the inability to withhold prepotent responses, widely referred to as an impaired response inhibition. In this section, the rapid-response impulsivity construct, as assessed by behavioural paradigms, along with results from clinical and neuroimaging studies using these tasks, and the relationship between self-report measures of impulsivity and response inhibition tasks, are described in detail.

The first conceptualisations of inhibition were developed by philosophers such as Plato and Descartes, inhibition was associated to the individual's will and self-awareness (Bari & Robbins, 2013). They believed that only with high levels of consciousness and self-awareness, an individual was able to use inhibitory control over one's own behaviour (Bari & Robbins, 2013). In the scientific literature, inhibition was first investigated by physiologists at the beginning of the 19th century. Their experiments focused on the stimulation of certain brain areas and vagal nerves, which they found to inhibit spinal reflexes and affect heart rate, respectively. These findings marked a starting point for the development of theories on inhibitory action (Bari & Robbins, 2013; Gaskell, 1886; Weber & Weber, 1966). The beginning of the 20th century saw great progress of work on inhibition, for example, Pavlov, examined inhibition in the context of conditioned reflexes. Freud investigated inhibition and defined it 'the expression of a restriction of an ego-function" (Freud, 1959; p. 4) and Eysenck worked on the differences between introverts and extraverts, which included the concept of inhibition, and is regarded as a fundamental step towards the inclusion of this concept in personality research (Bari & Robbins, 2013).

One of the main objectives of the research on inhibition is to establish which brain regions are responsible for the ability to withhold prepotent responses, or successful response inhibition. Although lesion experiments in both animals and humans, along with electrical stimulation methods, were used in the past for this purpose (Bari & Robbins, 2013; Delgado, 1964), modern approaches include the use of behavioural paradigms specifically designed to measure prepotent response inhibition, such as the Go/No-Go Task and Stop-Signal Task.

These paradigms allow the investigation of the neural circuitry responsible for response inhibition and are also widely used as a complementary measure to questionnaires in clinical studies and in personality research. Compared to psychometric instruments, laboratory behavioural tasks are regarded as more objective measures of impulsive behaviours, and specifically, of inhibitory mechanisms. Questionnaires depend on the accurate recollection of one's behaviour, honest answers and own self-perception, focusing on trait impulsivity as manifested across time and different situations (Dougherty et al., 2005); while behavioural

paradigms are designed to assess specific cognitive processes during a limited and exact moment in time (Cyders & Coskunpinar, 2011).

1.2.2.1. Behavioural measures of rapid-response impulsivity

Response inhibition has been defined as “the ability to deliberately suppress dominant, automatic, or prepotent responses” (Friedman & Miyake, 2004, p. 104) and research on prepotent response inhibition has generally employed the following tasks: The Go/No-Go task (GNGT; Rubia et al., 2001), a well-validated paradigm in which participants respond by pressing a key to a go cue but withhold responses to no-go cues (Hummer et al., 2013); the stop-signal task (SST; Logan, 1994), which is similar to the go/no-go task, with the main difference being that in some trials the go-cue is followed by a stop-signal cue, which instructs the participant to cancel the initiated response; the Stroop task (Stroop, 1935), which involves participants to name the ink colour in which a name of a colour is printed and the anti-saccade task (Hallett, 1978), which requires an individual to either suppress a reflexive saccade or to execute a reflexive pro-saccade toward a certain cue.

However, there is no general agreement among researchers on whether each of these tasks measure response inhibition only, and not also attention, working memory or conflict resolution (Chambers et al., 2009; Nigg, 2000). A meta-analysis by Criaud and Boulinguez (2013) showed that the Go/No-Go and Stop-Signal tasks are used equally often, and almost interchangeably, as no convincing reasons are typically provided for selecting one over the other (Nigg, 2000). It has been argued that the SST contains a higher load on response inhibition than the GNGT, because it requires the cancellation of a response which has already been started, and thus, it is a more precise paradigm to assess RI than the go/no-go task. Alternatively, it has also been argued that each task measures different processes: the GNGT assesses “action restraint” because it measures the inhibition of a planned response and the SST measures “action cancellation” because it assesses the inhibition of an initiated response (Bari & Robbins, 2013; Eagle, Bari, & Robbins, 2008; Schachar et al., 2007). This distinction is supported by growing evidence from neuroimaging studies (e.g., Swick et al., 2011, Sebastian et al. 2013, Dambacher et al., 2014a) which have shown different as well as common patterns of neural activations when both paradigms are examined. Thus, the SST may not be more precise than the GNGT when measuring RI, as both tasks might measure different aspects of inhibitory processing.

Results from clinical studies show response inhibition (RI), as measured by different response inhibition (RI) behavioural tasks, to be impaired in patients with ADHD, drug addiction or BSDs (Frangou, Haldane, Roddy, & Kumari, 2005; Giakoumaki et al., 2007; Hajek, Alda, Hajek, & Ivanoff, 2013; Larson, Shear, Krikorian, Welge, & Strakowski, et al., 2010). Previous studies

have found response inhibition to be impaired not only in affected relatives of BSD, OCD, SZ or ADHD patients, but also in unaffected relatives of these patients (Aron & Poldrack, 2005; Bari & Robbins, 2013; Chamberlain et al., 2007; Chambers, Garavan, & Bellgrove, 2009; Ersche et al., 2012; Frangou et al., 2005; Giakoumaki et al., 2007; Hajek et al., 2013; Menzies et al., 2007; Vink, Ramsey, Raemaekers, & Kahn, 2006; Zalla et al., 2004) which suggests that there might be a genetic component that may lead to a predisposition.

Task performance, as measured by commission errors on the Stop-Signal task, has been shown to be reduced in impulsive individuals compared to healthy controls (Bari & Robbins, 2013), while reaction time to stop signals (SSRT) has been found to be longer in patients with different pathologies such as BSDs, ADHD (Lijffijt, Kenemans, Verbaten, & van Engeland, 2005; Verbruggen & Logan, 2008), and drug addiction (Goudriaan et al., 2006; Verbruggen & Logan, 2008), compared to healthy controls. Interestingly, reduced task performance, as measured by slower SSRT, has also been found to predict dependency to alcohol and other drugs in adolescents at higher risk (Nigg et al., 2006); while an impaired performance on the SST, has been reported to predict alcohol dependence (Rubio et al., 2008).

Furthermore, impaired response inhibition, as measured by performance on RI behavioural paradigms, has also been related to aggravating conditions in BSDs (Bari & Robbins, 2013), such as suicide attempts (Swann, Dougherty, Pazzaglia, Pham, & Moeller, 2005), criminal offences (Swann, Lijffijt, Lane, Kjome, Steinberg, & Moeller, 2011) and substance addiction (Swann, Dougherty, Pazzaglia, Pham, & Moeller, 2004). These results suggest an association between rapid-response impulsivity and course of illness (Lijffijt, Lane, Moeller, Steinberg, & Swann, 2014), which would suggest that rapid-response impulsivity may serve as a marker of risk for developing a mental disorder (e.g., Aron & Poldrack, 2005; Hajek et al., 2013; Hidiroglu et al., 2015; Nigg et al., 2006; Rubio et al., 2008; Swann et al., 2004; 2005; 2009; 2011).

1.2.2.2. The relationship between behavioural and self-report measures of impulsivity

To conceptualise the multi-faceted construct of impulsivity, factorial models of impulsivity have greatly contributed to the literature on the relationship between self-report and behavioural measures. Aichert and colleagues (2012) for example, tested whether performance on tasks designed to measure response inhibition (the Stop-Signal task, the Go/No-Go task, the Stroop task and the antisaccade task) fell into the same construct of impulsivity, labelled ‘prepotent response inhibition’, and found all four tasks to contribute significantly to this latent construct. Then, a Structural Equation Modelling (SEM) approach was used to determine if the ‘trait impulsivity’ construct, as measured by the BIS-11 sum score, was causally associated to the ‘prepotent response inhibition’ construct, comprised by the four RI tasks. Results showed that

higher levels of trait impulsivity predicted reduced task performance. Although the model was well fitted statistically, trait impulsivity only explained 12% of variance of the 'prepotent response inhibition' construct (Aichert et al., 2012).

Previous studies have reported significant associations between the UPPS Impulsive Scale and both Go/No-Go and Stop-Signal tasks (Aichert et al., 2012). Theoretically, the Urgency subscale is linked to response inhibition and some evidence seem to support this idea. Gay, Rochat, Billieux, d'Acremont and Van der Linden (2008), for example, found a positive correlation between Urgency and commission errors on the Go/No-Go task. Cyders and Coskunpinar (2011) reported that the Urgency, (lack of) Planning and (lack of) Perseverance subscales of the UPPS significantly correlated with prepotent response inhibition. In a meta-analysis, a low effect size between Urgency and the Stop-Signal task was found (Sharma, Markon and Clark, 2014), while lack of Perseverance and lack of Planning showed a moderate effect size with this paradigm. Furthermore, inter-individual variability in the Stop-Signal task was best explained by the Urgency subscale of the UPPS (Wilbertz et al., 2014). Significant associations have been reported between commission errors on the GNGT and BIS-11 scores (Aichert et al., 2012; Enticott, Ogloff, & Bradshaw, 2006; Reynolds et al., 2006; Keilp, Sackeim, & Mann, 2006; Spinella, 2004). However, non-significant correlations have also been reported (Horn, Dolan, Elliott, Deakin, & Woodruff, 2003).

For studies investigating the relationship between the Stop-Signal task and impulsivity questionnaires, conflicting findings have been reported. Logan, Schachar and Tannock, (1997; using the Eysenck Personality Inventory, EPI) and Marsh et al. (2002; using the Eysenck 17) found a correlation between reduced task performance and higher scores on these self-report measures of impulsivity. Reduced performance on the SST has also been reported to correlate negatively with scores on the Sensitivity to Reward scale, while correlating positively with the Sensitivity to Punishment scale (Avila & Pacet, 2001). In contrast, other studies investigating the associations between task performance on the Stop-Signal task and scores on the BIS-11, EPI or the Eysenck 17 questionnaires, have not found significant associations (Rodriguez-Fornells et al., 2002; Cheung et al., 2004; Lijffijt et al., 2004; Keilp et al., 2005; Enticott et al., 2006; Reynolds et al., 2006; Aichert et al., 2012).

From these findings, it is still not clear which aspects of impulsivity, as measured by questionnaires, are related to response inhibition (Dick et al., 2010). Researchers have suggested that this is because behavioural approaches measure task performance during a limited and exact moment in time, while questionnaires focus on self-reported trait impulsivity manifested across time and different situations (Cyders and Coskunpinar, 2011; Lane, Cherek, Rhoades, Pietras, & Tcheremissine, 2003; Reynolds et al., 2006; Swann et al., 2010;

Dougherty et al., 2014). Altogether, we consider it necessary to examine these relationships further by conducting behavioural studies and factor-analytic approaches, which are described chapters 3 and 4, respectively.

1.2.3. Reward-delay impulsivity.

As previously discussed, factor-analytic approaches on behavioural tasks have found impulsivity to consist of two different dimensions: one comprised by response inhibition tasks, such as the Go/No-Go and Stop-Signal tasks, and the second factor comprised by tasks measuring the ability to delay reward, referred to as reward-delay impulsivity, impulsive choice or temporal discounting (Dougherty et al., 2003; MacKillop et al., 2016; Reynolds et al., 2006). In this section, the reward-delay impulsivity construct, as assessed by behavioural paradigms, along with results from clinical and neuroimaging studies using these tasks, and the relationship between self-report measures of reward sensitivity and reward-delay tasks, are described.

A comprehensive review by Ainslie (1975) illustrated reward-delay impulsivity, or impulsive choice as the author called it, with the story of Adam and Eve, which provides a very clear example of this behaviour. In day-to-day situations, human beings and more frequently, impulsive individuals, prefer an immediate gratification over a more advantageous gratification at a future time, even if they are aware of the negative consequences such decision might cause. It has been suggested that the anticipation of a reward may be responsible for generating an impulsive “state” (Bari & Robbins, 2013) which influences decision-making. In other words, heightened sensitivity towards rewards may result in an inability to delay gratification and, thus, in impulsive decision-making. As the delay until a reward is obtained increases, the value of a future reward given, decrease. This is known as temporal discounting (Ainslie, 1975).

Reward-delay impulsivity can be measured using reward-directed tasks or decision-making tasks, but more precisely, using delay discounting paradigms. Such task requires participants to choose between either smaller but immediate, or larger, but delayed rewards and intends to measure temporal discounting (Ainslie, 1975). For example, the delay discounting task by Kirby and colleagues (1999), consists of hypothetical choices between a smaller immediate reward (£11–£80) and a larger reward (£25–£85) delayed between 7 and 186 days. From a subject’s choices, a discounting rate can be calculated. This rate is the amount at which the subject is indifferent between the immediate and delayed option, and it has been reported to be stable over time and consistent across species (Bari & Robbins, 2013).

Compared to self-reports of reward sensitivity, such as the BIS/BAS scales or the Sensitivity to Reward subscale from the SPSRQ, delay discounting tasks are likely to be more precise measures of the ability to delay gratification, regardless of how sensitive individuals are to rewards, which nevertheless, also contributes to the discounting rate obtained in delay discounting tasks.

1.2.3.1. Behavioural measures of reward-delay impulsivity.

Previous studies have assessed the ability to delay reward using validated delay discounting paradigms. The most-widely used tasks to measure this dimension of impulsivity and results from clinical and personality studies using these paradigms are described in this section.

The Two-Choice Impulsivity Paradigm (TCIP; Dougherty et al., 2003) measures tolerance for delayed rewards using a discrete-choice procedure (Dougherty et al., 2005). In this task, participants can click on either a black circle or a black square to add points to a counter. If they click on the circle, the square disappears and the circle changes from black to grey for 5 seconds, after which the circle starts flashing, and participants can click on the flashing circle to earn 5 points. If they click on the square, the circle disappears and the square changes from black to grey for 15 seconds, after which the square starts flashing, and participants can click on the flashing square to earn 15 points. The sum of the chosen rewards can be seen on the counter. The main measure is the proportion of smaller-sooner reward choices. During this task, the discrete-choice procedure determines the total money earned with each choice, either 5 or 15 cents. This results in the participant being able to determine that always choosing the 15 cents option will award them three times the total amount of reward. However, it will also take three times longer; which can make interpretation of results complicated (Dougherty et al., 2003).

The Single Key Impulsivity Paradigm (SKIP; Dougherty et al., 2003) also assesses tolerance for delayed reward, but in this case, using a free operant procedure (Dougherty et al., 2005). The SKIP was designed to overcome some of the limitations found on tasks such as the TCIP, mentioned before. In the SKIP, participants are free to click the mouse button as many times as they desire to receive a reward, which is shown on a counter. In some versions of this task (e.g., Dougherty et al., 2003; Marsh et al., 2002), with each click, 1 cent is paid for every 2 seconds since their last response. For example, if the participant clicked the button 30 seconds after the last response, 15 cents would be paid, so that the longer they wait between consecutive responses, the more points a response earns. Two counters are always displayed on the monitor, one showing the accumulated earnings, the other displaying the reward earned in their last response. The primary measure of this task is the number of total responses.

The Delayed Reward Task (DRT; Cherek, Moeller, Dougherty, & Rhoades, 1997) measures the inability to delay gratification using an adjusting reward schedule. In this task, two letters, A and B, are presented simultaneously, participants are required to choose one of the two. Letter A always represents the impulsive choice and, when selected, the letter B option disappears while letter A begins to flash for 5 seconds. If letter A is clicked while flashing, participants receive a 5 cents reward. When letter B is selected, letter A disappears while letter B flashes for 15 seconds, and if letter B is clicked when flashing, participants receive a 15 cents reward. The length of the letter B delay increases 2 seconds after every trial this letter is selected, and decreases 2 seconds when letter A is selected, with a maximum of 7 seconds decrease. Because the number of trials is fixed, choosing the B option in every trial gives the maximal reward. The proportion of impulsive choices, that is, the number of times letter A is selected, is the primary measure of reward-delay impulsivity on this task.

The Delay Discounting Task (DDT; Kirby, 2009; Kirby, Petry, & Bickel, 1999) measures delay discounting and there are many versions of this task. Most of them change the magnitude of the reward or the delay to the future reward, for example (e.g., Petry & Casarella, 1999; Richards et al., 1999). The original version, is based on the Monetary Choice Questionnaire (MCQ; Kirby, 2009; Kirby et al., 1999) and consists of 27 choices between smaller, immediate and larger, delayed amounts of money such as, “Would you prefer (a) $34 today or (b) $50 in 30 days?”. The magnitude of the delayed choice varies between small and large amounts of delayed money, delayed by 7–186 days. The primary measure of the DDT is the discount rate, which is based on the pattern of choices and can be estimated using different approaches based on hyperbolic or exponential functions (see Kirby et al., 1999, for a detailed description). Other researchers have proposed that the proportion of smaller-sooner reward choices can also be used as the primary measure of delay discounting. This approach allows a more straightforward interpretation, is simpler to calculate and has shown similar results to those obtained using the hyperbolic discounting function (Benningfield et al., 2014).

Figure 1.1. Task design of the delay discounting task used in an fMRI study, taken from Ballard & Knutson, 2009.

The DDT has also been adapted to fMRI studies to investigate the neural activity of temporal discounting in high and low impulsivity individuals (e.g., Ballard & Knutson, 2009; Sripada et al., 2011). Ballard and Knutson (2009), for example, presented in each trial the question in four separate stages (see Figure 1.1): the first part showed the immediate option "Would you rather have $10 in 0 days", the second part showed the magnitude of the future reward "or $15", the third part showed the delay of the future reward "in 30 days", and the fourth part requested the participant to choose either the immediate or the future option "left (today) / right (30 days)". This design allows the specific examination of the neural correlates involved in the processing of the immediate option, the magnitude of the future reward and the delay of the future reward separately, which is not possible with any of the other reward-delay tasks, as these do not allow the specific examination of reward magnitude and delay. Their findings showed that high impulsivity individuals showed greater deactivations in the DLPFC and posterior parietal cortex to future rewards with long delays, than less impulsive individuals (Ballard & Knutson, 2009). During the display of the future magnitude, the MPFC and PCC were also found to negatively correlate with temporal discounting rates, but not during the future reward delay condition (Ballard & Knutson, 2009).

STUDIES	TASK	GROUPS COMPARED	SIGNIFICANT DIFFERENCES IN TASK PERFORMANCE
Swann et al., 2009	SKIP	BD patients vs controls	YES
Strakowski et al., 2009	DRT	BD patients vs controls	YES
Strakowski et al., 2010	DRT	BD patients vs controls	YES
Swann et al., 2011	TCIP	BD with and without anti-social personality disorder (ASPD) vs ASPD only vs BD only vs controls	NO (BD with ASPD greater difficulty in delaying reward)
Ahn et al., 2011	DDT	BD patients vs controls vs schizophrenic patients	YES (BD and SZ greater discounting rate than controls)
Mason et al., 2012	TCIP, fixed delays task	Hypomania-prone group vs controls	YES
Heffner et al., 2012	DRT	BD I patients	YES (Response time, male vs females)
Swann et al., 2013	TCIP	BD patients vs controls	NO
Dawson et al., 2014	DRT	BD I patients	YES (Impulsive choices, male vs female)
Duek et al., 2014	SKIP	BD patients vs controls	NO

Table 1.1. Studies that have investigated reward-delay using laboratory behavioural tasks in patients compared to healthy controls.

Higher rates of delay discounting have been found to be associated with impulsivity (Kirby et al., 1999), sensation seeking and extraversion personality traits, as measured by self-reports (Richards, Zhang, Mitchell, & de Wit, 1999). Higher rates of delay discounting have also been found in ADHD patients (Bitsakou et al., 2009; Paloyelis et al., 2010; Solanto et al., 2001), in compulsive gamblers (Ledgerwood, Alessi, Phoenix, & Petry, 2009; Reynolds et al., 2006), in obese individuals (Epstein et al., 2008; Fields et al., 2011; Weller et al., 2008), in acute alcohol, cocaine and methamphetamine users (e.g., Chambers et al., 2009; Coffey, Gudleski, Saladin, & Brady, 2003; Monterroso et al., 2007; Petry, 2001; Simon, Mendez, & Setlow, 2007) and in

tobacco smokers (Baker, Johnson, & Bickel, 2003), compared to healthy subjects. Furthermore, studies using tasks measuring delay discounting in patients with BSD, SZ or ADHD, have shown that performance in these tasks can differentiate between healthy individuals and patients (see table 1.1).

1.2.3.2. The relationship between behavioural and self-report measures of impulsivity

Researchers have also investigated how laboratory tasks of impulsivity relate to self-report measures of impulsivity. Reynolds et al. (2006) did not find significant associations between four behavioural tasks, the GNGT and SST measuring response inhibition, DDT measuring delay discounting and BART measuring risk taking, and three questionnaires focusing on impulsivity, the BIS-11 (Patton et al., 1995), the I_7 (Eysenck et al., 1985) and the Constraint subscale of the Multidimensional Personality Questionnaire, which included items assessing impulsivity (Patrick et al., 2002). Murphy and MacKillop (2012) investigated the relationship between a delay-discounting task and the UPPS but did not find significant correlations either. Recently, MacKillop and colleagues (2016) investigated the associations between reward-delay tasks, the DDT version of Amlung et al. (2013) and the MCQ by Kirby et al. (1999), with the UPPS and BIS-11 self-reports of impulsivity, but no significant associations were found.

A meta-analysis by Cyders and Coskunpinar (2011) found significant associations between the lack of premeditation and sensation seeking subscales of the UPPS and delay response. The authors (Cyders & Coskunpinar, 2011) assessed delay response as measured in seven studies, which used different reward-delay tasks: delay discounting (Petry & Casarella, 1999; Mitchell, 1999), discounting procedure (Richards et al., 1999), MCQ (Kirby et al., 1999), TCIP (Dougherty et al., 2005), SKIP (Dougherty et al., 2005), and Experiential Discounting Task (Reynolds & Schiffbauer, 2004). Consistent with the significant association found between the UPPS lack of premeditation subscale and reward-delay tasks, it has also been found that individuals scoring low on this subscale were more likely to prefer small and immediate rewards compared to larger and delayed rewards (Lynam & Miller, 2004). Following this meta-analysis, the same authors conducted a correlational analysis which included the TCIP and SKIP as the behavioural measures of delay-reward. They reported the TCIP mean delay latency significantly to be correlated with the Urgency subscale from the UPPS scale, but not with lack of premeditation or sensation seeking, as previously found (Cyders & Coskunpinar, 2012). Surprisingly, the SKIP did not significantly correlate with any of the UPPS subscales (Cyders & Coskunpinar, 2012).

This lack of consistency in results, similar to those reported in rapid-response impulsivity, could be explained by the possibility that behavioural tasks and questionnaires may not measure the same impulsivity components. Therefore, reward-delay tasks may not be capable of assessing particular dimensions of impulsivity as measured by the questionnaires (Stojek, Fischer, Murphy, & MacKillop, 2014). If, as mentioned in the previous section, response inhibition tasks have not been consistently reported to significantly correlate with self-reports of impulsivity, or even, with specific self-report measures of motor impulsivity, such as the BIS-11 Motor or UPPS Urgency subscales, it is easier to understand why reward-delay tasks are not consistently reported to significantly correlate with self-report measures of impulsivity either. However, the disparity in results reported by Cyders and Coskunpinar (2011; 2012) may suggest that the differences within the reward-delay tasks and their design might also play an important role, as the results changed completely depending on which tasks, and which version of them, were included in the analyses. Thus, the selection of reward-delay tasks requires thorough consideration.

Another important limitation found in all these correlational analyses is the lack of comparisons between reward-delay tasks and specific self-report measures of reward-delay impulsivity, as measures such as the BIS/BAS or the SR scales have not been included (e.g., Cyders & Coskunpinar, 2011; 2012; MacKillop et al., 2016; Murphy & MacKillop, 2012). The investigation of the relationship between reward-delay tasks and self-report measures of impulsivity and of reward sensitivity is necessary. For this reason, a correlational analysis and a confirmatory factor analysis were conducted, these included self-report measures of impulsivity and those of reward sensitivity, along with rapid-response and reward-delay behavioural tasks. I suggest this approach will provide a better understanding of the multidimensionality of impulsivity and particularly, of its underlying constructs, as measured by self-reports and behavioural tasks, and the overlap between them.

1.2.4. How can reward-delay and rapid-response impulsivity personality characteristics be classified within the impulsivity personality trait?

One major theoretical issue that has dominated the field for many years concerns the classification of the different personality characteristics associated with impulsive behaviour. For some decades, researchers have debated how to classify impulsivity within the theoretical models of personality. Using factor analysis, it has been suggested that impulsivity, should be included in Psychoticism, and venturesomeness, which contains risk-taking and sensation seeking items, should be included within the Extraversion factor (Zuckerman & Glicksohn, 2016).

To provide a clear example of the difference Impulsivity (Imp) and Venturesomeness (Vent) means in terms of behaviour, Sybil Eysenck used a driving analogy (Eysenck, 1993; p.144): "Our concept of Imp and Vent can best be described by analogy to a driver who steers his car around a blind bend on the wrong side of the road. The driver who scores high on Imp never considers the danger and he might be exposing himself to and is genuinely surprised when an accident occurs. The driver who scores high on Vent on the other hand, considers the position carefully and decides consciously to take the risk, hoping no doubt for the 'thrill' of the sensation seeking arousal caused by what hopes will be merely a 'near miss'." According to this analogy, the driver scoring high on Venturesomeness decides consciously, motivated by the need for the adrenaline rush, while the Impulsive driver is not conscious of his decision, as he is pushed by impulse (Zuckerman & Glicksohn, 2016).

Self-report measures of trait impulsivity used in most studies nowadays, such as the BIS-11 and the UPPS, include subscales specifically related to motor impulsivity, BIS-11 Motor and UPPS Urgency. These subscales measure the Impulsivity construct as defined by Sybil Eysenck in her driving analogy. Whereas Venturesomeness, is generally assessed using subscales measuring risk-taking, non-planning or sensation seeking (Eysenck, Daum, Schugans, & Diehl, 1990; Eysenck, Pearson, Easting, & Allsopp, 1985), such as the- BIS-11 Non-Planning, UPPS (Lack of) Premeditation and UPPS Sensation Seeking subscales.

In recent years, hypothesis-testing, confirmatory factor analysis and Structural Equation Modelling (SEM) methods have facilitated investigation of the latent structure of behavioural and questionnaire measures of impulsivity. A recent study by MacKillop and colleagues (2016), used SEM and found three latent constructs: impulsive choice (DDT/MCQ), impulsive action (GNG, CPT, SST) and impulsive personality traits (BIS-11, UPPS). From these results, it was also concluded that impulsivity should not be used as a singular term. The authors defined impulsive choice as 'discounting of delayed rewards' which is identical to the reward-delay impulsivity construct explained through this chapter. However, according to Eysenck's framework (1985), the second factor is Venturesomeness, which includes sensation or thrill seeking and risk-taking items (Eysenck et al., 1985; 1990). In the study (MacKillop et al., 2016), the authors purposely decided not to include measures assessing reward sensitivity or risk-taking, which means the construct of impulsive choice can only be operationalised by using a variable version of the delay-discounting task (Amlung et al., 2013) and the Monetary Choice Questionnaire (MCQ; Kirby et al., 1999), both measuring delay discounting. It is possible that the second factor might not be represented well enough to show a significant association with impulsive action, as the impulsive choice decision-making process might be influenced by sensation-seeking and/or risk-taking behaviours.

Results from this study showed that, on the one hand, the behavioural tasks comprising impulsive action and the questionnaires comprising impulsive personality traits showed a significant correlation, which is not always found (see subsection 1.2.2.2). On the other hand, the association between impulsive choice and impulsive action was very low and non-significant, which theoretically, should be significantly correlated. I believe an exploratory factor analysis including all the variables tested in MacKillop et al. (2016), in addition to specific measures assessing reward-sensitivity or risk-taking, might clarify the association between impulsive action and impulsive choice and, thus, help in disentangling this old and major, theoretical issue.

1.3. Magnetoencephalography and the neural mechanisms of impulsivity.

1.3.1. Interpreting the MEG signal.

MEG signals can be examined in many ways, but here two of the most relevant approaches are described. First, by analysing the averaged signal based on the timing of an event (Stufflebeam, 2011), which improves the signal-to-noise ratio of the activity of interest during the time-locked event (Vrba & Robinson, 2001). Event-related fields (ERFs) are magnetic fields recorded by MEG, their equivalent of EEG being the event-related potentials (ERPs), these are generated by a motor event or the presentation of a stimulus (Vrba & Robinson, 2001) which can be averaged (generally, around 100 trials) and subsequently, be analysed. In EEG, ERPs are changes in voltage elicited by the presentation of specific stimuli (Friedman et al., 2001). Studies using EEG, use *N* for negative potential and *P* for positive potential to report the changes in voltage, alongside either the component number or latency, such as N2 or P300, that reflect the second negative component or positive component starting after 300 ms from the stimulus onset. In MEG, the influx or efflux of magnetic fields result in differences in topography, these fields have both a source and a sink, which results in polarity not being constant, ERFs on one side of the scalp can be positive and negative on the other side (Castro-Meneses et al., 2016). In studies using MEG, changes are generally reported by using the prefix *M*, for magnetic, followed by the peak's ordinal position, such as M2, or by adding an "m" to the components mentioned in EEG studies, as in N2m, for example. Along the current thesis, terms like "larger", "smaller", "increased" and "decreased" are used to describe the comparison of the amplitudes of peaks of high and low impulsivity groups, that are more positive or more negative between the two groups. The analysis of ERFs allows the examination of any potential difference between high and low impulsivity groups that might explain their distinct task performance, and to be localised in sensor and source level.

Second, analysing the MEG signal in the time-frequency representation (TFR) allows the identification of oscillatory components implicated in cognitive processes (Gross, 2019). Oscillations originate as a result of the total EPSPs of thousands of neurons (Lopes da Silva, 1991) and can be classified in certain frequency bands: delta (1-4 Hz), theta (4–8 Hz), alpha (8–12 Hz) and beta (12–30 Hz); however, the precise band limits differ among researchers and across studies. The presentation of stimuli, as in the behavioural tasks used in this thesis, can elicit both evoked and induced activity. Evoked activity is both time- and phase-locked and can be averaged, as in ERFs, induced activity however, is not phase-locked and can be analysed by calculating the power of the signal as a function of time in the frequency bands of interest (Hari et al., 2018). In Chapters 5 and 6, the oscillatory pattern of neural activity involved in response inhibition and delay discounting were investigated in high and low impulsivity groups, as assessed by well-validated self-reports. This approach allows the examination of any potential difference in TFRs between high and low impulsivity groups that might explain their distinct task performance, and to be localised in sensor and source level using beamformer techniques, see methods section 2.7.3. for details.

1.3.2. Neural mechanisms of impulsivity

The behavioural measures of rapid-response and reward-delay impulsivity previously described, also allow the examination of the neural mechanisms produced during response inhibition and delay discounting processes. Neuroimaging studies using these tasks have benefited from techniques such as functional magnetic resonance imaging (fMRI), electroencephalography (EEG) and magnetoencephalography (MEG) (e.g., Boehler et al., 2009; Falkenstein, Hoormann, & Hohnsbein, 1999; Luijten et al., 2014; Simmonds, Pekar, & Mostofsky 2008). In the following sections, a summary of previous neuroimaging findings on rapid-response and reward-delay impulsivity is presented.

1.3.2.1. Neural Correlates of Rapid-Response Impulsivity.

Response inhibition has been examined by employing the go/no-go task or the stop-signal task separately, or by combining both in the same study. The go/no-go task is a well-validated paradigm which allows for the examination of response inhibition (Simmonds et al., 2008). In the stop-signal task, during a certain amount of trials the GO cue is followed by a STOP cue, which instructs the participant to cancel the initiated response (Sebastian et al., 2012).

Previous studies using the go/no-go task alone during fMRI showed an activation in the anterior portion of the supplementary motor area (pre-SMA), anterior cingulate cortex (ACC), orbitofrontal cortex (OFC), right inferior frontal gyrus (rIFG) and middle frontal gyrus (MFG) (e.g., Braver, Barch, Gray, Molfese, & Snyder, 2001; Luijten et al., 2014; Simmonds et al., 2008). Regarding the neural findings from the stop-signal task, it has been suggested that a

network of cortical and sub-cortical regions, the rIFG, pre-SMA, and the subthalamic nucleus, are critical for inhibiting a response in this task (Aron, Robbins, & Poldrack, 2014). Specifically, several studies using fMRI have also observed greater activation in the right pre-SMA on successful stop trials compared to failed stop trials (e.g., Aron & Poldrack 2006; Boecker et al. 2011; Cai, George, Verbruggen, Chambers, & Aron, 2012).

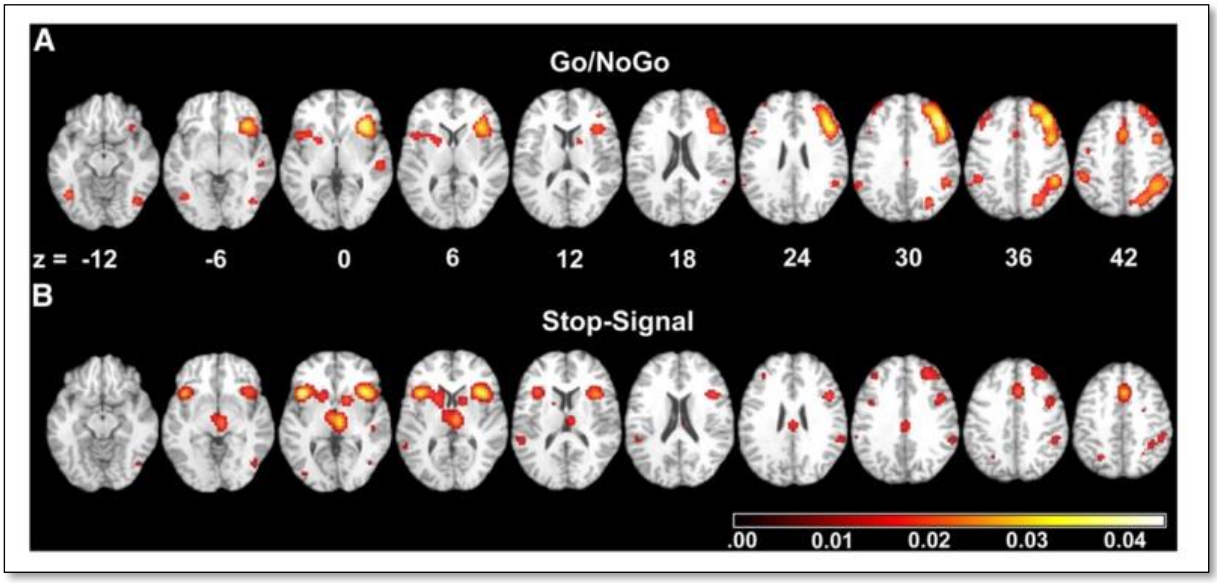

Figure 1.2. Activation likelihood estimation (ALE) map showing significant inhibition-related activation clusters, see Table 1.2. for details of the active areas, taken from the meta-analysis by Swick et al. (2011). The scale bar shows the ALE statistic, which becomes more significant from left (dark red) to right (white).

Different and common activation patterns have also been reported when the neural correlates of the Go/No-Go and Stop-Signal tasks are directly compared in the same participants. Table 1.2 summarises these findings, while Figure 1.2 shows an example of the different activation clusters observed in each task (from Swick et al., 2011). Overall, researchers have emphasised the critical role of the inferior frontal and medial cortical regions that include the pre-SMA and parts of the midcingulate cortex, across the two tasks (Huster et al., 2013; Swick et al., 2011). Furthermore, these studies have suggested the existence of a task-independent inhibition network, which is shared by both tasks and might be comprised of overlapping regions found in many of the previous studies (e.g., Rubia et al., 2001; McNab et al., 2008; Zheng et al., 2008; Sebastian et al., 2013; Dambacher et al., 2014). They also suggested the existence of task-dependent specific brain regions, see Table 1.2 for details on the differences, that may explain the differences when both tasks are combined (Dambacher et al., 2014).

	Common activity	Task-dependent activity	
		Action restraint (GNGT)	**Action cancellation (SST)**
Rubia et al., 2001	Lateral PFC Medial PFC Parietal cortices	Left-lateralized prefrontal and parietal activation	Right hemispheric activation
McNab et al., 2008	Right IFG MFG Left IFG Left insula		
Zheng et al., 2008	Right middle prefrontal cortex Middle occipital cortex		
Swick et al., 2011 (see Fig. 1)	Right anterior insula Pre-SMA	More fronto-parietal network, especially the right MFG and right inferior parietal lobule	More cingulo-opercular network, especially the left anterior insula and bilateral thalamus
Sebastian et al., 2013	Inferior frontal cortex Pre-SMA Parietal regions		
Dambacher et al., 2014	Inferior frontal cortex Pre-SMA Thalamic brain areas ACC	Right superior frontal gyrus, left middle frontal gyrus and bilateral anterior cingulate cortex.	Right middle frontal gyrus, posterior cingulate cortex and parietal regions.

Table 1.2. Differences and common activations of response inhibition reported in fMRI studies combining the Go/No-Go (measures action restraint) and the Stop-Signal (measures action cancellation) tasks.

1.3.2.1.1. Response inhibition: ERPs and EFRs

Both EEG and MEG are far more precise than fMRI when the objective is to temporally measure rapid brain processes, such as those occurring during response inhibition. Investigating the electrophysiological responses evoked during response inhibition paradigms, such as the go/nogo and stop-signal tasks, is considered a suitable approach (Gao et al., 2019), as these are suggested to reflect inhibitory processes (Huster et al., 2013). While the differences on the responses evoked by these tasks between good and bad performers or high and low impulsivity individuals, might indicate a potential inhibitory deficit. Both NOGO and STOP conditions elicit two ERP effects when compared to GO trials, a frontocentral negativity reported between 140 and 300 after stimulus onset, the N2, and the P3, a

frontocentral positive response peak 150 ms observed after the N2 (e.g., Falkenstein et al., 2002; Huster et al., 2013; Johnstone et al., 2007).

NOGO-N2

A stronger negative deflection detected during NOGO and STOP trials than in GO trials, has been reported between 200 and 300 ms after stimulus onset (Nakata et al., 2005; Ramautar et al., 2004; Schmajuk et al., 2006), and is considered an indicator of response inhibition (Gao et al., 2019). In healthy individuals, a larger NOGO-N2 amplitude in good performers compared to those with reduced performance (Falkenstein et al., 1999), serving as a measure of response inhibition efficiency (Schmiedt-Fehr et al., 2011). This modulation has been suggested to consist of two distinct processes, one associated with response inhibition (Bokura et al., 2001) and another one reflecting conflict monitoring and error processing (Dimoska et al., 2006; Greenhouse & Wessel, 2013; Huster et al., 2013; Krämer et al., 2011). The neural source of the NOGO-N2 effect has been reported to be the midcingulate cortex (Huster et al., 2019), the orbitofrontal area and the ACC (Luijten et al., 2014).

EEG studies have investigated the differences in ERPs between groups based on task performance, personality traits or certain behavioural issues; yielding important results on the NOGO-N2 effect. Falkenstein and colleagues (1999) for example, compared good and poor performers based on their number of commission errors and observed larger NOGO-N2 amplitudes in good than bad performers. In 2001, van Boxtel et al. classified individuals based on their SSRT instead but did not find significant differences between groups. In a study by Chen et al. (2005), individuals were divided into two groups based on criminal offenses committed, impulsive-violent offenders and controls, who did not commit impulsive-violent offenses, and found significantly smaller NOGO-N2 amplitudes in impulsive-violent offenders than in controls. Others have not observed significant differences in the NOGO-N2 amplitude between high and low impulsivity individuals, as measured by their individual reaction times, calculated as a function of their general response speed (Ruchsow et al., 2008). This calculation was made based on the method by Pailing et al. (2002), they suggested that this would show whether their errors were due to deficient motor response control or because of their response being influenced by impulsivity.

The disparity of results might be explained by the differences in the assessment of impulsivity, Chen et al. (2005) assessed impulsivity indirectly through criminal offenses committed while Ruchsow et al. (2008) used reaction times as an indicator. Nevertheless, the assessment of impulsivity in the latter study was not extensive nor comprehensive, since the association between behavioural and self-report measures of impulsivity is not clear (see section 1.2.4 for

an extensive explanation; also, Jauregi et al., 2018; Lansbergen et al., 2007; Lijffijt et al., 2004).

Regarding other personality characteristics such as trait anxiety, Sehlmeyer and colleagues (2010) compared healthy undergraduates based on trait anxiety and anxiety sensitivity, group comparisons showed the NOGO-N2 to be significantly amplified in subjects with higher trait anxiety. The larger amplitude of the NOGO-N2 has also been found in individuals with behavioural issues, such as alcohol, tobacco, social networking and smartphone excessive users. Kreusch and colleagues (2014) for example, reported heavy drinkers to display larger NOGO-N2 amplitudes than light drinkers in a modified go/nogo task which used alcohol-related cues. Detandt et al. (2017) used a modified go/nogo task in which the background was either neutral, smoking or non-smoking related, they found the NOGO-N2 latencies to be shorter in smokers compared to non-smokers in smoking-related backgrounds. In a recent study, excessive social networking users showed larger NOGO-N2 amplitudes than non-excessive users in a modified go/nogo task with social networking-related cues (Gao et al., 2019).

Altogether, these findings suggest that the differences reported in individuals with behavioural issues or high trait anxiety, as larger NOGO-N2 amplitude or shorter latencies compared to controls, might reflect inhibitory deficits (e.g., Detandt et al., 2017; Gao et al., 2019; Kreusch et al., 2014; Sehlmeyer et al., 2010). However, the contradictory results reported by Chen et al. (2005) and Falkenstein et al. (1999) open the question of whether this might be incorrect or not.

STOP-N2

A stronger negative deflection in STOP trials (STOP-N2) compared to GO trials has been reported between 200 and 370 ms over frontal areas in studies using ERPs (e.g., Ramautar et al. 2004; 2006; Schmajuk et al., 2006; Upton, Enticott, Croft, Cooper, & Fitzgerald, 2010), but not in a previous MEG study (e.g., Boehler et al., 2009). A larger STOP-N2 amplitude has been reported in successful trials compared to failed inhibition trials (Schmajuk et al., 2006) and to peak earlier during successful trials compared to unsuccessful trials (Ramautar et al., 2004; 2006); thus, the STOP-N2 effect has been suggested to represent increased inhibitory activity (Schmajuk et al., 2006). The source of the STOP-N2, however, is not clear. For instance, Schmajuk and colleagues (2006) reported it over right inferior frontal regions, while others have reported it over medial frontal regions (Ramautar et al., 2004). Note however, that very few of the EEG studies described here have used current-source-density (CSD) transformations (e.g., Kamarajan et al., 2008; 2015a), which enhances the spatial resolution

of EEG (L Kamarajan et al., 2015a; Liebrand, Pein, & Tzvi, 2017) is considered more sensitive for source localisation (Kamarajan et al., 2015a; Nunez, 1989).

To the author's knowledge, previous EEG studies directly comparing high and low impulsivity individuals in the Stop-Signal Task have not reported any results on the STOP-N2. However, in children with ADHD, a diagnosis related to response inhibition (Dimoska et al., 2003), the STOP-N2 amplitude has been found to be reduced compared to that of healthy children, along with a lower reaction time to the stop signals (SSRT; Pliszka et al. 2000; Dimoska et al. 2003).

NOGO-P3

The NOGO-P3 is a positive potential found between 300 to 600 ms after stimulus onset, of which larger amplitudes in NOGO than in GO trials over frontocentral regions have been reported (e.g., Huster et al., 2013; Luijten et al., 2014; Wessel & Aron, 2015). It has been suggested to serve as an index of response inhibition (Huster et al., 2013; 2019; Wessel & Aron, 2015). However, because the effect is observed relatively late after stimulus onset, some researchers have argued it might represent the monitoring of successful motor inhibition (Bruin et al., 2001; Huster et al., 2013; Schmajuk et al., 2006), resulting from not responding to the NOGO stimuli.

Previous EEG studies comparing high and low impulsivity individuals have reported different findings regarding the amplitudes of the NOGO-P3. Some studies have reported the NOGO-P3 amplitude to be reduced in high impulsivity individuals compared to low impulsivity individuals, using the BIS-11 Motor subscale (Benvenuti et al., 2015). Others reported the same while using the individual reaction time scores as explained before (Ruchsow et al., 2008a), and others have not found significant differences when comparing impulsive-violent offenders to controls (Chen et al., 2005). Further supporting the reduced NOGO-P3 amplitude finding in high impulsivity individuals, a negative association between the BIS-10 (Barratt, 1985) and the NOGO-P3 amplitude has previously been reported (Ruchsow et al., 2008b). In a clinical study, a reduced NOGO-P3 amplitude was observed in individuals diagnosed with bipolar disorder (Ruchsow et al., 2008a) compared with controls; interestingly, impulsivity has been reported to characterise bipolar spectrum disorders (Strakowski et al., 2009).

Consistent with these results, studies focusing on young individuals have shown that in those with internet gambling disorder and excessive social networking users, a reduced NOGO-P3 amplitude has been observed compared with controls (Gao et al., 2019; Li et al., 2019). Further evidence for the association between reduced NOGO-P3 amplitude and deficits in inhibitory control comes from research done on addiction studies, which have reported the same finding (on alcohol, Kamarajan et al., 2005; cocaine, Sokhadze, Stewart, Hollifield, & Tasman, 2008; internet addiction, Dong et al., 2010). Moreover, Luijten and colleagues (2016)

conducted logistic regression analyses which suggest that smaller NOGO-P3 amplitudes are associated with increased relapse risk in smokers.

Although the literature seems consistent, two studies have reported larger NOGO-P3 amplitudes in non-clinical populations with impulsive characteristics, smokers and excessive internet users compared to controls (Detandt et al., 2017; Dong et al., 2010), which are hard to reconcile with the remainder of the literature, casting some doubt on a simplistic interpretation of the NOGO-P3 as an indicator of response suppression. Nonetheless, the majority of studies favour the notion that reduced NOGO-P3 amplitudes are associated with impaired response inhibition (Huster et al., 2019).

STOP-P3

A stronger positive deflection found in STOP trials (STOP-P3) compared to GO trials has also been reported in previous studies to occur between 370 and 650 ms after the presentation of the STOP cue (e.g., Ramautar et al. 2004), which is similar to the NOGO-P3 effect found in NOGO trials (e.g., Bokura et al., 2001). This modulation has been reported to highly correlate with SSRTs (Wessel & Aron, 2014), but, because the modulation continues after the response to the stop signal is given, researches have suggested it might not be related to the stopping process but to the evaluation of the process (Kok et al., 2004).

Clinical studies have reported a smaller STOP-P3 amplitude in children and adults with ADHD (Bekker et al., 2005; Dimoska et al., 2003; Overtoom et al., 2002) compared to controls. Non-clinical participants scoring high on self-report measures of impulsivity reported larger STOP-P3 amplitudes than those scoring low, using Eysenck's Impulsiveness/ Venturesomeness/Empathy Questionnaire, Eysenck et al., 1993, (Dimoska & Johnstone, 2007). Lansbergen et al., (2007) also reported high impulsivity individuals, as measured by the I7 Impulsivity questionnaire (Eysenck, Pearson, Easting, & Allsopp, 1985), to show larger STOP-P3 amplitudes than those scoring low. The authors of these two studies suggested the larger amplitude in STOP-P3 reflects the demand for increased inhibitory effort in those scoring high on impulsivity measures (Dimoska & Johnstone, 2007; Lansbergen et al., 2007). However, in a more recent EEG study, Shen et al. (2014) reported the STOP-P3 amplitude to be significantly smaller in individuals scoring high on a Chinese Impulsiveness Scale (Li et al., 2002) than in those scoring low on this measure.

It could be argued that because of the similarities between the Go/No-Go and Stop-Signal tasks as measures of response inhibition, the differences in NOGO-P3 and STOP-P3 amplitudes between individuals scoring high and low on impulsivity questionnaires, would follow a similar pattern too. However, most, but not all previous studies, reported the NOGO-P3 amplitudes to be reduced in high impulsivity individuals compared to low impulsivity

individuals (e.g., Benvenuti et al., 2015; Ruchsow et al., 2008a), while studies on the STOP-P3 have reported both (e.g., Dimoska & Johnstone, 2007; Lansbergen et al., 2007; Shen et al., 2014).

Previous studies on the differences between high and low impulsivity individuals regarding the NOGO-P3 and STOP-P3 modulations have investigated each task separately and on different samples. Based on inconsistencies in the findings to date, the literature would benefit from the examination of the neural correlates of both NOGO and STOP trials in the same task and in the same sample. This approach could help in disentangling what the differences on the P3 amplitude are between high and low impulsivity individuals. In Chapter 4, the NOGO-P3 and STOP-P3 effects were examined further, by simultaneously testing the Go/No-Go and Stop-Signal tasks using MEG in a healthy undergraduate population divided by their level of impulsivity, as assessed by self-report measures.

NOGO-N1

Most of the previous studies examined the N2 and P3 effects, as they represent the main indicators of response inhibition, and thus, these were described first. However, an earlier effect has also been reported, although it does not reflect motor inhibition, as suggested by evidence explained next. During NOGO trials, a stronger negative deflection has been reported (NOGO-N1) compared to GO trials, between 100 and 200 ms after the stimulus onset (De Jong et al., 1990; Filipovic et al., 2000). Researchers have suggested this effect might represent the visual detection of the stimulus (Boehler et al., 2009), the attentional processing of the visual stimulus (Vogel & Luck, 2000), or processing of infrequent events (Kenemans, 2015).

The main results reported during NOGO trials in studies comparing high and low impulsivity individuals have not reported modulations of the N1. Some have reported larger P1 amplitudes in binge drinkers to cues with alcohol-related images than to neutral images (Petit et al., 2012). Similarly, larger N1 amplitudes in excessive social networking sites users to social networking-related cues compared to control images have been reported, suggesting that N1 modulation might reflect the amount of attention towards the salient stimuli (Gao et al., 2019). Since the N1 could reflect visual detection (Boehler et al., 2009) and/or attentional processing of stimuli (Vogel & Luck 2000), investigating potential differences in the NOGO-N1 between low and high impulsivity scorers could yield novel results. It could be argued that less efficient visual detection or attentional processing of the cue in high impulsivity individuals might result in reduced task performance, and therefore reduced amplitude of the N1 could be expected in the HI group compared to the LI group.

STOP-N1

Studies testing the Stop-Signal Task while recording ERPs and/or ERFs have reported a stronger negative deflection between 100 and 160 ms for successful versus failed response inhibition over right and frontocentral regions (STOP-N1; Bekker et al., 2005; de Jong et al., 1990; Dimoska & Johnstone, 2007); which serves as a marker of the discrimination process during attentional processing (e.g., Vogel and Luck 2000; Hopf et al. 2002).

In a MEG study, Boehler et al. (2009) observed that in unsuccessful trials, the STOP-N1 amplitude was larger to the GO cue than in successful trials. Inversely, the negative deflection generated by the STOP cue showed a larger amplitude for successful than for unsuccessful trials, indicating successful inhibition resulted from increased processing of the STOP cue, while increased processing of the GO cue enabled the motor response (Boehler et al., 2009). These findings suggest that heightened processing of the STOP cue is related to success in stopping (Boehler et al., 2009), which is consistent with previous studies (Bekker et al., 2005; Schmajuk et al., 2006).

In an EEG study comparing high and low impulsivity individuals as assessed by the I7 questionnaire (Eysenck et al., 1985), the amplitude of the STOP-N1 modulation was not found to be significantly different between groups (Lansbergen et al., 2007). Another study however, reported a larger STOP-N1 amplitude in high impulsivity individuals compared to those scoring low on Eysenck's Impulsiveness/ Venturesomeness/Empathy (IVE) Questionnaire (1993; Dimoska & Johnstone, 2007). It was suggested that the enhanced STOP-N1 they observed may reflect sensation seeking behaviour in high impulsivity individuals (Dimoska & Johnstone, 2007). Considering that Boehler et al. (2009) reported that enhanced processing of STOP-N1 facilitated successful stopping, it could be argued that the larger STOP-N1 amplitude found in high impulsivity compared to low impulsivity individuals (Dimoska & Johnstone, 2007) might be part of a compensation mechanism in high impulsivity individuals.

Conclusions and open questions

Neuroimaging studies have consistently reported that during the NOGO condition, individuals characterised by impulsivity show larger NOGO-N2 amplitudes compared to GO trials, while the NOGO-P3 amplitude is reduced in the same comparison (e.g., Cheng et al., 2016; Gao et al., 2019; Kreusch et al., 2014; Sehlmeyer et al., 2010). Although some studies have found contradictory results, such as reduced NOGO-N2 amplitude (Chen et al., 2005), no differences in NOGO-N2 (Ruchsow et al., 2008) or larger NOGO-P3 amplitude (e.g., Detandt et al., 2017; Dong et al., 2010), it could be argued that results from the majority of previous studies suggest otherwise. It is hypothesised that high impulsivity individuals, as assessed by both rapid-response and reward-delay impulsivity self-report measures, will show larger NOGO-N2 and

reduced NOGO-P3 amplitude compared to low impulsivity individuals, in line with previous literature.

Results on the NOGO-N1 are not as clear, the lack of studies focusing or reporting differences on this effect in individuals with impulsive characteristics, makes this analysis necessary. NOGO-N1 amplitudes to certain cues compared to neutral stimuli might reflect the amount of attention towards the stimuli (Gao et al., 2019). It could be argued that less efficient visual detection or attentional processing of the cue in high impulsivity individuals might result in reduced task performance and therefore, reduced amplitude of the N1 is expected in the HI group compared to the LI group.

The electrophysiological results on the Stop-Signal task are very contradictory, as both this and the go/nogo task are used to measure motor inhibition, similarities between findings on NOGO and STOP conditions would be expected. To our knowledge, researchers have not found the STOP-N2 equivalent of the larger NOGO-N2 amplitude associated with response inhibition in an adult impulsive population, whereas others have reported reduced STOP-N2 amplitude clinical populations, such as children with ADHD (Dimoska et al. 2003). While studies on the STOP-P3 have reported both smaller and larger amplitudes (e.g., Dimoska & Johnstone, 2007; Lansbergen et al., 2007; Shen et al., 2014), the NOGO-P3 counterpart has been found to be reduced in high impulsivity compared to low impulsivity individuals (e.g., Benvenuti et al., 2015; Ruchsow et al., 2008a).

Differences between high and low impulsivity individuals on the NOGO-P3 and STOP-P3 effects have been investigated using each task separately and on different samples; mostly resulting in contradictory evidence. I believe the literature would benefit from the examination of the neural mechanisms involved in both NOGO and STOP conditions in the same task and in the same sample. This approach allows a fair comparison between the two inhibition paradigms, while disentangling what the differences on the STOP-P3 amplitude are between high and low impulsivity individuals.

In Chapter 5, the spatio-temporal distribution of the specific ERF components generated during the response inhibition paradigm was investigated in high and low impulsivity groups. The NOGO/STOP-N1, NOGO/STOP-N2 and NOGO/STOP-P3 effects were specifically examined further, by simultaneously testing the Go/No-Go and Stop-Signal tasks using MEG in a healthy undergraduate population.

1.3.2.1.2. Response inhibition: time-frequency representations (TFRs)

Theta and delta band activity

Electrophysiological studies have investigated the oscillatory activity generated during the go/nogo and stop-signal tasks by conducting time frequency analyses. These analyses have shown that theta (4-8 Hz) power in NOGO and STOP trials to be increased compared to GO trials, around 200 and 600 ms after stimulus onset over medial-frontal regions (e.g., Harper et al., 2014; Huster et al., 2013; Nakata et al., 2013); overlapping with the NOGO/STOP-N2 and NOGO/STOP-P3 effects observed in ERP/ERF studies (Huster et al., 2013). Specifically, source imaging studies have shown that theta activity might originate from the ACC (Bokura et al., 2001; Cohen, 2011; Luu & Tucker, 2001; Luu et al., 2003; Pandey et al., 2012).

Oscillatory activity in the theta band has been associated with cognitive processes such as alertness (Basar, 1999), focused attention and signal detection (Basar-Eroglu et al., 1992), short-term memory (Klimesch, 1999*)* and reward processing (Cohen et al., 2007; Kamarajan et al., 2008; Marco-Pallares et al., 2008). Frontal-midline theta has been suggested to be an integrative mechanism of early stimulus detection and response selection processes (Cavanagh & Frank, 2014; Mückschel et al., 2017), necessary for cognitive control processes (Cavanagh & Frank, 2014) with a greater demand during response inhibition (Mückschel et al., 2017).

An increase in delta (1-4 Hz) power has been also observed in NOGO trials compared to GO trials (Harper et al., 2014), it has been reported to be crucially involved in response inhibition and to serve as an index of motor inhibition (Kaiser et al., 2019). Event-related oscillations in the delta band have been reported to be associated with attentional, signal detection and decision-making processes (Basar, 1999; Basar-Eroglu et al., 1992).

Previous studies have found that during the Go/No-Go Task, oscillatory activity in delta and theta bands and inhibitory processes are strongly related (e.g., Harper et al., 2014; Lavallee et al., 2014). Time-frequency analyses provide additional information to that provided by ERPs/ERFs (Doñamayor et al., 2012) and have reported delta and theta oscillatory activity to be associated with the response inhibition indexes mentioned above. In NOGO trials, a medial-frontal theta magnitude change has been observed between 200 and 600 ms after stimulus onset (Schmiedt-Fehr et al., 2011). Although delta and theta activity are considered to enable motor inhibition, delta band activity has been found to be a more specific index of response inhibition than theta, as the latter has been observed during both response inhibition and certain motor conflicts with similar task demands (Kaiser et al., 2019). The changes in oscillatory activity in delta and theta bands found in previous studies temporally overlap with NOGO-N2 and NOGO-P3 effects, which might indicate they are part of the same brain function

(Harper et al., 2014; Kaiser et al., 2019). Centro-parietal delta oscillations have been reported to significantly contribute to both NOGO-N2 and NOGO-P3 effects, one study reported delta to contribute significantly more than theta to the NOGO-P3 amplitude (Harper et al., 2014). Theta band oscillations have been reported to be associated with both NOGO-N2 and NOGO-P3 effects (Huster et al., 2013). Even though the literature on spectral analysis of the stop-signal task is very limited, a recent EEG study using scalp-wide current source density (CSD) transformation, which attenuates the influence of problematic volume conductance, found increased midfrontocentral theta during the STOP condition (Lockhart et al., 2019).

Several studies have reported decreased delta and/or theta oscillatory activity specifically during the Go/No-Go task in individuals diagnosed with ADHD (Krämer et al., 2009), in abstinent alcohol-dependent individuals (Colrain et al., 2011; Kamarajan et al., 2004; Pandey et al., 2016) in young adults classified as binge drinkers (Lopez-Caneda et al., 2017) and in young individuals at risk for alcoholism (Kamarajan et al., 2006), compared to healthy controls. In some studies, this reduction was significantly different only during NOGO trials (e.g., Krämer et al., 2009; Kamajaran et al., 2006) and found over frontal regions, which has been suggested to reflect a deficit in fronto-parietal networks recruited during inhibitory processing (Colrain et al., 2011; Kamarajan et al., 2004; Lopez-Caneda et al., 2017). Some of these authors have proposed, that weaker low-frequency oscillatory activity related to inhibitory processing, such as delta and theta, might lead to a predisposition to develop disorders characterised by disinhibition or alcohol-related disorders, and might serve as a vulnerability marker for developing certain disorders (Lopez-Caneda et al., 2017).

Alpha band activity

Oscillatory activity in the alpha band has been described to have an inhibitory role, which suppresses the processing of task-irrelevant stimuli that results in a more efficient task performance (Jensen & Mazaheri, 2010; Klimesch et al., 2007). Others, however, have suggested that alpha oscillations might be involved in the processing of task-relevant stimuli (Mo et al., 2011; von Stein et al., 2000). To reconcile these two hypotheses, it has been suggested that the specific role of alpha depends on different factor, for example. the region from which it is measured, the characteristics of the stimuli being presented (Mo et al., 2011; Walz et al., 2015) or task demands (Jensen & Mazaheri, 2010). Alpha band oscillations are associated with cognitive processes such as attention (Jensen & Mazaheri, 2010; Klimesch et al., 2007), visual perception (Hanslmayr, Gross, Klimesch & Shapiro, 2011) and working memory (Klimesch, 1999).

Increased alpha band (8–12 Hz) power has been associated with response inhibition during NOGO trials during a sensorimotor Go/No-Go Task (Nakata et al., 2013). In addition,

significantly decreased alpha power in young individuals at risk for alcoholism (Kamarajan et al., 2006) and in abstinent alcohol-dependent adults (Pandey et al., 2016) compared to control subjects, has also been reported. Decreased alpha power has been suggested to reflect an early attentional deficit that might affect the inhibition process (Pandey et al., 2016). To our knowledge, no significant result in alpha band activity has been reported during the stop-signal task, but it could be argued that individuals with impulsive characteristics might show a similar decrease in alpha power to those seen in previous studies (Kamarajan et al., 2006; Pandey et al., 2016).

Beta band activity

Activity in the beta band has been suggested to be involved in motor preparation, execution and inhibition processes (Pfurtscheller & Lopes da Silva, 1999). Beta band activity has been reported to show an event-related desynchronization (ERD) during movement preparation and execution, and an event-related synchronization (ERS) after movement (Krämer et al., 2011). Some authors have suggested the ERD in the beta band to reflect activation in the motor cortex (Krämer et al., 2011; Ritter, Moosmann, & Villringer, 2009; Rau, Plewnia, Hummel, & Gerloff, 2003; Leocani, Toro, Zhuang, Gerloff, & Hallett, 2001; Pfurtscheller & Lopes da Silva, 1999). Other studies have found an ERD and an ERS in the beta band only and over motor areas, suggesting ERD to reflect motor activation and ERS motor inhibition (Krämer et al., 2011; Ritter Neuper, Wortz, & Pfurtscheller, 2006; Pfurtscheller & Lopes da Silva, 1999).

In NOGO and STOP trials, beta (12-30 Hz) power has been seen to increase over frontal areas (Alegre et al., 2004) or central areas (Krämer et al., 2011). Previous studies have shown motor activity, such as motor preparation and execution, to be associated with beta oscillatory activity (Krämer et al., 2011; Marco-Pallares et al., 2008; Swann et al., 2009). Marco-Pallares and colleagues (2008) using EEG, for example, reported an increase in beta during successful STOP trials compared to GO trials and unsuccessful STOP trials. Swann et al. (2009) using intracranial recordings, found an increase in beta over right inferior frontal areas in successful STOP trials, but not in unsuccessful STOP trials, while a decrease in beta power compared to the baseline over motor areas was reported to be stronger in unsuccessful STOP trials compared to successfully inhibited trials. Consistently, the same researchers reported increased beta activity in successful stop trials compared to unsuccessful ones (Swann et al., 2012). Overall, these studies have shown changes in beta oscillatory activity in the IFC and preSMA to be related to motor execution and successful inhibition (Huster et al., 2013).

However, in those studies in which potential beta band power between groups has been specifically investigated (e.g., Kamarajan et al., 2006), no significant differences were found

for example, between young individuals at risk for alcoholism and controls. Results in beta band power have more prominently been reported in studies using the stop-signal task, whereas findings in theta and delta band activity predominantly come from experiments using the go/nogo task.

Conclusions and open questions

Previous findings from studies have shown decreased power in delta, theta and/or alpha bands in individuals diagnosed with ADHD, in abstinent alcohol-dependent individuals, in young adults classified as binge drinkers or at risk for alcoholism, compared to controls (e.g., Colrain et al., 2011; Kamarajan et al., 2004; 2006; Krämer et al., 2009; Lopez-Caneda et al., 2017; Pandey et al., 2016). Even though these disorders and behaviours are known to be characterised by impulsivity, to our knowledge, no study has investigated the oscillatory activity in these frequency bands in healthy individuals scoring high and low on impulsivity directly, which has previously been done when examining ERPs (e.g., Benvenuti et al., 2015; Chen et al., 2005; Dimoska & Johnstone, 2007; Lansbergen et al., 2007; Ruchsow et al. 2008; Shen et al., 2014),.

Similarly, no significant differences have been reported in beta band power (e.g., Kamarajan et al., 2006), but researchers have proposed fronto-central beta activity to serve as a potential marker of inhibition in electrophysiological studies (Huster et al., 2013). Therefore, any potential differences in beta band activity between high and low impulsivity individuals might explain what contributes to the reduced task performance seen in impulsive individuals. Results in beta band power have more prominently been reported in studies using the stop-signal task, whereas findings in theta and delta band activity predominantly come from experiments using the go/nogo task. We believe that investigating both the go/nogo and stop-signal tasks simultaneously and in the same sample, will provide novel findings while clarifying the role of each of the frequency bands examined here.

In line with the literature, impulsive individuals, as assessed by high scores in rapid-response and reward-delay impulsivity self-report measures, were expected to show decreased power in delta and theta bands as well as reduced alpha and beta ERD.

1.3.2.2. Neural correlates of reward-delay impulsivity.

Researchers have suggested that an increase in reward-seeking actions and *immature cognitive control* might serve as a marker of risk for addictive behaviours in teenagers (Li et al., 2019). This, alongside previously mentioned findings, see section 1.2 for details, are part

of the rationale for most of the experiments described in this thesis. Altogether, it could be argued that inhibitory deficits and excessive reward-delay sensitivity represent major contributors towards impulsivity, both are necessary to precisely define/conceptualise impulsivity. Compared to studies investigating response inhibition, studies specifically examining reward-sensitivity are very few.

The neural mechanisms associated with reward-delay impulsivity have been examined using reward-delay paradigms. These have been employed in fMRI studies, mainly the card guessing paradigm and the monetary incentive delay task (MID), see section 1.2.3.1 for details. Both tasks are designed to examine reward processing and have provided supporting evidence for the BAS/reward hypersensitivity model (Nusslock et al., 2014). Previous studies have examined left versus right frontal EEG activity, a measure regarded as a neurophysiological indicator of BAS sensitivity and reward-related emotions (Harmon-Jones, Gable, & Peterson, 2010; Nusslock et al., 2012). Increased left frontal EEG activity, which refers to an approaching or engaging predisposition towards a stimulus, was associated with bias to respond to reward cues (Pizzagalli, Sherwood, Henriques, & Davidson, 2005) and with high self-reported BAS sensitivity (Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997; Nusslock et al., 2014). Results from these studies indicate that during reward anticipation, an increase in left lateral orbitofrontal cortex (OFC) activation is found in individuals at risk for mood disorders (Harada et al., 2013). Table 1.3 summarises findings reported during reward-directed paradigms in fMRI when comparing impulsive clinical and non-clinical populations with controls.

ACTIVE REGIONS	TASK	FINDINGS	STUDIES
Medial prefrontal cortex (mPFC)	DDT	mPFC inversely correlates with trait-impulsivity as measured by the BIS-11	Sripada et al., 2011.
Orbitofrontal cortex (OFC)	MID	Unusual increase in left lateral OFC in BD I and II, and in individuals at risk for BD.	Bermpohl et al., 2010 Nusslock et al., 2012 Caseras et al., 2013 Chase et al., 2013 Harada et al., 2013 Nusslock et al., 2014
Ventral striatum (VS)	DDT/ MID	BD and higher BAS scores participants show increased activity during reward anticipation.	Caseras et al., 2013

Table 1.3. Summary of findings reported during reward-directed paradigms in fMRI when comparing impulsive clinical and non-clinical populations.

The delay discounting task (DDT), see section 1.2.3.1 for details, can be easily used with neuroimaging techniques such as fMRI. Temporal discounting, as measured by this task, has

been reported to covary with impulsivity (Hariri et al., 2006). While also offering advantages over the SKIP, TCIP and DRT, see section 1.2.3.1. Activity in the anterior mPFC generated during the DDT, has been found to inversely correlate with trait-impulsivity as measured by the BIS-11 scores (Sripada et al., 2011). Similarly, a study by Luhmann et al. (2008) reported a negative correlation between participants' higher rates of impulsive choices on the DDT and activity in the mPFC. Correlations between the activity in the ventral striatum and trait-impulsivity, as measured by the BIS scores (Forbes et a., 2009), and temporal discounting rates has also been previously reported (Hariri et al., 2006). These findings suggest that the ventral striatum (VS) and medial prefrontal cortex (mPFC) might be directly related to trait impulsivity and reward-delay impulsivity, along with other active areas.

For instance, research using the DDT has shown the posterior cingulate cortex (PCC), mPFC and VS to be implicated during task execution (Ballard & Knutson, 2009; Luhmann et al., 2008; Sripada et al, 2011). Analysing each of the three DDT conditions, immediate reward, future reward magnitude and future reward delay, has produced relevant results. Specifically, these studies have reported the mPFC, PCC and VS to be active during the presentation of immediate rewards (Tanaka et al., 2004; Wittmann et al., 2007) and during the presentation of the magnitude of the future reward (Ballard & Knutson, 2009). Future reward magnitude has also implicated the nucleus accumbens (NAcc; Sripada et al., 2011), while brain activity during future reward delay remains unspecified (see Table 1.4 for a summary of the main fMRI findings using the DDT).

ACTIVE REGIONS	FINDINGS	STUDY
PCC, mPFC, precuneus and medial temporal regions	More active when an immediate reward was present compared to when absent.	Sripada et al., 2011
PCC, mPFC and VS	Sensitive to the subjective value of a reward	Sripada et al., 2011
PCC, mPFC and VS	Active during the presentation of immediate rewards	Tanaka et al., 2004 Wittmann et al., 2007
DLPFC, TPJ and posterior parietal cortex	Activity negatively correlated to the increasing magnitudes of future rewards	Ballard & Knutson, 2009
PCC, mPFC and NAcc	Activity positively associated with the increasing magnitudes of future rewards	Ballard & Knutson, 2009
NAcc	Active during the magnitude of a future reward	Ballard & Knutson, 2009

Table 1.4. Summary of main activations found using the DDT in fMRI. PCC = posterior cingulate cortex; mPFC = medial prefrontal cortex; VS = ventral striatum; NAcc = nucleus accumbens; DLPFC = dorsolateral prefrontal cortex; TPJ = temporal-parietal junction.

1.3.2.2.1. Reward-delay impulsivity – ERP/ERFs.

Studies using EEG or MEG have focused on reward anticipation coding, reward processing or gambling tasks. Electrophysiological studies have reported larger evoked responses over occipital cortex during the presentation of reward cues compared to the presentation of non-reward cues between 100ms and 155ms after stimulus onset (e.g., peaking at 100ms in Apitz & Bunzeck, 2012; peaking at 155ms in Thomas, Vanni-Mercier, & Derher, 2013). The amplitude of these evoked responses during early visual processing has been reported to be related to the value of the reward presented (Doñamayor et al., 2012).

A later effect in electrophysiological studies on reward cues compared to non-reward cues, at approximately 170ms after stimulus onset has also been reported in the fusiform gyrus (Apitz & Bunzeck, 2012) and at 200 ms over the dorsal PCC (Doñamayor et al., 2012). Interestingly, the amplitude of this later response has also been found to be larger in the context of reward compared to non-reward contexts (Apitz & Bunzeck, 2012). ERP studies using monetary gambling tasks to investigate reward processing, have generally found two main effects: a negative effect at around 200-250 ms, usually referred to as feedback-related negativity (FRN) or N2, and a positive effect at around 300-500 ms, usually referred to as feedback-related positivity (FRP) or P3 (Kamarajan et al., 2009; 2015).

During delay discounting tasks, a P2 effect has previously been found (e.g., Gui et al., 2016; He, Huang, Yuan, & Chen, 2012) and is considered to represent the first evaluation of reward magnitude and reward delay (Gui et al., 2016) while others suggested it represents *stimulus evaluation and quick assessment* (Potts et al., 2006). Depending on the delay of the future magnitude, increasing from 2 weeks to 50 years, the amplitudes of P2 and P3 have been found to also change (He et al., 2012). Together with the P2, a frontal N2 effect has been reported to be associated with preference for immediate rewards, some authors consider it to be the key component in temporal dynamics of the interaction between reward and time valuation (Gui et al., 2016; 2018).

Neurophysiological studies have also provided supporting evidence for the BAS/reward hypersensitivity model by examining left versus right frontal electroencephalographic (EEG) activity, a measure regarded as a neurophysiological indicator of BAS sensitivity and reward-related emotions (Harmon-Jones et al., 2010; Nusslock et al., 2012). Increased left frontal EEG activity, which refers to an approaching or engaging predisposition towards a stimulus, has been associated with bias to respond to reward cues (Pizzagalli et al., 2005) and with high self-reported BAS sensitivity (Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997; Nusslock et al., 2014).

As described, previous studies have found N2, P2 and P3 effects during delay discounting tasks (e.g., Gui et al., 2016; He et al., 2012; Potts et al., 2006). This paradigm can be used to examine the sensitivity to the immediate reward, the sensitivity to the delay of future rewards and the sensitivity to the magnitude of the reward. These processes cannot be examined when using other reward-delay tasks such as TCIP, SKIP or DRT, mainly because of their task design. Although other reward-related paradigms, such as the MID or card guessing paradigm described in 1.2.3.1, have been used to examine the reward process, their objective is to examine the neural processes produced by the decision and feedback phases (Nusslock et al., 2014). It could be argued that individuals who prefer immediate rewards know that the consequences of their immediate choices are often negative but cannot avoid choosing the immediate option; that is why the focus here is on intertemporal choice in impulsive individuals. Consequently, in the current thesis the neural correlates involved in temporal discounting were specifically examined, that being the processing of the three conditions separately: immediate reward, the magnitude of the future reward and the delay of the future reward. Because the DDT allows these examinations, it might be a paradigm with great potential to measure reward-delay impulsivity in MEG, a technique with much better temporal resolution than fMRI.

Considering previous findings, differences between high and low impulsivity individuals in temporal discounting are expected. The P2 component for example, has been reported to reflect the first valuation of the reward magnitude and delay (Gui et al., 2016). This suggests the reward evaluation process might occur differently in high impulsivity individuals and be reflected by larger P2 amplitudes compared to low impulsivity individuals. The frontal N2 effect previously found to correlate with immediate rewards (Gui et al., 2016) is also a potential candidate for group differences, as a stronger N2 effect can be expected in high impulsivity individuals, reflecting an increased preference for immediacy.

1.3.2.2.2. Reward-delay impulsivity – Oscillatory activity

Previous studies using MEG and/or EEG have examined the neural oscillations associated with reward processing, reward anticipation, and reward delivery (e.g., Doñamayor et al., 2012; Kamarajan et al., 2015b), providing insight into the oscillatory patterns involved in these processes, while the specific oscillatory pattern produced by delay discounting is still unknown.

As described in 1.3.3.1, research on reward processing using monetary gambling tasks have shown the ERPs to be characterised by two main effects, a negative effect between 200 and 250 ms after stimulus onset, referred to as N2 or feedback-related negativity, and a positive effect between 300 and 500 ms after stimulus onset, referred to as P3 or feedback-related positivity. While some studies have reported theta oscillations to predominantly contribute to

both N2 and P3 effects (e.g., Kamarajan et al., 2008; 2015; Luu et al., 2004; Cohen et al., 2007), others have reported theta to be associated with N2 and delta with P3 (Bernat et al., 2011; 2015; Harper et al., 2014).

The N2 effect has been reported to index an initial response to primary visual stimulus attributes (Bernat et al., 2015) and to exhibit an increase in theta power following the presentation of reward cues compared to the presentation of non-reward cues (Doñamayor et al., 2012). The P3 effect on the other hand, has been found be sensitive to both primary and secondary stimuli attributes, and more specifically, to reward magnitude, where stronger delta was associated with larger P3 amplitudes (Bernat et al., 2015). Regarding reward magnitude, Doñamayor et al. (2012) also reported that a decrease in beta power was more pronounced the more reward magnitude decreased. The authors suggested the decrease in beta power could be related to motor preparation (Doñamayor et al., 2012). Mainly because decreases in beta power (ERD) have previously been associated with motor control (e.g., Neuper et al., 2006; Zhang et al., 2008). Moreover, the decrease in beta was found near sensorimotor areas and it was related to between-subject variability in reaction times,

As described before, researchers agree that ADHD is associated with trait-impulsivity (e.g., BIS-11 scores, Malloy-Diniz et al., 2007), exhibiting impaired response inhibition (e.g., longer stop-signal traction time, Lijffijt et al., 2005) and higher delay discounting (e.g., Bitsakou et al., 2009; Paloyelis et al., 2010). Past studies have shown an increase in theta and a decrease in beta band activity in individuals diagnosed with ADHD compared to controls during intertemporal choice (Gui et al., 2018; Loo et al., 2013; Monastra, Lubar, & Linden, 2001). The authors suggest that increased impulsivity and increased theta band activity are related, whereas inhibitory control is associated with stronger beta band activity (Gui et al., 2018).

In a study by Kamarajan and colleagues (2015b), adolescents and young adults at high risk of alcoholism were compared with aged-matched participants at low risk during a monetary gambling task. The authors report that individuals at high risk showed lower theta power than subjects at low risk during reward processing, specifically between 200 and 500 ms after stimulus onset, which includes both N2 and P3 effects. Interestingly, individuals at high risk also showed increased impulsivity, as measured by the BIS-11, compared to individuals at low risk. The authors suggested that, because lower theta power was found in both gain and loss conditions of the monetary gambling task, it could be possible that individuals at high risk might struggle to evaluate the consequences of their choices more generally (Kamarajan et al., 2015b). I believe activity in the theta band needs further investigation to clarify these contradictory findings.

Conclusions and open questions

I suggest that investigating reward-delay impulsivity using the delay discounting task (DDT) allows the specific examination of the oscillatory pattern generated by the presence of an immediate reward, the magnitude of a future reward and the delay to receive that future reward; and is therefore the optimal choice for examining reward-delay sensitivity. During the execution of this task, contradictory results have been reported. While some studies reported stronger theta band activity to be associated with impulsivity (Gui et al., 2018), others reported a decrease in theta power in individuals scoring high on impulsivity compared to those scoring low, as measured by the BIS-11 (Kamarajan et al., 2015b). Further examination of potential group differences is necessary to clarify the implications of theta band activity.

As a result of stronger delta being associated to larger reward magnitudes (Bernat et al., 2015) and a decrease in beta power being associated with reward magnitude (Doñamayor et al., 2012), it could be argued that activity in these bands might directly reflect sensitivity to reward magnitude. Potential differences in power between high and low impulsivity individuals might explain what contributes to impulsive temporal discounting. More evidence is required while using an advantageous method such as MEG. In Chapter 6, the time-frequency analyses performed in sensor and source space during the delay discounting task, in a young population divided by their level of self-report impulsivity, are explained.

1.4. Overview of this thesis.

This thesis seeks to investigate impulsivity as comprehensively as possible, by clarifying the classification of the different personality characteristics comprising the impulsive behaviour and its dimensionality. This was accomplished by comparing rapid-response and reward-delay task measures of impulsivity in low- and high-impulsivity groups, as measured by self-reports, to examine whether these measures are sensitive enough to pick up the differences in healthy individuals. The real association between behavioural paradigms and self-reports was investigated to provide a better assessment of impulsivity as a multifaceted construct. Ultimately, by examining the neural circuitry and the oscillatory brain activity of rapid-response and reward-delay impulsivity separately, in individuals scoring high and low on impulsivity questionnaires, I intend to clarify the role of these neural mechanisms and to potentially identify vulnerability markers in individuals scoring high on impulsivity.

In order to do so, three different approaches will be used:

(1) a behavioural study in which both self-report measures and behavioural paradigms are used to compare rapid-response and reward-delay impulsivity in low and high scoring

individuals, as measured by questionnaires, where the associations between behavioural tasks and questionnaires are also examined,

(2) an exploratory factor analysis and structural equation modelling approach used to clarify the structure and classification of the impulsivity domains,

(3) a neuroimaging study involving magnetoencephalography, to clarify the role of the neural mechanisms involved in task-based response inhibition and temporal discounting, and to identify vulnerability markers in individuals scoring high on impulsivity, both in sensor- and source-space.

1.4.1. Chapter 3: Behavioural study - Analysis 1:

In this experiment, the relationship between rapid-response impulsivity and reward-delay impulsivity, and their respective measures, will be investigated in an undergraduate student population. Participants were classified into low-impulsivity vs. high-impulsivity groups based on questionnaires measuring: (a) level of rapid-response impulsivity, (b) level of reward-delay impulsivity and (c) level of both rapid-response and reward-delay impulsivity.

To examine the mechanisms underlying the two dimensions of impulsivity, the aim is to compare rapid-response and reward-delay task measures of impulsivity in low-scoring and high-scoring individuals, as measured by self-reports. I believe this will clarify whether the behavioural measures are sensitive enough to pick up any differences between the two groups. Secondly, associations between behavioural measures of rapid-response and reward-delay impulsivity will be examined to further elucidate the different and contradictory results reported to date.

Based on the results of previous studies, it is hypothesised that:

- Low-scoring, high-scoring and combined groups will perform significantly different on behavioural tasks assessing impulsivity, specifically, high-scoring individuals will show a significantly reduced performance on response inhibition tasks, the GNGT and SST, along with an inability to delay reward on the DDT.
- Performance on the behavioural tasks assessing each of the two dimensions of impulsivity will correlate with the corresponding questionnaires measuring each dimension.

1.4.2. Chapter 4: Behavioural study - Analysis 2: Exploratory Factor Analysis and Structure Equation Modelling.

The classification of the different personality characteristics associated with impulsive behaviour has been the object of study for many decades now. The most recent study on this

regard (MacKillop et al., 2016), showed that a three-factor model provided the best fit to the data: impulsive action, impulsive choice and impulsive personality traits. However, sensation-seeking and reward-delay measures were not included in this model. Here, a new approach is proposed in which the Venturesomeness related scales, such as non-planning and sensation-seeking (Zuckerman & Glicksohn, 2016), along with the EPQ designed by Eysenck (1985), where Psychoticism and Extraversion subscales measure Impulsivity and Venturesomeness respectively, and measures of reward-delay sensitivity, such as the BAS subscales of the BIS/BAS scale, will also be included in a hypothesis-testing confirmatory factor analysis.

The measures included in this experiment are: (1) behavioural tasks measuring rapid-response impulsivity, the go/no-go and stop-signal tasks, (2) BIS-11 questionnaire measuring motor, attention and non-planning impulsivity, (3) UPPS Impulsive scale measuring motor impulsivity, lack of perseverance and premeditation, and sensation-seeking, (4) EPQ measuring Psychoticism, Extraversion and Neuroticism, (5) the delay discounting task measuring reward-delay impulsivity and, (6) the BAS subscales of the BIS/BAS, measuring reward sensitivity, fun seeking and drive to accomplish objectives.

The aim of this experimental chapter is to test whether the three dimensions of impulsivity described in this introductory chapter, i.e., rapid-response impulsivity, reward-delay impulsivity and impulsive personality traits, would benefit from including certain psychometric measures and still be empirically differentiated into three latent domains as in previous studies (e.g., MacKillop et al., 2016).

- It is hypothesised that a three-factor model of impulsivity would benefit from including measures of reward-delay impulsivity, and yet, be differentiated into three latent constructs.

1.4.3. Chapters 5 and 6: MEG Data, Sensor- and Source-Space.

There is limited data on the neural mechanisms that underlie impulsivity, and particularly, how the behavioural measures of RI and reward sensitivity, in which high impulsivity individuals have been found to show an impaired performance, are related to those neural mechanisms. By investigating the neural mechanisms involved in these two processes in individuals scoring high and low on impulsivity questionnaires, this study aims to clarify the role of these neural mechanisms and to potentially identify vulnerability markers in individuals scoring high on impulsivity. Multi-modal experimental methods will be used, including: questionnaires and self-reports to measure impulsivity as a trait, which will help us to identify high impulsivity individuals, specific behavioural tasks to measure performance in RI and how sensitive participants are to immediate rewards; and neuroimaging techniques, such as MEG.

An integrative approach is proposed to increase our understanding of the neural circuitry that is involved in response inhibition (experiment 3) and reward-delay sensitivity (experiment 4). This will allow us to increase our understanding of the spatio-temporal and oscillatory pattern of neural activity involved in response inhibition and, specifically, during both action restraint (NOGO condition) and action cancellation (STOP condition), while examining possible group differences. Using the delay discounting task, potential temporal differences between high and low impulsivity individuals will also be investigated. Our objective is also to extend findings from previous studies described here, by further clarifying whether risk for psychopathology can be determined based on this data.

Regarding ERFs during response inhibition, using the combined go/nogo/stop-signal task, it is hypothesised that:

- High impulsivity individuals will show reduced NOGO-M1 and STOP-M1 amplitudes in visual areas compared to low impulsivity individuals.
- High impulsivity individuals will show reduced NOGO-M2 and NOGO-M3 amplitudes compared to low impulsivity individuals.
- High impulsivity individuals will show larger STOP-M2 and reduced STOP-M3 amplitude compared to low impulsivity individuals.

Regarding oscillatory activity during response inhibition, it is hypothesised:

- Decreased power in delta, theta, alpha and beta bands in impulsive individuals compared to low impulsivity individuals.

Regarding event-related fields during the delay discounting task, it is hypothesised:

- Reduced M1 amplitudes in high impulsivity compared to low impulsivity individuals.
- In the Immediate condition, high impulsivity individuals will show larger M2 amplitudes in frontal sensors than low impulsivity individuals
- In the future Magnitude and Delay conditions, high impulsivity individuals will show larger M2 amplitudes compared to low impulsivity individuals.
- High impulsivity individuals will show reduced M3 amplitudes than low impulsivity individuals.

Regarding oscillatory activity during the delay discounting task, it is hypothesised:

- High impulsivity individuals will show more delta band power than low impulsivity individuals.
- High impulsivity individuals will show more theta band power compared to low impulsivity individuals.

- High impulsivity individuals will show more alpha suppression than low impulsivity individuals.
- High impulsivity individuals will show less beta band power compared to low impulsivity individuals.

1.4.4. General discussion

In the last chapter of this thesis, the research questions proposed at the beginning, the experiments conducted to clarify these questions and the results found here, will be discussed in detail. The implications of the current findings are also described.

CHAPTER 2: General methods

2.1. Chapter summary.

This chapter provides a description of the methods employed in Chapters 3, 4, 5 and 6. The self-report and behavioural measures of impulsivity employed, along with detailed descriptions of the sample, procedures and statistical analyses are included here. As magnetoencephalography (MEG) was also used in chapters 5 and 6, to examine potential differences on the spatio-temporal dynamics of impulsivity between high and low impulsivity groups, a specific MEG section is also included. An overview of the studies described in this thesis is presented in Table 2.1, alongside details of the specific sample, data analysis and measures of impulsivity used in each study.

Chapter number	Data analysis	Dataset	Sample	Age and gender	Behavioural tasks	Self-reports
Chapter 3 Behavioural study: Analysis 1	Spearman's r MANCOVA	A	167	143 females, Mage = 19.43, SDage = 1.72	Go/NoGo Task Stop-Signal Task Delay Discounting Task	BIS-11 BIS/BAS UPPS EPQ
Chapter 4 Behavioural study: Analysis 2	Exploratory factor analysis SEM	A	167	143 females, Mage = 19.43, SDage = 1.72	Go/NoGo Task Stop-Signal Task Delay Discounting Task	BIS-11 BIS/BAS UPPS EPQ
Chapter 5 MEG study: Experiment 1	ERFs TFRs Sensor and source space	B	34	28 females, M*age*=18.94, SD*age*=3.9	Combined Go/NoGo/Stop-Signal task	BIS-11 BIS/BAS
Chapter 6 MEG study: Experiment 2	ERFs TFRs Sensor and source space	B	34	28 females, M*age*=18.94, SD*age*=3.9	Delay Discounting Task	BIS-11 BIS/BAS

Table 2.1. Overview of the studies presented in this thesis, data analysis, sample details, behavioural and self-report measures of impulsivity tested. BIS-11, Barratt Impulsiveness Scale; BIS/BAS, Behavioural Inhibition System Behavioral Activation System scales; UPPS behaviour scale, Urgency, Premeditation, Perseverance, Sensation; EPQ, Eysenck Personality Questionnaire.

2.2. Ethical Considerations.

Experimental procedures were in accordance with the Declaration of Helsinki and approved by the Aston University Ethics Committee. For the behavioural study (REC # 452), chapters 3 and 4, and neuroimaging study (REC # 923), chapters 5 and 6, healthy young adults were

recruited. All participants (aged 18 to 26) gave their written informed consent before taking part in any of the study procedures. Participants were explained their rights to withdraw from the study at any time and reassured that this decision would not negatively affect them in any way. All documents collected during the studies were kept in a locked cabinet inside a coded room at Aston University. The data collected using university computers was kept safely, coded and moved to two computers secured with password, only one document was kept as reference of each participant's names and subject number, all the other files used numbers only.

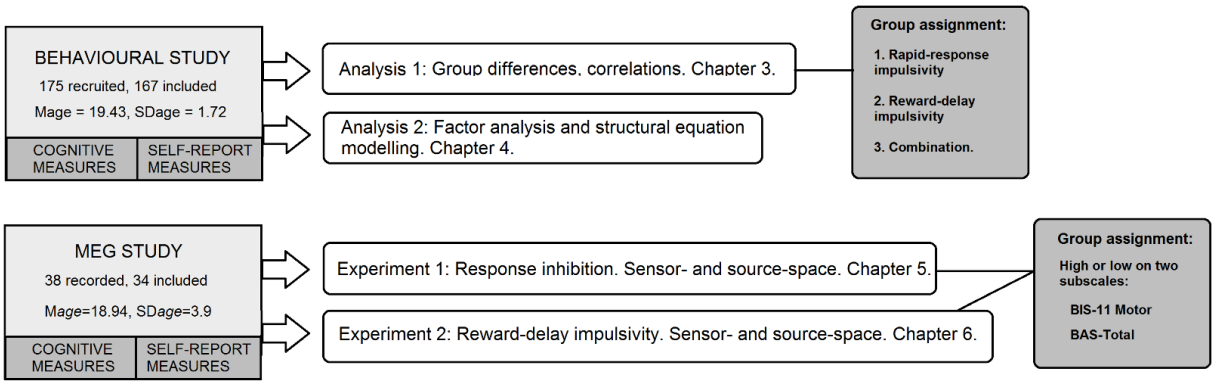

Figure 2.1. Overview of the experiments presented in this thesis, data analysis, sample details, behavioural and self-report measures of impulsivity tested.

2.3. Sample

2.3.1. Behavioural study

Healthy young adults with normal or corrected-to-normal vision were recruited from the student population at Aston University. The studies were advertised using posters and the 'Research Participation Scheme' (SONA) website for psychology undergraduate students. 175 undergraduate students who were aged 18 to 26 and enrolled in psychology or business courses at Aston University participated, see Figure 2.1 and Table 2.1 for details. Participants received either course credit or £7.50 for their time during the behavioural study (experiment 1). Eight participants were excluded from the analysis due to incomplete data, resulting in a total of 167 participants (143 females, Mage = 19.43, SDage = 1.72). The demographic and health questionnaire completed during the study showed our sample was ethnically diverse, consisting of White (40%), Asian (37%), Black (13%), Chinese (5%), and Mixed (7%) heritage. Data collected during the behavioural study was analysed using two different approaches. First, referred to as Analysis 1 (Chapter 3) in this thesis, statistical analyses were conducted to test the differences on impulsivity measures between high and low impulsivity groups and to examine correlations between measures. Second, here referred to as Analysis 2 (Chapter 4), an exploratory factor analysis and structural equation modelling was applied to all the variables tested in the behavioural study.

2.3.2. MEG study

Young adults were recruited from the behavioural study and from further BIS-11 and BIS/BAS only testing. Those who were offered to take part in the MEG study, had scored either high or low on a specific subscale of each questionnaire, see section 2.4.4 for details on how they were assessed. Thirty-eight participants agreed to participate, four participants were excluded from MEG analysis, as a result of low task performance or noisy data. The thirty-four participants who were finally included (28 females, M*age*=18.94, SD*age*=3.9, see Figure 2.1), were ethnically diverse, consisting of White (53%), Asian (41%) and Black (6%) heritage. They received either course credit or £10 for each of the two sessions necessary to complete the MEG study.

2.4. Procedure

2.4.1. Behavioural study

During each session, which lasted approximately 60 min, each participant completed the cognitive tasks measuring rapid-response impulsivity and reward-delay sensitivity in a randomised order. All tasks were administered using EPrime 2.0 Professional (Psychology Software Tools). Demographic and self-report questionnaires were completed in randomised order via Bristol Online Surveys, using the same computer. A researcher was present during all data collection. Cognitive tasks and self-report measures were presented in counter-balanced order.

2.4.2. MEG study: MEG and MRI sessions

2.4.2.1. The MEG session

This session was approximately 90 minutes long. Before participants accepted to take part in the MEG study, they were sent detailed documentation on the MEG and MRI sessions, to ensure they knew what it would be like. During arrival, the procedure was further explained and the possibility of asking any question was given. Consent and screening forms were then signed. Then all metals, such as jewellery, glasses, zips and make up were removed, as these would interfere with the MEG scanner. Next, five small metal discs were attached to the participant's head, so that the head position can be tracked in the scanner. A digital pen, the Polhemus Fastrak*,* was passed over the scalp to generate a digitised version of the participant's head. The behavioural tasks were then explained using a computer which showed what they would see during the experiment. Throughout the procedure, participants were given the opportunity to ask questions. Participants were taken to the scan room, seated comfortably and reassured. A response pad with coloured buttons was provided to allow participants to make a response. During the recording, the participants could communicate

with the researcher, if needed, using two-way intercom system. After approximately 55-60 minutes, the MEG recording was completed.

2.4.2.2. The MRI session

On a different day, the same participants were booked for a 30 minutes long magnetic resonance imaging session where the scanner produces anatomical images of the brain. Using a Siemens MAGNETOM Trio 3T scanner, a structural T1 scan was obtained, this was later used for co-registration with MEG data. Participants were sent detailed information about the imaging session beforehand. They were asked to complete a questionnaire which determines if it is safe for them to be scanned in an MRI scanner. Upon arrival, a researcher explained the procedure and asked the participants to complete a detailed questionnaire to ensure nothing containing metal entered the MRI room. Height and weight of participants were collected to calculate the specific absorption rate to ensure safe heating levels. Another researcher reviewed their details to ensure it was safe for them to go into the scanner. Participants were given earplugs and then placed on a small bed, moved until the head was inside the MRI scanner. Although the scanner produced loud noises, they were able to communicate through the intercom. Participants were told they could withdraw from the study if they felt uneasy anytime.

2.4.3. Group assignment for the behavioural study (Analysis 1)

To analyse the behavioural data (Analysis 1) and to establish which participants were to take part in the MEG study, participants were assigned to either a low- or a high- impulsivity group. Groupings were based on (1) rapid response impulsivity; (2) reward delay impulsivity; and (3) a combination of rapid response impulsivity and reward delay impulsivity.

For (1) Their BIS-11 Motor sub-scale scores: To assess rapid-response impulsivity, high- and low-impulsive individuals were selected from the sample using the highest 35th percentile and the lowest 35th percentile, respectively. This procedure is in line with that of Wilbertz et al. (2014) and resulted in two extreme groups, i.e., a high rapid-response impulsivity group and a low rapid-response impulsivity group.

For (2) Their BAS Total scores: To assess reward-delay impulsivity, high-impulsive individuals were selected from the sample using the highest 15th percentile on the BAS Total scale. This resulted in a high reward-delay impulsivity group. For the low reward-delay impulsivity group moderate BAS scores (between the 40th and 60th percentiles) were chosen. The reason for choosing the moderate BAS score and not the low BAS score is because low BAS scores have previously been linked with unipolar depression (Fowles, 1988; Depue & Iacono, 1989; Kasch et al., 2002) as well as excessively decreased goal-directed activity, loss of interest and

anhedonia (Alloy et al., 2012). Additionally, a moderate BAS score is closer to the mean on the BAS sensitivity dimension thus representing a more normalised statistical perspective. This procedure in line with that of Alloy et al. (2012) and resulted in a low reward-delay impulsivity group.

For (3) A combination of rapid-response impulsivity and reward-delay impulsivity: Here, participants whose scores fulfilled both of the above criteria were included. The high-impulsivity group consisted of participants scoring within the highest 35th percentile of the BIS-11 Motor sub-scale and within the highest 15th percentile on the BAS-Total scale. The low-impulsivity group included participants scoring below 35% on the BIS-11 Motor sub-scale and between the 40th and 60th percentiles on the BAS-Total scale

2.4.4. Group assignment for the MEG study (experiments 1 and 2)

As a result of the findings observed in the behavioural study, the criteria to assign the corresponding participants to either a low- or a high-impulsivity group was also based on the combination of rapid-response impulsivity and reward-delay impulsivity scores, grouping number 3 in 2.4.3. The high impulsivity group (high impulsivity [HI] group; n = 17) consisted of participants scoring within the highest 35th percentile of the BIS-11 motor subscale and above 85% on the BAS-Total scale. The low impulsivity group (low impulsivity [LI] group; n = 17) included participants scoring below 35% on the motor scale and moderately on the BAS-Total scale, between 40th and 60th percentiles (see Alloy et al., 2012). Because low BAS has been linked with unipolar depression (Depue & Iacono, 1989; Fowles, 1988; Kasch et al., 2002), the moderate BAS group was chosen instead of the low BAS group.

2.5. Behavioural measures.

The four tasks described here were programmed and administered using E-Prime 2.0 Professional (Psychology Software Tools; http://www.pstnet.com/), see Table 2.1 for details on which behavioural measures were used in each experiment.

2.5.1. Rapid-Response Impulsivity: Go/No-Go Task

In the behavioural study, a modified version of the go/no-go task described by Rubia et al. (2001) was used. This task presented 75% go trials instead of 70% go trials as others had done. This decision was based on previous studies (e.g., Zheng et al., 2008), which argued that using a simpler go/no-go task, i.e., one with a lower percentage of no-go trials, would reduce load of other cognitive functions, e.g., working memory processes, apart from action restraint.

Participants completed a practice block of 20 trials and two 5-min blocks with pseudo-randomised trials, having a rest between both. The total number of trials in each block is dependent upon the speed with which participants respond. In the first block total number of trials ranged between 143 and 155; in the second block between 114 and 128. To have the same number of completed trials for all participants, the lowest number of trials for each block was used to calculate variables of interest (commission errors, reaction time). Trials were selected based on the order of presentation, using the first 143 of the first block and the first 114 of the second block, discarding the following trials.

Reaction times (RTs) on the GNGT (GNGT_RT) were calculated for correct responses to go trials only. The proportion of commission errors in the GNGT was calculated as the total number of no-go trials in which a response was given, divided by the total number of no-go trials (NOGO-Errors). See Figure 2.2 for a description of the timing parameters.

2.5.2. Rapid-Response Impulsivity: Stop-Signal Task

In the behavioural study, the Stop-Signal task was also tested. During the task, a go stimulus is occasionally followed by a stop signal (delayed by 250ms), in a pseudo-randomised order and at an occurrence rate of 25% of the total trials; see Figure 2.2 for a description of the task and timing parameters.

Participants completed a practice block of 20 trials and two 5-min blocks with pseudo-randomised trials, having a rest between both. The total number of trials in each block is dependent upon the speed with which participants respond. In the first block total number of trials ranged between 102 and 127; in the second block between 103 and 126. To have the same number of trials for all participants, the lowest number of trials completed across all participants for each block was used to calculate the variables of interest (stop-signal reaction time, commission errors on stop trials). Trials were selected based on the order of presentation, using the first 102 of the first block and the first 103 of the second block, discarding the following trials.

The variables of interest were the Stop-Signal Task reaction times (SSRTs) on go trials and proportions of commission errors on stop trials. This proportion was calculated from the total number of stop trials in which a response was given, divided by the total number of stop trials (SST-Errors) and the SSRT. The SSRTs were estimated by subtracting the stop-signal delay, 250ms, from the mean GO trial reaction time (as in Logan et al., 1997). See Figure 2.2 for a description of the timing parameters.

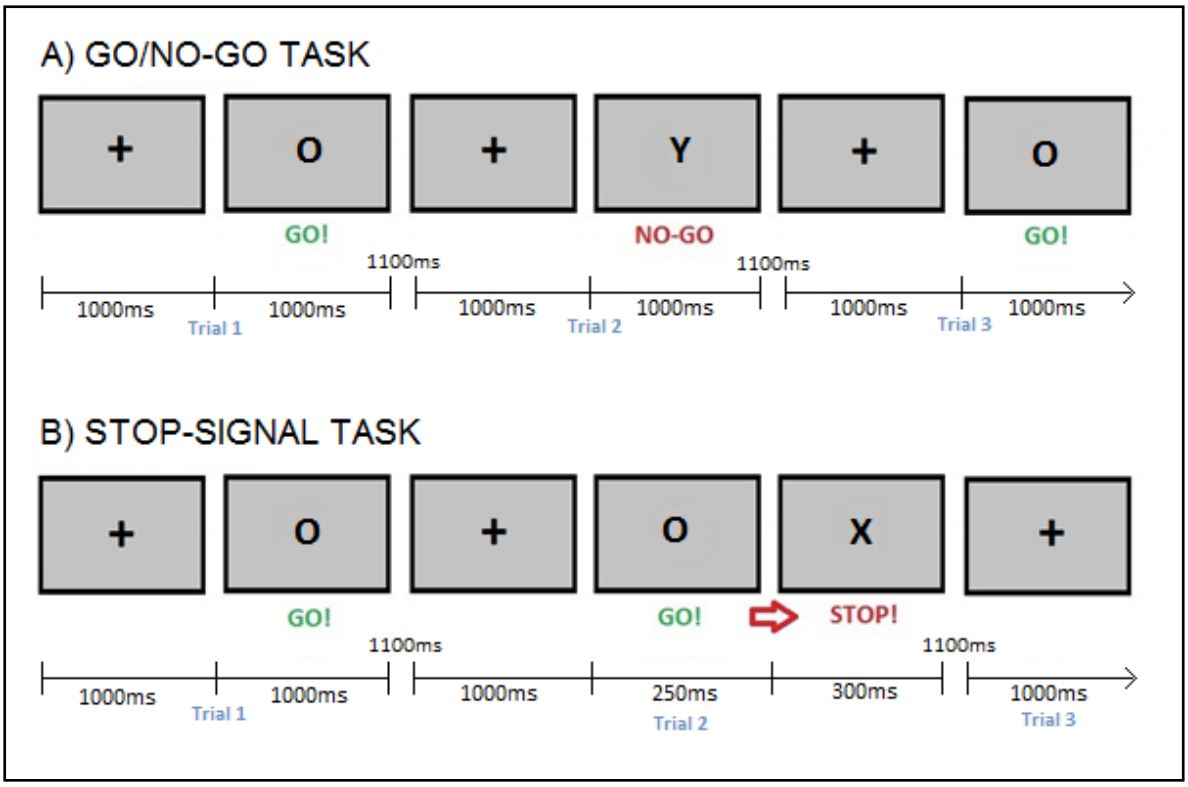

Figure 2.2. Example of trials: (a) the go/no-go task, where participants provided a response as fast as possible to a "go" (letter O) stimulus by pressing a button on a keyboard but refrained from reacting to a "no-go" (letter Y) stimulus; and (b) the stop-signal task, in which participants were requested to withhold their response when the go cue (letter O) was followed by a stop-signal (letter X). In both tasks the fixation cross is presented for 1000ms and the go cues for a maximum of 1000ms or disappears when a response is given, being the inter-stimulus interval (ISI) 1100ms as in Rubia et al. (2001) on the SST. The stop-signal delay (SSD) was set at 250ms after the presentation of the go cue and the stop cue lasted for 300ms, as in Dambacher et al. (2014).

2.5.3. Rapid-Response Impulsivity: Combined Go/NoGo/Stop-Signal Task

Certain limitations were observed as a result of the response inhibition tasks design during the behavioural study, such as the fixed stop-signal delay, see section 3.5 for details. To overcome these limitations, the chosen paradigm for the MEG study had to have successfully been implemented in previous MEG studies. A combined Go/No-Go/Stop-Signal Task (GNGSST) was adapted from Boehler et al., 2009, see Figure 2.3 for task description and timing parameters. This visual response inhibition task allowed the investigation of both NOGO and STOP conditions simultaneously, in the same task, while also maintaining the recommended GO probability. It was programmed and administered using EPrime 2.0 Professional (Psychology Software Tools; http://www.pstnet.com/), and visualised on a projection screen located 86 cm from participants in the MEG room, see details on MEG in section 1.3.

Each trial started with a variable fixation period between 1300 and 1500 ms randomised across trials. This was followed by the presentation of one of two possible traffic light symbols: a “GO sign” represented as a green walking symbol that required a button press with the right index finger, and a “NOGO sign” as reflected by a green stopping symbol, which required withholding a response. Each trial ended when a response was given or when no response was given within 800 ms since the target was presented. In 20% of the trials, the GO-cue was followed by a STOP-cue, represented by a red stopping symbol instructing the participant to cancel the initiated response. The stop signal appeared after a stimulus onset asynchrony (SOA) set by a staircase-procedure, in which the SOA was increased by 1 stimulation-frame (17 ms) after a successful stop trial and reduced by 1 frame after an unsuccessful stop trial. The initial SOA was always 200 ms and the total duration of the trial always 800 ms. In other trials, the GO-cue was followed by a NOGO-cue, which instructed the participant not to respond, the NOGO-cue was presented after a 200 ms SOA, while the total duration of the trial was always 800 ms. The reason for including this condition was that because the SOA was the same as in the STOP condition, it would make the visual stimulation in both conditions comparable, while presenting the NOGO-cue first, instead of the GO-cue, would reduce any motor-related processes (Boehler et al., 2009).

Based on the GNGSST parameters used in Boehler et al. (2009), the GO-cue was presented in a pseudo-randomised order and at an occurrence rate of 60% of the total trials, the NOGO-cue accounted for 10% of total trials, the STOP-cue for 20% and the CONTROL condition for 10% of total trials. Three blocks were presented, and each block consisted of 200 trials, 600 trials being presented in total, 360 GO-trials, 120 STOP-trials, 60 NOGO-trials and 60 Control-trials.

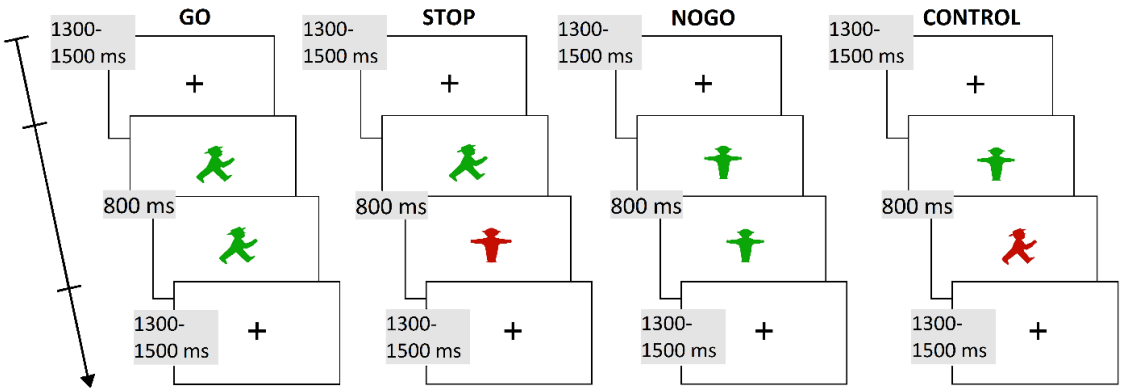

Figure 2.3. Trial types in the Go/NoGo-Stop-Signal task. In GO trials the green “GO cue” was presented for 800 ms. In STOP trials the green “GO cue” was substituted by a red “STOP cue” after an SOA set by a staircase-procedure, the total stimulus duration was 800 ms. In NOGO trials a green “NOGO cue” was presented for 800 ms. Control-trials started identically to NOGO trials, but after SOAs corresponding to those of the STOP trials the green “NOGO cue”

was replaced by a red stimulus “GO cue”, mimicking the visual stimulation timeframe of a STOP trial.

2.5.4. Reward-Delay Impulsivity: Delay Discounting Task

In Chapter 3, 4 and 6, the delay discounting task (Kirby et al., 1999) was used. It consists of 27 hypothetical choices between a smaller immediate reward (£11–£80) and a larger reward (£25–£85) delayed between 7 and 186 days. The DDT designed for experiments 1 and 2, had a 1000ms pause between sentences and participants could take as long as necessary to choose. The task specifically designed for MEG, see Figure 2.4 for details, presented each part of the choice (immediate reward, future reward magnitude and future reward delay) for 2s and requested to choose one option by pressing a button. The primary measure of delay discounting was the proportion of the smaller, sooner rewards chosen out of the 27 choices. The most widely-used strategy to calculate the discounting rate is based on either a hyperbolic or an exponential function (e.g., Coffey et al., 2003; Dougherty et al., 2014; Mazur, 1987), however, the proportion score has also been used previously to assess delay discounting (Benningfield et al., 2014; Ersner-Hershfield et al., 2009; Magen et al., 2008). This alternative is a more straight-forward approach, as it does not make assumptions about the shape of the discounting curve and has similar results to the hyperbolic model (Benningfield et al., 2014), so it was decided to employ it for the current study.

This design allows the specific examination of the neural correlates involved in the processing of the immediate option, the magnitude of the future reward and the delay of the future reward separately. The only difference between the current task and the one by Ballard and Knutson (2009) is that here, the amount of money and days shown during the immediate, future reward magnitude and delay conditions, were selected based on the 27 choices from the Monetary Choice Questionnaire by Kirby et al. 1999. Instead, in Ballard and Knutson (2006), the amount of reward magnitudes and delay for the future reward offered was decided by the authors of the study.

Each participant completed two blocks of trials, the first containing 54 trials and the second 27, accounting for 81 total trials. These were the same 27 choices tested in three runs, two in one block and one in another, it was decided that an additional run would make the MEG recording too long, as it also included the three GNGSST runs. Because both the response inhibition task and the delay-discounting task described here were completed at the same time, the order of the blocks was decided to be presented as it follows: first block of GNGSST, first block of DDT, second block of GNGSST, second block of DDT, third block of GNGSST. Each GNGSST block was approximately 12 minutes long, interleaving GNGSST and DDT block of trials helped maintaining their attention.

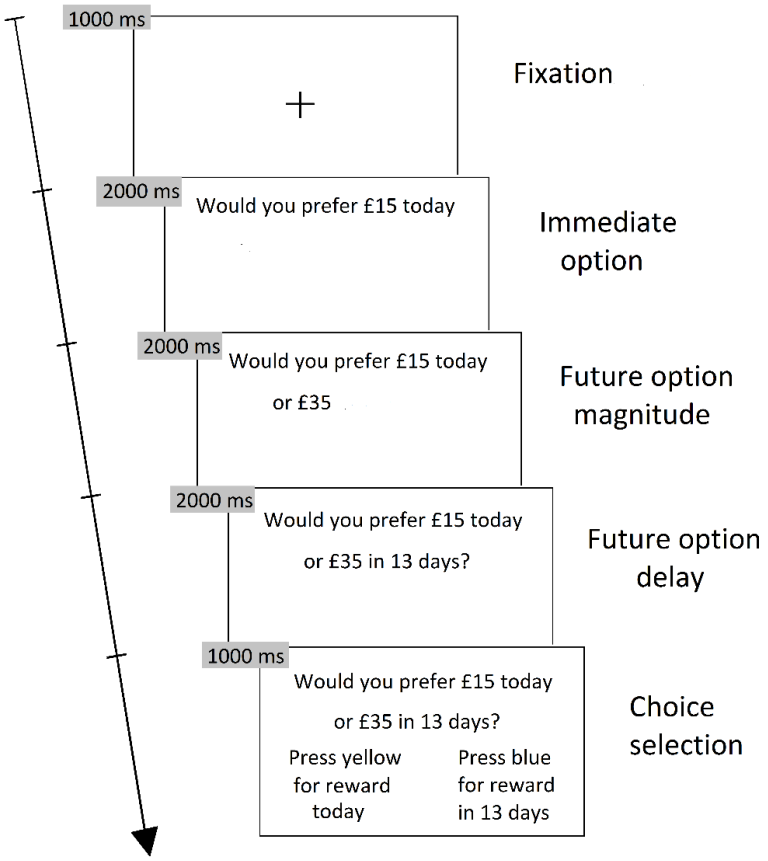

Figure 2.4. Example of one trial in the temporal discounting task. The first screen presented the immediate option offering a reward of £15 to be hypothetically received today. Next, the magnitude of the future option was presented, in which a hypothetical reward of £35 was offered. Next, the delay of the future option was presented, a delay of 13 days. Finally, the choice was shown, participants choose by pressing one button for the immediate reward today or the other for the larger reward in a future day.

2.6. Questionnaire measures.

The questionnaires were presented to participants via Bristol Online Surveys (https://www.onlinesurveys.ac.uk/) and all are included in Appendix I, see Table 2.1 for details on which self-report measures were used in each experiment.

2.6.1. Barratt Impulsiveness Scale.

The Barratt Impulsiveness Scale (BIS-11; Patton, Stanford, & Barratt, 1995) is a well-validated and reliable measure of trait impulsivity, possibly the most frequently used impulsivity questionnaire in research and clinical studies (Stanford et al., 2009). It consists of 30 items, comprising three subscales: non-planning (lack of future sense, 11 items), motor (acting on the spur of the moment without thinking, 11 items), attentional (distractibility, lack of sustained attention, 8 items), rated on a four-point Likert scale (1=rarely/never, 2=occasionally, 3=often,

4=almost always/always). High scores in the sum of all subscales of the BIS-11 indicate high levels of trait impulsivity as a heterogeneous concept while high scores in each specific subscale describe which components of impulsivity have a heavier weight.

Specifically, in experiments 1 and 2, the BIS-11 is one of the self-report measures of impulsivity tested, and in experiments 3 and 4, either high or low scores on the BIS-11 motor subscale was one of the two conditions, along with the BAS-Total scores, to be assigned to a high or low impulsivity group.

2.6.2. Eysenck Personality Questionnaire.

The Eysenck Personality Questionnaire (EPQ; Eysenck et al., 1985) is a 48-item questionnaire (responding YES or NO) with four subscales: Psychoticism, with items related to impulsivity; Extraversion, which relates to sociability and venturesomeness; Neuroticism, which measures emotional stability, along with a Lie scale assessing defensiveness. Higher values in each subscale indicate higher levels of the corresponding personality trait. This self-report measure, and similar versions of Eysenck's personality questionnaires, have previously been used to investigate the association between impulsivity self-reports and behavioural paradigms (e.g., Logan et al., 1997; and Marsh et al., 2002) but yielded inconsistent results. The EPQ was examined in experiments 1 and 2, adding this measure allowed further exploration of impulsivity dimensions towards providing a more robust measures of impulsivity.

2.6.3. UPPS Impulsive Behaviour Scale.

The UPPS Impulsive Behaviour Scale (UPPS; Whiteside & Lynam, 2001) is a 44-item, Likert-type scale (1=strongly disagree, 4=strongly agree) which has four factor-analytically-based subscales: Urgency, (lack of) Premeditation, (lack of) Perseverance and Sensation Seeking. High scores on this questionnaire indicate high levels of trait impulsivity. This scale was included as an additional measure of the two impulsivity dimensions explored here, because it includes one specific subscale for each dimension (Urgency - for rapid-response impulsivity and (Lack of) Premeditation - for reward-delay). Furthermore, Wilbertz and colleagues (2014) found the Urgency subscale, rather than the BIS-11, to be the only measure that explained individual variability on response inhibition performance. Hence, this measure was tested in experiments 1 and 2. I consider this might help clarifying how strong the relationships are between the two impulsivity dimensions investigated here.

2.6.4. Behavioural Inhibition System/Behavioural Activation System scales.

The Behavioural Inhibition System/Behavioural Activation System scales (BIS/BAS; Carver & White, 1994) consist of 20 items that are rated on a 4-point Likert-type scale (1=strongly disagree, 4=strongly agree) and comprise three BAS subscales (reward responsiveness, drive

and fun seeking) and one BIS subscale (reactions to the expectation of punishment). These scales assess participants' sensitivity of the behavioural approach system (BAS) and the behavioural inhibition system (BIS) to positive and negative cues. High scores on this questionnaire indicate high sensitivity to the BAS or BIS system (see Chapter 1 for further details). High scores in the sum of all subscales of the BAS subscales, BAS-Total, indicate high levels of BAS sensitivity, while high scores on each specific subscale describe which components of BAS sensitivity have a heavier weight.

The BAS subscales are tested in experiments 1 and 2, and in experiments 3 and 4, either high or low scores on the BAS-Total scale was one of the two conditions, along with the BIS-11 Motor scores, to be assigned to a high or low impulsivity group.

2.7. Statistical analysis

The methods employed for the statistical analysis of experiment 1 and 2 will be explained. Note however, that because the MEG analyses require a more detailed description, these will be explained in section 2.8.

2.7.1. Behavioural study

The behavioural and self-report measures described above were explored in a healthy sample in Analysis 1, Chapter 3. The sample was divided into two groups based on specific impulsivity scores, and the behavioural measures of impulsivity, the go/nogo, stop and delay discounting task, were tested between groups using statistical analysis. The Kolmogorov–Smirnov test for normality and Levene's test for homogeneity of variances were used to assess whether assumptions were met. Spearman's r was used to examine correlations between self-report measures and cognitive tasks as a first, exploratory step. Subsequently, multivariate analyses of covariance (MANCOVA) were conducted to test differences between groups on the self-report measures and the cognitive tasks, separately. Conducting multiple ANCOVAs or a single MANCOVA on both self-report measures and cognitive tasks, would increase the chance of committing a Type I error. For significant MANCOVAs, univariate ANOVAs were conducted to test a priori hypotheses, using Bonferroni correction as implemented in SPSS, which adjusts the p-value. In cases where MANCOVA results were non-significant, although this would imply that impulsivity groups were not significantly different overall for those measures, the specific a priori hypotheses were tested using univariate ANOVAs. Univariate ANOVAs allow the identification of the specific dependent variables that contributed to the non-significant overall effect, while the Bonferroni correction was used to protect from Type I error.

Variables included in the analyses were the cognitive tasks measures, the go/nogo, stop and delay discounting task, and the scores on the self-report measures. A covariate was also included, participants' history of mental health, this was assessed by past or current visits to a mental health professional, a question from the demographic and health questionnaire. Sex or age did not meet all the assumptions to be included as a covariate, i.e., independence of covariate and independent variable, homogeneity of the regression slopes and, when included, did not change the result of the analyses. Partial eta-squared η^2 values were calculated to measure effect size and interpreted using Cohen's (Cohen, 1988) guidelines for determining small (0.01), medium (0.06) and large (0.14) effects. All statistical analyses were performed in SPSS (Version 22.0; SPSS Inc., Chicago, IL, United States).

2.7.2. Exploratory factor analysis

In the second analysis of the behavioural study, Chapter 4, all the measures tested in Analysis 1 were initially included in the analysis, examined for normal distributions and modified if necessary, using transformations. The GNGT proportion of commission errors, SST proportion of commission errors and the DDT proportion of preferred choices were transformed using the arcsine transformation, which takes the arcsine of the square root of a number. SST Reaction times were calculated using the median. Given the diverse findings regarding the appropriate number of factors reported in previous exploratory and confirmatory factor analysis studies, first, an exploratory principal axis factoring factor analysis was conducted, also referred to as common factor analysis. This analysis was performed using Factor 10.4 software (Lorenzo-Seva & Ferrando, 2006) and included 15 variables. Common factor analysis was conducted because the aim was to identify the least number of factors that can explain common variance. This differs from alternative methods, such as principal component analysis, which considers all variance in the data with the primary objective of data reduction (Hair, Black, Babin, & Anderson, 2010; Chapter 5).

The model resulting from our common factor analysis, and its set of relationships, were then tested using structural equation modelling (SEM), via AMOS 23.0 (Arbuckle, 2014). Structural path analyses subsequently examined the associations between factors. The main difference between our common factor analysis and SEM is that in the latter, the variables and the associations to their latent construct need to be specified, while the common factor analysis does not need prior specification (Hair et al., 2010; Chapter 11). Based on the recommendations by Hu and Bentler (1999) the following indices were used to evaluate model fit: root-mean-square error of approximation (RMSEA; <0.08), comparative fit index (CFI; >0.90), and Tucker-Lewis index (TLI; >0.95). These indices will be used to indicate which model might fit best using a descriptive approach. This approach was chosen because the models analysed here are non-nested, i.e., factors are not necessarily related and therefore

other methods of assessing model fit, such as chi-square difference test, would not be meaningful.

2.8. Analysis of MEG data

MEG data were acquired using a 306-channel Neuromag MEG scanner (Vectorview, Elekta, Finland) with 204 planar gradiometers and 102 magnetometers, in a magnetically shielded room and at a sampling rate of 1000 Hz. Five head position indicator (HPI) coils were attached to specific sites on the subject's head for continuous head position tracking. During the MEG recording, participants completed five blocks of trials, three GNGSST blocks and two DDT blocks interleaved, and because of the size limits when saving raw MEG data, each block of trials was saved separately. Each run was first pre-processed separately using Maxfilter software (version 2.0, Elekta-Neuromag) with temporal signal space separation (tSSS) using a .9 correlation, which removes the interference produced by external sources of noise (Taulu and Simola, 2006). For the GNGSST, runs 2 and 3 were mapped onto run 1 to compensate for possible head movements occurred between runs. For the DDT, each participant's head position in run 2 was transformed to that of the start of run 1. Although Elekta software was used for head modelling, all the analyses were conducted using Fieldtrip, toolbox version 20161024 (Oostenveld et al., 2011) in Matlab 2014b (Mathworks Inc., Natick, MA).

2.8.1. Pre-processing

After tSSS, each participant's recording of each block of trials was band-pass filtered between 0.5-50Hz (as in Boehler et al., 2009) and band-stop filtered (49.5-50.5Hz) to remove residual 50Hz line noise. For the analysis of the Go/NoGo/Stop-Signal Task (GNGSST), each participant's entire recording was segmented into trials of 1600 ms, 800 ms baseline and 800 ms after onset display, as in previous MEG studies (Boehler et al., 2009; Nakata et al., 2005). In Boehler et al. (2009), the response to the GO cue occurred on average 450 ms after stimulus onset, while the SSRT was 240 ms. In a recent EEG study by Raud et al. (2019) for example, the response to the GO cue during the go/nogo task occurred 400 ms after stimuli onset, while the response to the GO cue during the Stop-Signal task was observed 600 ms after the cue was displayed. In our MEG study, the mean reaction time for the correct GO trials were 530 and 515 ms for the HI and LI groups, respectively, while mean SSRT in the HI group was 260 ms and 240 ms in the LI group. For the delay discounting task (DDT), data was segmented into trials of 9000 ms (1000 ms pre, 8000 ms post fixation onset), each of the three conditions was presented for 2000 ms and the choice for 1000 ms.

All trials were manually examined to identify any trials containing artefacts, such as eye blinks, muscle contractions or head movements, which were then manually removed if the trial-by-channel variance was higher than 8x10-23 (as in Seymour et al., 2017). In the GNGSST, these pre-processing steps resulted in an average of 280 successful GO trials (from 360 GO trials), 80 successful STOP trials (from 240 STOP trials) and 50 (60) successful NOGO trials (from 60 NOGO trials) per participant. The GNGSST was designed in E-Prime based on the task used in the MEG study by Boehler and colleagues (2009). As in their study, the current MEG study intended to compare Hi and LI groups on successful and unsuccessful inhibition, as measured by successful and unsuccessful STOP trials. In their study, subjects successfully inhibited the responses in 50% of trials (Boehler et al., 2009), however, this did not happen in our experiment. Our results showed some subjects had much lower rates of unsuccessful stopping, impeding the analysis of HI vs LI successful trials and of HI vs LI unsuccessful trials. Thus, only successful HI versus LI stop trials were compared. During the DDT, an average of 64 trials per participant resulted from the pre-processing steps, in each of the three conditions (immediate reward, future reward magnitude, future reward delay) for statistical comparison. When it was ensured that the data was adequately clean for the following analyses, the blocks of trials were concatenated for each task, GNGSST (Experiment 3) and DDT (Experiment 4).

2.8.2. Sensor level analyses

For analyses in sensor-space, an independent component analysis (ICA) was first conducted to correct for cardiac, ocular and movement artefacts. Components containing these artefacts, see image 2.1 for an example of an artefact, were then removed from the data while the remaining components were projected to sensor-space again. From here, data was examined in sensor level using two approaches: analysing the averaged signal based on the timing of an event and analysing the MEG signal in the time-frequency representation.

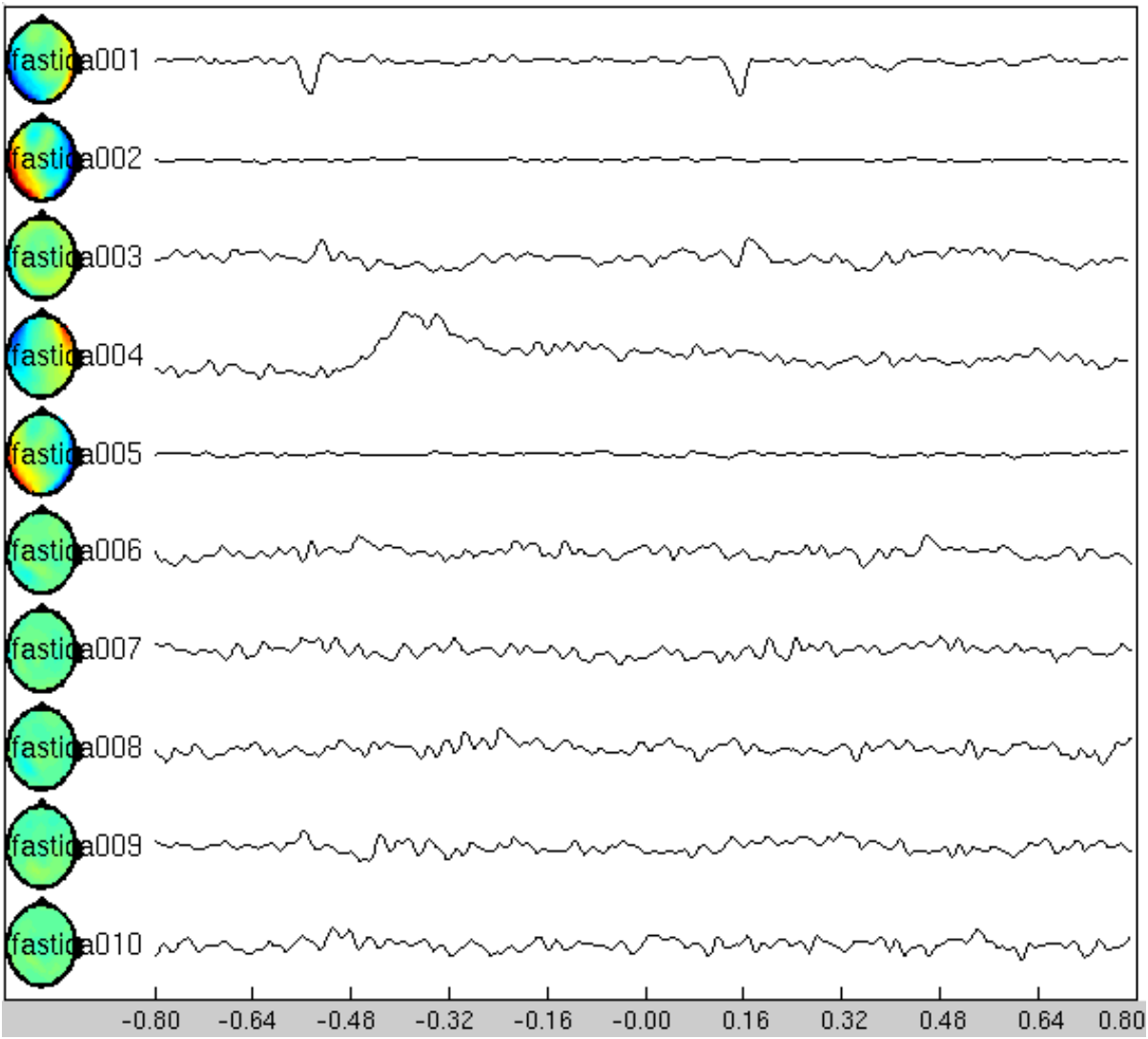

Image 2.1. Screenshot taken during ICA, components to be rejected are visually inspected for artefacts, using Fieldtrip toolbox in Matlab.

2.8.2.1. Analysis of event-related fields

The averages of the ERFs, time-locked to the onset of the stimulus, were computed across trials for each participant for each condition in the GNGSST or the whole trial in the DDT. Next, the 204 planar gradients were combined for each of these averages, resulting in 102-channel combined planar gradiometers, and baselined between -100 and 0 ms from the onset of the stimulus. Cluster-based non-parametric permutation tests allow examining for differences between groups, specifically, the Monte Carlo method was utilised to control for family-wise error (Maris & Oostenveld, 2007), with 1000 iterations, two-tailed and with a cluster alpha of 0.05. To conduct these tests, every pair of trials, one from each condition, were compared by using a *t*-value. Those pairs with a significant *t*-value were then clustered with temporally adjacent cortical locations, statistics on clusters were conducted by calculating the sum of t-

values on each cluster (Maris & Oostenveld, 2007). For cluster-based non-parametric permutation testing, this was performed with 1000 iterations, two-tailed *t*-test and with a cluster alpha of 0.05. This approach allowed the comparison of ERFs generated during NOGO and STOP conditions between HI and LI groups of subjects, 17 participants in each group, between 0 and 800 ms after stimulus onset (as explained in 2.7.1). In the DDT, the ERFs generated in HI and LI groups of subjects, with 17 participants in each group, were statistically compared across the three conditions of interest: the immediate reward condition (1000 to 3000 ms), future reward condition (3000 to 5000 ms) and future delay condition (5000 to 7000 ms). Finally, *t*-statistics, multi-channel plots of the averaged ERFs and topographical plots were generated, which can be found in the results section of MEG experiments 1 and 2 (chapters 5 and 6).

2.8.2.2. Time-frequency analysis

Frequency analysis was conducted using a Hanning taper from 1 to 30 Hz in steps of 1 Hz in all trials resulting from the pre-processing and ICA steps. Each time window had a length of 200 ms in steps of 50 ms, trials were 800 ms pre- and post-cue long, resulting in four cycles per time window. In the DDT, each time window also had a length of 200 ms in steps of 50 ms, trials of each condition (2000 ms), were compared to baseline (2000 ms), resulting in ten cycles per time window. Next, each participant's trials were averaged separately for each condition, NOGO, STOP, DDT immediate, DDT future magnitude and DDT future delay, and planar gradiometers combined as in ERFs analysis (see 2.8.2.1). Delta (1-4 Hz), theta (4-8 Hz), alpha (8-12 Hz) and beta band (13-30 Hz) power were compared between HI and LI groups, during NOGO, STOP and DDT conditions, while correcting for multiple comparisons using cluster-based non-parametric permutation tests (Maris & Oostenveld, 2007), with 1000 iterations, two-tailed and with a cluster alpha of 0.05. This approach allowed *t*-statistics, time-frequency representations and topographical plots to be generated, these can be found in the results section of MEG experiments 1 and 2 (chapters 5 and 6).

2.8.3. Source level analyses

The first step of source level analyses was to perform MEG-MRI co-registration for each participant, resulting in the generation of individual head-models.

2.8.3.1. MEG-MRI Co-registration

Using a Siemens MAGNETOM Trio 3T scanner with a 32-channel head coil, a structural T1 scan was obtained for each participant, consisting of 192 slices, to use in following steps for source reconstruction. To co-register the MRI with the MEG data, the T1 was matched with fiducial positions and head shape data acquired during head digitisation prior to MEG data

acquisition and aligned to the sensor array. The method used for co-registration was based on Nolte (2003) and was conducted using Elekta software and Fieldtrip. Firstly, the head shape acquired with the Polhemus Fastrak was extracted using Seglab (Elekta software), then the anatomical data was segmented, and the resulting files were used to create a single-shell head model for each participant. Next, to align the MEG and MRI data, subject-specific volumetric grids were created based on a template grid (8mm resolution), which were warped into the standard MNI-space (Montreal Neurological Institute), enabling comparisons across individuals and between groups. See image 2.2 for an example. The individual volumetric grids were then used to compute the forward solution, expressed as a leadfield matrix.

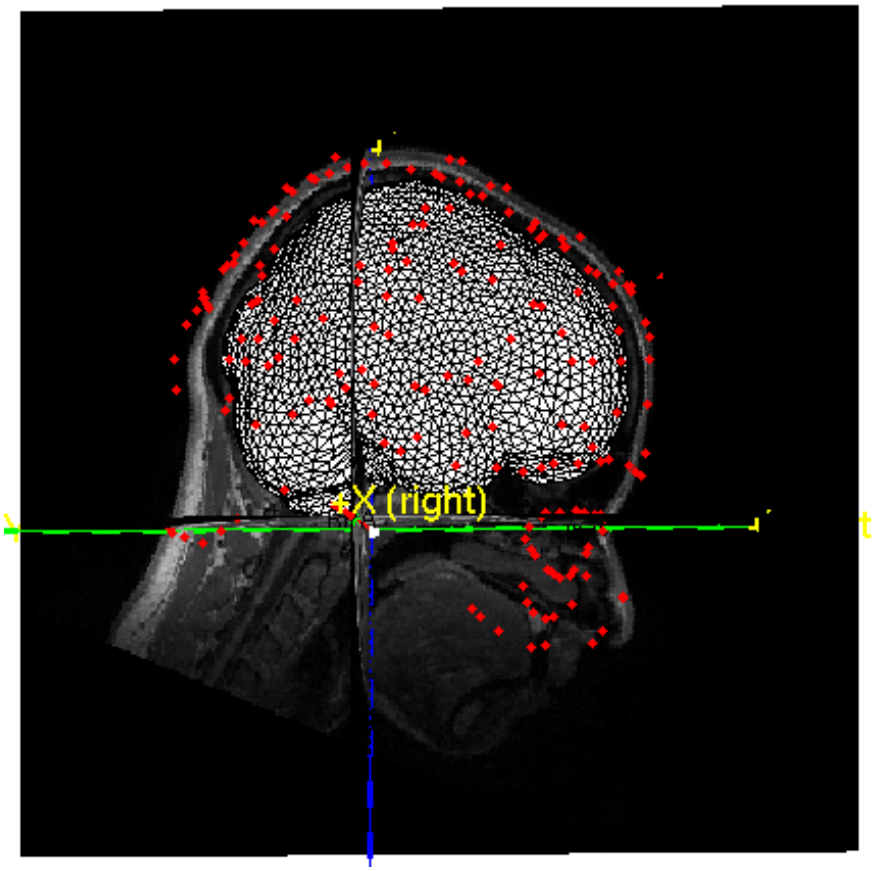

Image 2.2. Participant 18, screenshot taken during co-registration, example of anatomic data aligned with fiducial positions and head shape data acquired during head digitisation.

The subject-specific leadfields, along with covariance or cross-spectral density matrices, were used for source analysis of event-related field and oscillatory activity, respectively. By using beamformers, the common spatial filters were computed at each location of the volumetric grid in each condition, see next sections for details. Three participants could not be scanned using MRI due to health and safety reasons and thus, a standard T1 template was used for source analysis (see Gohel, Lim, Kim, Kwon, & Kim, 2017). To compensate for using Maxfilter on each run separately, a principal component analysis (PCA) was applied, this reduced the dimensionality of the data to components that contributed to 99% of the variance in the

covariance or cross-spectral density matrices. From here, data was examined in source-space using two approaches: analysing the averaged signal based on the timing of ERFs and identifying the oscillatory components involved.

2.8.3.2. Analysis of event-related fields

After rejecting any components containing artifacts using PCA in Fieldtrip, the time windows of interest were defined based on the significant results from the cluster statistical analysis conducted, see section 2.8.2.1. Then the covariance matrix computed for each condition on the defined time windows. To analyse the ERFs generated during each condition in source-space, a beamformer technique was applied, the linearly constrained minimum variance beamformer (LCMV; Van Veen, van Drongelen, Yuchtman, & Suzuki, 1997). First, using the individual leadfield matrices and head models computed before and the MEG data as a covariance matrix, one spatial filter per voxel was calculated on the defined time window, in each condition for each participant.

To statistically test for differences in the amplitudes of EFRs between HI and LI groups during the GNGSST and DDT, cluster-based non-parametric permutation testing was used to avoid the multiple-comparisons problem (Maris & Oostenveld, 2007). Specifically, the Monte Carlo method with 1000 iterations, two-tailed and a cluster alpha of 0.05 was applied in Fieldtrip. Using the MRI anatomical data and a template anatomical atlas, both in the same coordinate system, statistical parametric mapping (SPM), the data was interpolated to a cortical sheet from canonical MNI brain. The interpolated data was then projected to an MNI white-matter surface, for example, see image 2.3. In MEG experiments 1 and 2, differences between HI and LI groups in ERFs during NOGO, STOP and DDT conditions in source-space were shown using these projections.

2.8.3.3. Frequency and time-frequency analysis

After rejecting any components containing artifacts using PCA in Fieldtrip, the time windows of interest were defined based on the significant results from the cluster statistical analysis conducted in 2.8.2.2. Using the fast-fourier-transform (FFT) of signals from the gradiometers and a Hanning taper, the cross-spectral density matrix was computed for each condition. To analyse the oscillatory activity generated in source-space, a beamformer technique appropriate for frequency data was applied, the dynamic imaging of coherent sources technique (DICS; Groß et al. 2001). Using the individual leadfield matrices and head models, the beamformer was applied to the data resulting from the frequency analysis conducted initially. Statistical analysis of differences in oscillations between HI and LI groups during the GNGSST and DDT, was conducted in the same manner as in 2.8.3.2, in delta (1-4 Hz), theta (4-8 Hz), alpha (8-12 Hz) and beta (13-30 Hz) bands separately. Finally, the anatomical,

functional and statistical data was projected to an MNI white-matter surface, for example, see image **2.3**. In MEG experiments 1 and 2, differences between HI and LI groups in the frequency domain, during NOGO, STOP and DDT conditions in source-space were shown using these projections.

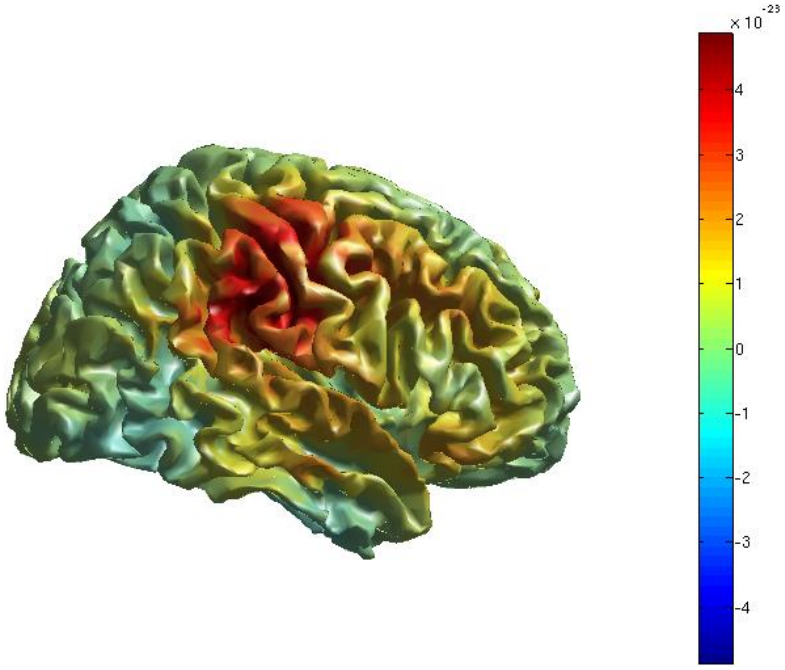

Image 2.3. Participant 7's reconstruction of the activity generated during the GO condition plotted onto a standard MNI brain at 400 ms after stimulus onset.

CHAPTER 3: Linking measures of response inhibition and reward responsiveness to trait impulsivity

3.1. CHAPTER AIMS

Although the two dimensions of impulsivity, rapid-response and reward-delay impulsivity, have been extensively investigated, there are still considerable issues that need to be clarified: First, the lack of consistency regarding the extent of associations between cognitive tasks and self-reports; and secondly, the need to further understand the relationship between these two dimensions of impulsivity. This study aimed to directly relate self-report measures of impulsivity to cognitive measures of impulsivity in individuals at low- or high-levels on two impulsivity dimensions, rapid-response impulsivity and reward-delay impulsivity. Participants were classified into high- or low-impulsivity groups based on (1) level of rapid-response impulsivity (determined by BIS-11 Motor subscale scores); (2) level of reward-delay impulsivity (determined by BIS/BAS subscale scores) and (3) a combination of rapid-response impulsivity and reward-delay impulsivity levels. Impulsivity was assessed using Go/No-Go, Stop-Signal and Delay-Discounting tasks and self-report measures. Behavioural differences were tested between high and low impulsivity groups and correlational analyses of all variables were also conducted. Here, results from these analyses are presented and discussed. A version of this chapter has been published in a peer-reviewed journal:

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3.2. INTRODUCTION

Impulsivity, as a trait, is defined as “a predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions to the impulsive individual or to others” (Moeller et al., 2001; p. 1784). Thus, impulsivity is a socially relevant construct, impacting on society (e.g., business, criminal justice, education) as well as individuals (e.g., aggressive or anti-social behaviours) (Stanford et al., 2009). It has been reported to characterise several mental disorders, such as attention deficit/hyperactivity disorder (ADHD; Nigg, 2001), drug addiction (Jentsch & Taylor, 1999; Bari & Robbins, 2013), and bipolar spectrum disorders (Strakowski et al., 2009). Trait impulsivity is assessed by using self-report measures such as the Barratt Impulsiveness Scale (BIS-11, Patton & Stanford, 1995), the UPPS Impulsive Behaviour Scale (Whiteside & Lynam, 2001) or the Eysenck Personality Questionnaire (EPQ; Eysenck et al., 1985), specifically the Psychoticism subscale (Holt et al., 2003; Lewis et al., 2009).

Impulsivity is regarded as a multifaceted construct that comprises two dimensions: rapid-response impulsivity (also referred to as response inhibition or impulsive action) and reward-

delay impulsivity or impulsive choice (Winstanley, et al., 2004, Alloy et al., 2009; Dawe & Loxton, 2004; Dawe et al., 2004; MacKillop et al., 2016; Swann, 2010). Rapid-response impulsivity refers to a tendency to perform immediate actions, often without any forethought or a diminished ability to inhibit a pre-potent response (Moeller, et al., 2001; Hamilton et al., 2015). Impulsive choice is described as diminished ability or willingness to tolerate delays (Hamilton et al., 2015). Although both constructs link back to core theoretical definitions of impulsivity, they tend to correlate only weakly or not at all (Broos, et al., 2012; Lane, et al., 2003; Reynolds, et al., 2006). These dimensions of impulsivity can be assessed using cognitive tasks, in addition to self-report measures. However, it is not clear which aspects of trait impulsivity, as assessed by self-report measures, such as the BIS-11, are related to response inhibition and/or to reward-delay impulsivity, as different results have been reported in studies using both self-report measures and cognitive tasks (Dick et al., 2010).

Rapid-response impulsivity has been investigated using the go/no-go task (GNGT) and the stop-signal task (SST) (Criaud & Boulinguez, 2013; Hamilton et al., 2015). These paradigms have been proposed to assess two different processes. GNGT assesses “action restraint” as it measures the inhibition of a planned response. Omission errors, i.e., withholding a response to a “Go” stimulus, and commission errors, i.e., responding to a “No Go” stimulus, index rapid-response impulsivity. SST measures “action cancellation” as it assesses the inhibition of an initiated response (Bari & Robbins, 2013; Eagle et al., 2008; Schachar et al., 2007; Swick et al., 2011). This distinction is supported by growing evidence from neuroimaging studies (e.g., Dambacher et al., 2014; Sebastian et al. 2013; Swick et al., 2011) which have shown different, in addition to common, neural patterns of activations when both paradigms are examined. The current study examined both paradigms to assess both functional aspects of impulsivity.

Self-report measures that tap into rapid-response impulsivity include the Motor-subscale of the BIS-11 and the Urgency subscale of the UPPS, the latter being reported to significantly correlate with SST performance (Wilbertz et al., 2014). However, significant associations between cognitive tasks assessing response inhibition and self-report measures are not consistently reported (e.g., Aichert et al., 2012; Enticott et al., 2006; Horn et al., 2003; Keilp et al., 2005; Lijffijt et al., 2005; Malesza & Ostaszewski, 2016; Reynolds et al., 2006; Rodriguez-Fornells et al., 2002; Spinella, 2004).

The second dimension of impulsivity, reward-delay impulsivity or impulsive choice has been investigated in terms of the behavioural approach system (BAS) hypersensitivity model (e.g., Alloy & Abramson, 2010; Alloy et al., 2012; Duek et al., 2014; Molz et al., 2013; Nusslock et al., 2014). The reward hyper-sensitivity model proposes that individuals with a hyper-sensitive BAS may show exaggerated approach behaviours towards reward and goal cues (Molz et al.,

2013). This can lead to drastic fluctuations of BAS activation and deactivation. Activation of the BAS by positive cues (e.g., positive life events, specifically those involving goal-striving and goal-attainment), can result in characteristics, or symptoms, such as increased energy, optimism, decreased need for sleep (Alloy et al., 2009). In contrast, deactivation of the BAS by negative cues (e.g., negative life-events, specifically those including failure to obtain - or loss of - goals/rewards) can result in depressive characteristics, or symptoms, resembling depressions, such as anhedonia, decreased energy or sadness (Alloy & Abramson, 2010; Urosevic et al., 2008).

It has also been suggested that BAS hyperactivity may result in impulsive decision-making (Mason et al., 2012). When a reward cue activates the hyper-reactive BAS, anticipation of this reward may be responsible for generating an impulsive "state" (Bari & Robbins, 2013) which influences decision-making. BAS hyper-sensitivity towards rewards may result in an inability to delay gratification. This behaviour is assessed using questionnaires such as the BIS/BAS scales (Carver & White, 1994). The UPPS (Lack of) Premeditation subscale has also been used to assess this inability to delay gratification (Alloy et al., 2008, 2009; Lynam & Miller, 2004, Stojek, Fischer, Murphy, & MacKillop, 2014; Urosevic et al., 2008). It has been reported that individuals scoring low on this subscale were more likely to prefer small and immediate rewards compared to larger but delayed rewards (Lynam & Miller, 2004, Stojek et al., 2014;).

Previous experimental studies have assessed reward-delay impulsivity using the delay discounting task (DDT; Kirby et al., 1999). This task requires participants to choose between either small but immediate, or large but delayed rewards, typically amounts of hypothetical money. Higher rates of delay discounting are associated with self-report measures of impulsivity (e.g., Kirby et al., 1999), sensation seeking (Richards et al., 1999) or suicidal ideation and behaviour (Cáceda et al., 2014). Greater discounting has also been reported in populations with impulse control problems, such as compulsive gamblers (Ledgerwood et al., 2009; Reynolds et al., 2006), acute alcohol, cocaine and methamphetamine users (Bari & Robbins, 2013; Coffey et al., 2003) or tobacco smokers (Baker et al., 2003). Similarly to inconsistent results reporting correspondence between cognitive tasks and self-report measures assessing rapid-response impulsivity, evidence shows that reward-delay tasks and self-report measures do not always correlate well (e.g., Murphy & Mackillop, 2012; Reynolds et al., 2006). Stojek and colleagues (2014), for instance, suggested that reward-delay tasks may not be sufficiently sensitive to assess particular impulsivity dimensions, as measured by questionnaires, yet, the reverse could also be true due to the biased nature of self-report measures.

Current study

The current study set out to re-investigate previous failures of finding a strong relationship between cognitive and self-report measures of impulsivity dimensions, specifically response inhibition and reward-delay. Therefore, it is of vital importance to investigate these two dimensions in conjunction. Here, self-report measures of trait impulsivity were administered to classify individual impulsivity levels, and then relate this classification to cognitive measures of impulsivity. The aim was to clarify the robustness of self-report questionnaires as genuine predictors of behavioural impulsivity. Although the two dimensions of impulsivity, rapid-response and reward-delay impulsivity, have been extensively investigated, there are still considerable issues that need to be clarified: First, the lack of consistency regarding the extent of associations between cognitive tasks and self-reports; and secondly, the need to further understand the relationship between these two dimensions of impulsivity. In the present study, associations between rapid-response impulsivity and reward delay impulsivity will be investigated in an undergraduate student population. Inhibitory control and reward sensitivity seem naturally inter-related concepts, however, studies of such interactions have been limited. Contrary to most published reports, Meda et al. (2009) showed good correspondence between cognitive and self-report measures of impulsivity, including a delay-discounting task and BIS/BAS, BIS-11 measures.

Here, participants were classified into high and low impulsivity groups, depending on the absence, or presence, of the following: (a) level of rapid-response impulsivity – as determined by BIS-11 Motor sub-scale scores, (b) level of reward-delay impulsivity – as determined by BAS Total scores and (c) a combination of both levels. Factor analyses of the impulsivity construct have proposed that impulsivity domains such as rapid-response impulsivity and reward-delay impulsivity reflect discrete impulsivity dimensions (e.g., MacKillop, et al., 2016). However, overlap has also been reported (e.g., Meda et al., 2009), suggesting that impulsivity domains may be less distinct. A combined group was therefore added to investigate to what extent trait dimensions of rapid-response impulsivity and reward-delay impulsivity together would impact on and interact with cognitive tasks. These groups performed response inhibition and reward-delay experimental tasks to clarify the relationship between self-report measures and behavioural effects, and to assess how sensitive these measures are to differences between high- and low-impulsivity groups. Associations between our cognitive tasks assessing rapid-response and reward-delay impulsivity will also be examined to further elucidate the contradictory results reported to date. Overall, this will help clarifying the relationship between self-report and cognitive measures of trait impulsivity as well as the relationship between the dimensions of response-inhibition and reward delay.

It was hypothesised that: (1) Performance on the cognitive tasks assessing each of the two dimensions of impulsivity (i.e., rapid-response and reward-delay impulsivity) will correlate with the corresponding self-report measures assessing each dimension. (2) Single high-impulsive vs. single low-impulsive and combined high impulsivity vs. low-impulsive groups will perform significantly differently on cognitive tasks assessing impulsivity: High-impulsive individuals (single and combined high-risk) are expected to show impaired response inhibition on the GNGT and SST, and an inability to delay reward on the DDT. (3) Single high-impulsive vs. single low-impulsive and combined high- vs. low-impulsive groups will score significantly differently on self-report measures assessing rapid-response and reward-delay impulsivity: High-impulsive individuals (single and combined impulsivity dimensions) are expected to score significantly higher on self-report measures than the low-impulsive group.

3.3. METHODS

3.3.1. Participants

See section 2.3.1 for details.

3.3.2. Procedure

See section 2.4.1 for details.

3.3.3. Cognitive tasks

See sections 2.5.1, 2.5.2 and 2.5.4 for details.

3.3.4. Self-report measures

See section 2.6 for details.

3.3.5. Group Assignment

See section 2.4.3 for details.

3.3.6. Statistical Analysis

See section 2.7.1 for details.

3.4. RESULTS

3.4.1. Correlational analyses

As some of the variables did not meet assumptions for parametric analysis, Spearman's rho was used for all analyses to examine the correlations between the variables from the self-report measures and the cognitive tasks. The significance level was adjusted using Bonferroni correction, so that significant correlations reported here are based on the adjusted p values (see Table 3.1). Significant positive correlations were observed between the DDT and the BIS-11 Total score rs=0.28 ($p<0.0001$, 2-tailed) and BIS-11 Non-Planning subscale rs=0.29 ($p<0.0001$, 2-tailed). Similarly, the self-report measures assessing different aspects of impulsivity, and their subscales, also showed significant correlations: The BIS-11 Total score and the three BIS-11 subscales (motor, attention and non-planning) significantly correlated with all UPPS subscales (urgency, premeditation and perseverance) except for Sensation Seeking (which significantly correlated with the BIS-11 Total score only), with the EPQ Psychoticism subscale and BAS Fun subscale, while the BIS-11 Motor subscale also significantly correlated with BAS Drive. The BAS Reward subscale significantly correlated with two UPPS subscales, (Lack of) Perseverance and (Lack of) Premeditation, while the BAS Drive subscale significantly correlated with (Lack of) Perseverance. The BAS Fun subscale significantly correlated with the Urgency, (Lack of) Premeditation and Sensation Seeking UPPS subscales (for all coefficients, see Table 3.1).

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1.GNGT Number Errors	1	-.25*	.29*	-.32*	.18	.17	.19	.14	.11	.03	-.05	.01	.07	.09	.09	.07	.03
2.GNGT RT		1	-.05	.24*	-.01	.01	-.02	.01	.01	.04	.15	-.02	.00	.10	-.04	-.08	-.10
3.SST Number Errors			1	-.71*	.03	.10	.06	.12	.10	.02	-.03	-.02	.05	.13	.04	-.01	.03
4.SSRT				1	-.04	-.06	-.04	-.09	-.03	.04	.10	.01	-.07	-.02	-.04	.01	-.08
5.DDT					1	.28*	.29*	.16	.21	.09	.06	.07	.13	.07	.09	.04	.06
6. BIS-11 Total						1	.89*	.72*	.85*	.03	.14	.44*	.31*	.54*	.61*	.49*	.17*
7. BIS-11 Non-Planning							1	.47*	.62*	-.08	.02	.28*	.25*	.47*	.65*	.53*	.09
8. BIS-11 Attention								1	.52*	.09	.06	.31*	.23*	.49*	.36*	.40*	.19
9. BIS-11 Motor									1	.13	.29*	.51*	.26*	.43*	.49*	.26*	.18
10. BAS Reward										1	.34*	.34*	-.12	-.03	-.33*	-.31*	.20
11. BAS Drive											1	.42*	-.03	.02	-.05	-.33*	.18
12. BAS Fun												1	.23	.26*	.24*	.08	.51*
13. EPQ Psychoticism													1	.12	.16	.21	.15
14. UPPS Urgency														1	.39*	.41*	.06
15. UPPS Premeditation															1	.48*	.09
16. UPPS Perseverance																1	-.03
17. UPPS Sensation seeking																	1

Table 3.1. Correlational analyses between impulsivity variables using Spearman's rho; Bonferroni adjusted p-value: 0.05/17=0.002. GNGT, Go/No-Go Task; RT, reaction time; SST, Stop-Signal Task; SSRT, Stop-Signal reaction time; DDT, Delay Discounting Task; BIS-11, Barratt Impulsiveness Questionnaire 11; BAS, Behavioural Activation System; EPQ, Eysenck Personality Questionnaire; UPPS, Urgency, Premeditation Perseverance, Sensation (Questionnaire). *p<0.002 (2-tailed)

3.4.2. Cognitive tasks and questionnaire results based on impulsivity group

3.4.2.1. Rapid-response impulsivity

Rapid-response impulsivity was assessed by computing the scores of the BIS-11 Motor subscale. Those with scores within the highest 35th percentile of the motor scores in the whole sample constitute a high rapid-response impulsivity group (n=73). Participants within lowest 35th percentile on the BIS-11 Motor subscale constitute a low rapid-response impulsivity group (n=65). This procedure is in line with Wilbertz et al. (2014). Means and standard deviations of variables are presented in Table 3.2 and Table 3.3.

Variable	Low Rapid-response impulsivity group (*n*=65)	High Rapid-response impulsivity group (*n*=73)	F(1,135)	Partial Eta Squared
GNGT – number of commission errors	5.17(6.3)	5.71(5.6)	0.27	0.00
GNGT – Mean RT	363.19(55.4)	358.96(64.8)	0.18	0.00
SST – number of commission errors	12.91(9.1)	15.03(9.9)	1.7	0.01
SSRT	275.19(160.4)	258.89(162.9)	0.41	0.00
DDT	0.53(0.2)	0.59(0.1)	7.56**	0.05

Table 3.2. MANCOVA results for behavioural measures for *Rapid-response impulsivity group*: Presented are raw means (standard deviations), F-statistics for each univariate ANOVAs with their corresponding p-values and partial eta-squared estimates. For the DDT, the proportion of small-immediate/large-delayed reward choices is shown. GNGT, Go/No-Go Task; RT, reaction time; SST, Stop-Signal Task; SSRT, Stop-Signal reaction time; DDT, Delay Discounting Task. *p<0.05, **p<0.01.

A MANCOVA was performed to contrast the two groups on the cognitive tasks while controlling for mental health history (see Table 3.2). The overall MANCOVA showed that groups classified by their level of motor impulsivity were not significantly different from each other (Wilks λ=0.94, $F(5,131)=1.77$, $p=0.123$, $\eta^2=.06$). Although the overall MANCOVA was not significant, univariate ANOVAs were conducted to test a priori hypotheses, using Bonferroni correction as implemented in SPSS. This revealed significant differences between groups for the proportion of smaller vs. larger reward choices in the DDT, with a close to medium effect size estimate ($F(1,135)=7.56$, $p=0.007$, $\eta^2=.05$).

Variable	Low Rapid-response impulsivity group (*n*=65)	High Rapid-response impulsivity group (*n*=73)	F(1,135)	Partial Eta Squared
BAS Total	39.02(4.1)	41.41(4.8)	9.86**	0.07
BAS Reward	17.51(1.8)	17.75(1.9)	0.66	0.01
BAS Drive	10.68(1.9)	11.21(2.4)	2.01	0.02
BAS Fun	10.83(1.8)	12.45(2.1)	23.56**	0.15
EPQ Psychoticism	2.00(1.5)	2.89(1.7)	10.21**	0.07
UPPS Urgency	26.17(5.3)	31.93(6.9)	32.14**	0.19
UPPS (Lack of) Premeditation	18.02(3.6)	22.92(5.3)	41.05**	0.23
UPPS (Lack of) Perseverance	18.57(4.9)	22.12(5.2)	17.41**	0.11
UPPS Sensation Seeking	32.09(7.1)	33.82(7.0)	2.12	0.02

Table 3.3. MANCOVA results for psychometric measures for *Rapid-response impulsivity group*: Presented are raw means (standard deviations), F-statistics for each univariate ANOVAs with their corresponding p-values and partial eta-squared estimates. BAS, Behavioural Activation System; EPQ, Eysenck Personality Questionnaire; UPPS, Urgency, Premeditation Perseverance, Sensation (Questionnaire). *p<0.05, **p<0.01

A MANCOVA was performed to contrast the two groups on the self-report measures while controlling for mental health history (see Table 3.3). The overall MANCOVA showed that groups classified by their level of motor impulsivity were significantly different from each other (Wilks λ=0.62, F(8,128)=9.84, p<0.0001, η^2=.38). Univariate ANOVAs revealed significant differences between groups for BAS Total scores (F(1,135)=9.86, p=.002, η^2=.07), BAS Fun Seeking scores (F(1,135)=23.56, p<0.0001, η^2=.15), EPQ Psychoticism scores (F(1,135)=10.21, p=0.002, η^2=.07), UPPS Urgency (F(1,135)=32.14, p<0.0001, η^2=.19), UPPS (Lack of) Premeditation scores (F(1,135)=41.05, p<0.0001, η^2=.23) and UPPS (Lack of) Perseverance scores (F(1,135)=17.41, p<0.0001, η^2=.11). Partial eta-squared estimates suggested medium to large effect sizes for these measures, ranging from 0.07 to 0.23.

3.4.2.2. Reward-delay impulsivity

Reward-delay impulsivity was assessed by computing the BAS Total (BAS-T) scores (as in Alloy et al., 2012). Participants with scores in the highest 15th percentile on the BAS-T (high BAS-T score cut point ≥ 45) were assigned to a high reward-delay impulsivity group (n=30). The low reward-delay impulsivity group (n=53) consisted of participants with moderate scores, i.e., 40th and 60th percentiles, on the BAS (cut points ≥ 38 and ≤ 41). Means and standard deviations of variables are presented in Table 3.4 and Table 3.5.

Variable	Low Reward-delay impulsivity group (*n*=53)	High Reward-delay impulsivity group (*n*=30)	F(1,80)	Partial Eta Squared
GNGT – number of commission errors	3.47(3.5)	5.07(4.2)	3.57	0.04
GNGT – Mean RT	359.10(53.8)	378.91(70.5)	2.44	0.03
SST – number of commission errors	13.68(9.5)	15.07(10.6)	0.33	0.00
SSRT	260.90(174.1)	279.30(165.9)	0.34	0.00
DDT	0.53(0.1)	0.60(0.1)	4.42*	0.05

Table 3.4. MANCOVA results for behavioural measures for *Reward-delay impulsivity group*: Presented are raw means (standard deviations), F-statistics for each univariate ANOVAs with their corresponding p-values and partial eta-squared estimates. For the DDT, the proportion of small-immediate/large-delayed reward choices is shown. GNGT, Go/No-Go Task; RT, reaction time; SST, Stop-Signal Task; SSRT, Stop-Signal reaction time; DDT, Delay Discounting Task. *p<0.05, **p<0.01.

A MANCOVA was performed to contrast the low reward-delay impulsivity group and the high reward-delay impulsivity group on all cognitive measures' variables (see Table 3.4), controlling for mental health history. The overall MANCOVA showed that the groups divided by their level of reward-delay impulsivity were significantly different (Wilks λ=0.87, F(5,76)=2.36, p=0.048, η^2=.13). Univariate ANOVAs were conducted to test a priori hypotheses, using Bonferroni correction as implemented in SPSS, which revealed significant differences between groups for the proportion of smaller vs. larger reward choices in the DDT, with a close to medium effect size estimate (F(1,80)=4.42, p=0.039, η^2=.05).

A MANCOVA was performed to contrast the low reward-delay impulsivity group and the high reward-delay impulsivity group on all self-report measures' variables (see Table 3.5), controlling for mental health history. The overall MANCOVA showed that the groups divided by their level of reward-delay impulsivity were significantly different (Wilks λ=0.55,

F(9,72)=6.44, p<0.0001, η^2=.45). Univariate ANOVAs revealed significant differences between groups for BIS-11 Total scores (F(1,80)=9.08, p=0.003, η^2=.10), BIS-11 Non-Planning scores (F(1,80)=4.39, p=0.039, η^2=.05), BIS-11 Motor scores (F(1,80)=16.97, p<0.0001, η^2=.18), UPPS (Lack of) Perseverance scores (F(1,80)=5.03, p=0.028, η^2=.06) and UPPS Sensation seeking scores (F(1,80)=11.32, p=0.001, η^2=.12). Partial eta-squared estimates suggested medium to large effect sizes for these measures, ranging from 0.05 to 0.18.

Variable	Low Reward-delay impulsivity group (*n*=53)	High Reward-delay impulsivity group (*n*=30)	F(1,80)	Partial Eta Squared
BIS-11 Total	61.46(11.6)	69.42(12.5)	9.08**	0.10
BIS-11 Non-Planning	22.25(4.8)	24.64(5.4)	4.39*	0.05
BIS-11 Attention	18.51(4.7)	19.25(4.1)	0.86	0.01
BIS-11 Motor	21.33(4.9)	25.83(4.7)	16.97**	0.18
EPQ Psychoticism	2.17(1.5)	2.83(1.7)	3.58	0.04
UPPS Urgency	29.19(6.7)	31.17(6.8)	2.24	0.03
UPPS (Lack of) Premeditation	20.42(4.4)	20.80(6.0)	0.21	0.03
UPPS (Lack of) Perseverance	20.89(5.1)	18.27(4.6)	5.03*	0.06
UPPS Sensation Seeking	31.32(6.9)	36.90(7.7)	11.32**	0.12

Table 3.5. MANCOVA results for psychometric measures for *Reward-delay impulsivity group*: Presented are raw means (standard deviations), F-statistics for each univariate ANOVAs with their corresponding p-values and partial eta-squared estimates. BIS-11, Barratt Impulsiveness Questionnaire 11; EPQ, Eysenck Personality Questionnaire; UPPS, Urgency, Premeditation Perseverance, Sensation (Questionnaire). *p<0.05, **p<0.01.

3.4.2.3. Rapid-response impulsivity and reward-delay impulsivity combined group

Here, groups divided by their level of rapid-response impulsivity and reward-delay impulsivity were combined into either a single high-impulsivity group or a single low-impulsivity group,

using the combined scores of the BIS-11 Motor and BAS-Total scales. The high-impulsivity group (n=23) consisted of participants scoring high on both scales (scores within the highest 35th percentile of the BIS-11 Motor subscale in the whole sample and the highest 15th percentile of BAS-Total scale). The low-impulsivity group (n=22) included participants scoring low on the BIS-11 Motor subscale (lowest 35th percentile) and moderately on the BAS-Total scale (between the 40th and 60th percentiles). Means and standard deviations of variables are presented in Table 3.6.

Variable	Low impulsivity group (*n*=22)	High impulsivity group (*n*=23)	F(1,42)	Partial Eta Squared
GNGT – number of commission errors	2.50(2.3)	5.17(4.5)	7.50**	0.15
GNGT – Mean RT	357.01(51.5)	388.19(76.6)	2.74	0.6
SST – number of commission errors	9.73(7.1)	15.91(10.3)	5.95*	0.12
SSRT	307.99(173.7)	279.86(147.4)	0.38	0.01
DDT	0.49(0.2)	0.62(0.1)	8.76**	0.17

Table 3.6. MANCOVA results for behavioural measures for *Rapid-response impulsivity and reward-delay impulsivity combined group*: Presented are raw means (standard deviations), F-statistics for each univariate ANOVAs with their corresponding p-values and partial eta-squared estimates. For the DDT, the proportion of small-immediate/large-delayed reward choices is shown. GNGT, Go/No-Go Task; RT, reaction time; SST, Stop-Signal Task; SSRT, Stop-Signal reaction time; DDT, Delay Discounting Task. *p<0.05, **p<0.01.

A MANCOVA was performed to contrast the two groups on all the cognitive measures of impulsivity (see Table 3.6), controlling for mental health history. The overall MANCOVA showed that the groups were significantly different (Wilks λ=0.64, F(5,38)=4.28 p=0.003, η^2=.36). Univariate ANOVAs revealed significant differences between groups for GNGT raw number of commission errors (F(1,42)=7.50, p=0.009, η^2=.15), SST raw number of commission errors (F(1,42)=5.95, p=0.019, η^2=.12) and the proportion of smaller vs. larger reward choices in the DDT (F(1,42)=8.76, p=0.005, η^2=.17). Partial eta-squared estimates suggested large effect sizes for these measures, ranging from 0.13 to 0.17.

3.5. DISCUSSION

In this study, different cognitive and self-report measures of impulsivity were compared within a sample of undergraduate students. How sensitive these measures are to detect differences between groups classified as being low in impulsivity and high in impulsivity was examined, based on two factors: level of rapid-response impulsivity (Swann et al., 2009) and reward-delay impulsivity (Alloy et al., 2006). The results comparing groups via general linear model analyses partly supported our three hypotheses: (1) The group with elevated rapid-response impulsivity had significantly higher scores on self-reports measuring reward-delay impulsivity than the low rapid-response impulsivity group. The group with elevated reward-delay impulsivity had significantly higher scores on self-reports measuring rapid-response impulsivity than the low reward-delay impulsivity group. (2) The high-impulsivity group, as defined by high scores on rapid-response impulsivity and reward-delay impulsivity self-report measures, showed significantly reduced task performance on the GNGT and SST and, on the DDT, preferred small but immediate rewards over larger, delayed rewards significantly more often than the low-impulsivity group. (3) Correlations between the cognitive tasks and self-report measures were also examined, since mostly contradictory results have been reported to date (Aichert et al., 2012; Cheung et al., 2004; Enticott et al., 2006; Horn et al., 2003; Keilp et al., 2005; Lijffijt et al., 2005; Malesza & Ostaszewski, 2016; Reynolds et al., 2006; Rodriguez-Fornells et al., 2002; Spinella, 2004). The aim was to clarify which aspects of trait impulsivity are related to response inhibition and which to reward responsiveness and hypothesised that the experimental tasks would significantly correlate with questionnaires measuring different aspects of impulsivity. This was met for the DDT, as there were significant positive correlations with the BIS-11 Total scale and the BIS-11 Non-Planning subscale. However, the rapid-response impulsivity tasks (GNGT, SST) did not directly correlate with any of the self-report measures. These results will be discussed in more detail in the following sections organised by impulsivity factors.

3.5.1. Rapid-response impulsivity

The BIS-11 is one of the most widely used measures of trait impulsivity, and the Motor subscale assesses the tendency to act on the spur of the moment. This sub-scale examines the lack of inhibitory control observed in rapid-response impulsivity, the impulsivity dimension of interest here. In the current study, high rapid-response impulsivity group, as identified by their level of rapid-response impulsivity measured by the BIS-11 Motor subscale, showed significantly higher scores in all reward-delay impulsivity measures:

(1) Behaviourally, on the DDT, participants in high rapid-response impulsivity group preferred small but immediate rewards over larger, delayed rewards significantly more often than low rapid-response impulsivity group.

(2) On the BAS Total score, which measures BAS sensitivity towards rewards.

(3) On the BAS Fun Seeking subscale, which measures willingness to approach new and potentially rewarding experiences, and which is the only BAS subscale that overlaps with impulsivity (Alloy et al., 2009).

(4) On the UPPS (Lack of) Premeditation subscale, which measures the inability to reflect on the consequences of one's actions before engaging in them, and which has been related to reward-delay impulsivity (Lynam & Miller, 2004).

Participants in the high rapid-response impulsivity group also had significantly higher scores on other motor impulsivity measures, such as the UPPS Urgency subscale, on measures theoretically related to rapid-response impulsivity, like the UPPS (Lack of) Perseverance subscale, and on trait impulsivity measures like the EPQ Psychoticism subscale. Contrary to our expectations, no significant differences were observed between participants in the high- and low-rapid-response impulsivity group on the GNGT and the SST tasks, which was surprising. Although high scores on the BIS-11 have been reported to be correlated with reduced performance in the GNGT (e.g., Enticott et al., 2006; Keilp et al., 2005; Reynolds et al., 2006), other studies have reported that healthy individuals scoring high on self-report measures of impulsivity do not show impaired performance in response inhibition paradigms (Aichert et al., 2012; Dimoska & Johnstone, 2007; Fallgatter & Herrmann, 2001; Horn et al., 2003; Lansbergen et al., 2007; Wilbertz et al., 2014). This could be the case here too, as behavioural approaches have been suggested to measure task performance during a limited and at an exact moment in time, while self-report measures might focus on self-reported trait impulsivity, manifested across time and different situations (Cyders & Coskunpinar, 2011; Lane et al., 2003; Reynolds et al., 2006).

The associations between response inhibition paradigms and rapid-response impulsivity questionnaires were also examined. Contrary to our predictions, neither the GNGT nor the SST were significantly associated with self-report measures assessing rapid-response impulsivity. Although previous studies have reported significant correlations between the GNGT and the BIS-11 (e.g., Enticott et al., 2006; Keilp et al., 2005; Reynolds et al., 2006; Spinella, 2004), others did not report such associations (e.g., Horn et al., 2003; Kulendran et al., 2016). Similarly, the SSRT, the main measure of the SST, has been shown to significantly correlate with the UPPS Sensation Seeking subscale (Aichert et al., 2012) and with the EPQ (e.g., Logan et al., 1997; Marsh et al., 2002). However, other studies have not reported such

relationship (e.g., Cheung et al., 2004; Enticott et al., 2006; Keilp et al., 2005; Lijffijt et al., 2005; Reynolds et al., 2006; Rodriguez-Fornells et al., 2002). It is possible that the fusion, or amalgamation of the different concepts of impulsivity has resulted in such inconsistencies (Cyders & Coskunpinar, 2011).

Taken together, our findings provide some evidence for a relationship between rapid-response and reward-delay impulsivity. Participants in the high rapid-response impulsivity group as measured by scores on the BIS-11 Motor subscale, showed significantly higher reward-delay impulsivity, both on the cognitive task, i.e., the DDT and self-report measures assessing reward sensitivity, i.e., the BIS/BAS. These findings are associated with medium to large effect sizes, despite modest sample sizes thus suggesting some robustness to the observations. No associations between self-report measures and cognitive tasks measuring rapid-response impulsivity were observed. Our results, therefore, suggest that the BIS-11 Motor subscale might not be assessing rapid-response impulsivity in the same way as the cognitive tasks assessing response inhibition. This may highlight the fact that self-report measures expected to specifically assess motor impulsivity (e.g., BIS-11 motor subscale, UPPS Urgency) might not directly be linked to the cognitive effects of rapid-response impulsivity. This finding is in line with other studies stating a lack of associations between cognitive tasks assessing impulsivity and trait measures of impulsiveness (e.g., Kulendran et al., 2016). As mentioned previously, task-based measures of impulsivity assess concepts like response inhibition at an exact moment in time and for a very short duration, while self-report measures focus more on trait impulsivity, expressed over time and different situations (Cyders & Coskunpinar, 2011; Lane et al., 2003; Reynolds et al., 2006). A lack of, or minimal correlations, between cognitive task of impulsivity and self-report measures have previously been argued to point towards the fact that these assessments may measure different aspects of impulsivity (e.g., Reynolds, et al., 2006; Lane et al., 2003) or that this information is collected in different ways, and may be an assessment-related confound. Here, however, both self-report measures and cognitive tasks were conducted on a computer.

Furthermore, although Wilbertz et al., (2014) used BIS-11 Total scores to sub-divide their sample, they subsequently showed that the UPPS Urgency subscale, which measures “the tendency to engage in impulsive behaviours under conditions of negative affect” (Whiteside et al., 2005; p. 561) better explains individual variability in relation to RI performance. Although the UPPS Urgency subscale is conceptually similar to the BIS-11 Motor subscale, except for its focus on negative affect, it seems it may also be more sensitive to behavioural effects of rapid-response impulsivity.

3.5.2. Reward-delay impulsivity

The BIS/BAS scales, specifically the BAS subscales, were used to classify participants by their level of reward-delay impulsivity (in line with Alloy et al., 2009). Participants scoring high on the BAS subscales (high reward-delay impulsivity group) to those with moderate scores (low reward-delay impulsivity group) were compared on the same subscales. Because low BAS has previously been linked with unipolar depression (Depue & Iacono, 1989; Fowles, 1988; Kasch et al., 2002), the moderate BAS group was chosen for comparisons with the high BAS group. The reason for not using the specific BAS Reward Responsiveness subscale was based on a previous study which related this subscale to the responsiveness to already obtained rewards (Alloy et al., 2009), not to the expectation of receiving a reward. The high reward-delay impulsivity group showed significantly higher scores on the BIS-11 Motor subscale. Although no significant differences were found on the GNGT and SST, results on the BIS-11 Motor subscale suggest that this group also exhibits rapid-response impulsivity characteristics.

The high reward-delay impulsivity group also had significantly higher scores on measures related to trait impulsivity, such as the UPPS Sensation Seeking subscale. Consistent with previous findings (e.g., Alloy et al., 2006; 2009), our results indicate that participants with high reward-delay impulsivity show higher trait impulsivity AND higher rapid-response impulsivity, as measured by self-report measures. Likewise, those with high rapid-response impulsivity scored significantly higher on reward-delay impulsivity measures. These results, along with the significant correlations observed between the BIS-11 and all three BAS subscales, and between the DDT and the BIS-11 Total and BIS-11 Non-Planning subscale, suggest that rapid-response and reward-delay impulsivity are closely related to each other.

Importantly, the high reward-delay impulsivity group not only showed significantly higher trait impulsivity compared to the low reward-delay impulsivity group, but also performed significantly different on the reward-delay impulsivity task, the DDT. This, together with the medium to large effect size reported for this sample further validates the construct of reward-delay impulsivity as being a crucial characteristic of trait impulsivity. Previous studies using the BIS/BAS scales comparing task performance of patients and healthy individuals have reported significant differences for risk-taking (Black et al., 2014) or reward tasks, such as the card-sorting task (Hayden et al., 2014), while others reported non-significant results when using paradigms assessing reward responsiveness (Alloy et al., 2012).

3.5.3. Rapid-response and reward-delay impulsivity combined

When combining both, the rapid-response impulsivity groups with the reward-delay impulsivity groups (see methods for detail), the group scoring high on both impulsivity dimensions and the group scoring low on both impulsivity dimensions, showed characteristics that were rather different to those of 'single' low vs. high-impulsive groups. The combined high rapid-response impulsivity and reward-delay impulsivity group showed significantly reduced task performance than the combined low rapid-response impulsivity and reward-delay impulsivity group on the two tasks measuring rapid-response impulsivity; they also preferred small but immediate rewards over larger, delayed rewards significantly more often. The latter indicates a more pronounced reward-delay impulsivity than that for the low rapid-response impulsivity and reward-delay impulsivity group.

While differences between high- and low-impulsivity groups are less clear when individuals are categorised based on either impulsivity dimension, differences between high- and low-impulsivity groups are more specific, and pronounced, when combining both dimensions. Although previous papers have not reported impaired performance in healthy individuals scoring high in trait impulsivity (e.g., Aichert et al., 2012; Wilbertz et al., 2014), our results suggest that self-report measures of rapid response impulsivity alone may not be sensitive enough to pick up the differences between groups in response inhibition paradigms (as seen in the current study, when using the BIS-11 Motor scores alone). However, when including a related, and perhaps necessary, dimension, namely that of reward-delay impulsivity, differences between groups were observed. However, the opposite could therefore also be true, i.e., our results also show that self-report measures of reward-delay impulsivity alone may not be sensitive enough to pick up the differences in response inhibition paradigms. However, when including a related, and perhaps necessary, dimension, namely that of rapid-response impulsivity, differences between groups were observed. These findings should be taken with caution, however, as the number of individuals in this combined impulsivity group is quite low, as only about 13.8% of participants scored high on both dimensions of impulsivity.

Additional analyses were therefore conducted to assess whether the differences in results between combined impulsivity groups and single impulsivity groups were affected by the difference in participant numbers. This would indicate whether the observed effects were due to comparing the very extreme ends of impulsivity or were truly related to the combination of the two impulsivity dimensions. To test this, separate MANCOVAs were performed for rapid-response and reward-delay impulsivity including only the top 22 low- and the top 23 high-scorers, i.e., the same number of individuals in each group as for the combined impulsivity group analysis. MANCOVAs showed that groups were not significantly different when

classified by level of reward-delay impulsivity (Wilks λ=0.86, F(5,38)=1.28, p>0.05) or by level of rapid-response impulsivity (Wilks λ=0.90, F(5,38)=0.87, p>0.05). These analyses showed no significant differences between groups in any of the three cognitive tasks. Therefore, it is the combination of the two dimensions of impulsivity that provides a more sensitive assessment.

3.6. CONCLUSION

In summary, this study compared different measures of impulsivity, to examine how sensitive these measures are to differences between low and high impulsivity groups, based on two impulsivity dimensions: rapid-response impulsivity and reward-delay impulsivity. Results show that the proposed measures were sensitive to differences between groups. Participants with higher impulsivity, as measured by high rapid-response impulsivity scores on the BIS-11 Motor subscale, show significantly increased trait impulsivity as well as behavioural and self-reported reward-delay impulsivity. Conversely, the high reward-delay impulsivity group had significantly higher trait impulsivity and behavioural and self-reported rapid-response impulsivity. When both dimensions of impulsivity were combined, the high-impulsivity group showed significantly reduced performance on both response inhibition paradigms (GNG and SST) and temporally discounted in a significantly more impulsive manner in the reward-delay task than the low-risk group. These findings provide evidence that combining impulsivity dimensions provides a better predictor of impulsivity level than each dimension alone.

CHAPTER 4: Towards a unified conceptualisation of impulsivity

4.1. CHAPTER AIMS

Although the multi-dimensional construct of impulsivity is generally accepted, agreement on how many dimensions it consists of is lacking. An exploratory factor analysis was conducted, which included widely used measures of impulsivity but also sensation-seeking and reward-delay sensitivity. It was hypothesised that a three-factor model of impulsivity would benefit from including measures of reward-delay impulsivity, and yet, be differentiated into three latent constructs. Healthy undergraduate students completed self-report questionnaires and experimental tasks assessing trait impulsivity, rapid response impulsivity and reward-delay impulsivity. Common factor analysis was conducted and included all the variables tested in Chapter 3. The resulting model and its relationships were tested using structural equation modelling (SEM). Here, results from these analyses are presented and discussed.

4.2. INTRODUCTION

Impulsivity is defined as a "predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences" (Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001; p. 1784). Although it is widely accepted that impulsivity is a multi-dimensional construct (Alloy et al., 2009; Dawe & Loxton, 2004; Dawe, Gullo, & Luxton, 2004; MacKillop et al., 2016; Swann, 2010) there is no general agreement about how many dimensions it consists of (Malesza & Ostaszewski, 2016), nor how it should be conceptualised. Instead, the concept of impulsivity adopted by different researchers seems to depend upon their personal theoretical perspective (some influenced by personality theories and others not) and preferred methods of study (behavioural tasks or self-report questionnaires). Inevitably, this has led to terminological confusion and an ongoing debate about what impulsivity really is (Gullo, Loxton, & Dawe, 2014; Hamilton et al., 2015).

In the 1970's, Eysenck and Eysenck attempted to incorporate impulsivity within the Big Three theory of personality (psychoticism, extroversion, neuroticism) with impulsivity included as a subscale of extroversion (Eysenck, 1967; Whiteside & Lynam, 2001). Gray (1987) proposed that impulsivity was a personality trait that was associated with sensitivity to positive reinforcement and conceived of it as a rotation of Eysenck's extroversion and neuroticism scales such that high impulsivity individuals were high on both extroversion and neuroticism. Some years later, Cloninger et al. (1991) proposed a model of personality in which impulsivity was included as part of Novelty Seeking (Whiteside & Lynam, 2001). Along the same line, Zuckerman, Kuhlman, Thornquist, & Kiers (1991) proposed the Alternative Big Five, a five-factor model of personality as replacement for Eysenck's Big Three, in which impulsivity was included as a component labelled Impulsive-Sensation Seeking (Zuckerman & Glicksohn,

2016). None of these attempts to describe impulsivity within established theories of personality have gained widespread acceptance (Whiteside & Lynam, 2001). However, it led other researchers (e.g., Barratt, 1993; Dickman, 1990) to adopt an approach in which impulsivity is conceptualised as a construct in its own right, comprising several separate lower-order traits (Whiteside, Lynam, Miller, & Reynolds, 2005).

Factor Analytical Studies of self-report measures of impulsivity

One of the most widely used self-report measures of impulsivity, the Barratt Impulsiveness Scale (BIS-11, Patton, Stanford, & Barratt, 1995), was designed based on a principal component analysis (PCA) and revealed three lower order traits: Motor Impulsiveness, Non-Planning Impulsiveness and Attentional Impulsiveness. Although the BIS-11 is a widely used scale to measure impulsivity, Whiteside and Lynam (2001) suggested that the BIS-11, and other similar self-report impulsivity measures, might not be sensitive enough to assess the different aspects of impulsivity. Whiteside and Lynam (2001) therefore, designed an impulsivity measure based on the well-established Five-Factor Model of personality (McCrae & Costa 1990). They conducted an exploratory factor analysis on nine frequently used self-report measures of impulsivity: EASI-III (Emotionality, activity, sociability, and impulsivity) Impulsivity Scales (Buss & Plomin, 1975), Dickman's Functional and Dysfunctional Impulsivity Scales (Dickman, 1990), BIS-11 (Patton, Stanford, & Barratt, 1995), I-7 Impulsiveness Scale (Eysenck, Pearson, Easting, & Allsopp, 1985), Personality Research Form Impulsivity Scale (Jackson, 1967), Multidimensional Personality Questionnaire Control Scale (Tellegen, 1985), Temperament and Character Inventory (Cloninger et al., 1991), Sensation Seeking Scale (Zuckerman 1994), and the Revised NEO Personality Inventory (NEO-PI-R; Costa & McCrae, 1992). This exploratory factor analysis identified the four factors which now constitute the UPPS Impulsivity Scale: Urgency, (lack of) Premeditation, (lack of) Perseverance, and Sensation Seeking. The authors suggested that these four factors reflect four different personality traits which lead to analogous behaviours (Whiteside & Lynam, 2001).

The relationship between self-report measures of impulsivity and reward-delay impulsivity

Although there are multiple psychometric measures of impulsivity, there are rather fewer measures of reward sensitivity. Carver and White (1994) designed the Behavioural Inhibition System/Behavioural Activation System (BIS/BAS) scales to measure reward sensitivity (Dawe & Loxton, 2004), based on the reinforcement sensitivity theory of personality (Gray & McNaughton, 2000). PCA of the BIS/BAS scale revealed four components: Behavioural Inhibition, Reward Responsiveness, Drive, and Fun Seeking (Carver & White, 1994). Subsequent factor analyses on this scale revealed that the BAS Fun Seeking subscale loads

on both reward-delay and rapid-response impulsivity factors and is associated with rapid-response impulsivity more than with reward-delay impulsivity (Caseras et al., 2003; Zelenski & Larsen, 1999).

Subsequent factor analyses have confirmed that self-report measures of impulsivity, for example, the BIS-11 (Patton, Stanford, & Barratt, 1995), the Eysenck Impulsiveness Scale (Eysenck & Eysenck, 1991), the Novelty Seeking Scale (Cloninger, Przybeck, Svrakic, & Wetzel, 1994), and the Sensation-Seeking Scale (Zuckerman, 1994) correlate with each other and load on the same factor. Measures of reward sensitivity, such as the BIS/BAS Scales (Carver & White, 1994) and the Sensitivity to Reward scale (Torrubia et al., 2001), load onto a different factor. However, both factors tend to correlate with each other (Alloy et al., 2009).

Factor analyses of behavioural measures of impulsivity

The results of factor analytical studies using only behavioural measures have been consistent with those investigating the relationship between impulsivity and reward sensitivity using self-report measures. This provides evidence that these two constructs are different, yet theoretically related. Dougherty, et al. (2003) conducted a confirmatory factor analysis (CFA) in which the categorisation of experimental tasks corresponded to this theoretical division: one factor was labelled “rapid-decision” and included the Stop-Signal Task (SST) and Immediate and Delayed Memory tasks. A second factor was labelled “reward-directed” and included a Two-Choice Delayed Reward task and a Single Key Impulsivity Paradigm.

Using PCA, Reynolds and colleagues (2006) identified two dimensions of impulsivity. First, the dimension of “impulsive disinhibition’’ characterised by performance measures of a Go/No-Go task (GNGT) and an SST, both measuring response inhibition. A second dimension was ‘‘impulsive decision-making’’ which includes performance measures of a Delay-Discounting task, which assesses the inability to delay gratification, and of a Balloon Analog Risk Task, which measures risk-taking behaviours. These observations together with findings assessing self-report measures are consistent with previous reports (e.g., Alloy et al., 2009; Dawe & Loxton, 2004; Dawe et al., 2004; MacKillop et al., 2016; Swann, 2010), emphasising that impulsivity comprises more than one dimension. Frequently reported dimensions include rapid response impulsivity and reward delay impulsivity. Rapid response impulsivity is defined as the ability to fully evaluate a stimulus before responding and is measured by the BIS-11 Motor subscale, the UPPS Urgency subscale, the GNGT or the SST. Reward-delay impulsivity, or reward sensitivity, is defined as the inability to delay gratification, and is assessed by the BIS/BAS scales, the Sensitivity to Reward scale or delay discounting tasks.

Factor analyses on combined self-report and behavioural measures of impulsivity

Several studies have conducted confirmatory factor analyses (CFA), on both self-report measures and behavioural tasks combined, to investigate if the same results can be obtained as when self-reports or behavioural tasks are assessed separately. CFA also aims to determine if these two types of measures assess the same construct. Aichert and colleagues (2012), for example, tested whether performance on response inhibition tasks (SST, GNGT, anti-saccade task, Stroop task) fall into the same construct, labelled "prepotent response inhibition". They reported that all four tasks contributed significantly to their model. They subsequently used Structure Equation Modeling (SEM) to determine if a 'trait impulsivity' construct, as measured by the BIS-11 sum score, is causally associated to the 'prepotent response inhibition' construct, which was comprised of response inhibition tasks. They observed that higher levels of trait impulsivity predicted reduced task performance. However, although their model was well-fitted statistically, trait impulsivity only explained 12% of variance of the "prepotent response inhibition" construct (Aichert et al., 2012).

Cyders and Coskunpinar (2012) conducted an exploratory principal axis factoring factor analysis where they included seven widely used psychometric and behavioural measures to assess impulsivity, reward-delay impulsivity, response inhibition and sensation seeking. Their results suggested a 7-factor solution: (1) Self-report measures; (2) Prepotent response inhibition; (3) Delay responding with prepotent response inhibition cross-loading; (4) Distortions in elapsed time with delay responding cross-loading; (5) Resistance to proactive interference; (6) Delay responding and sensation seeking cross-loading; (7) Resistance to distractor interference and sensation seeking cross-loading (Cyders & Coskunpinar, 2012). They conclude that these seven different factors represent different constructs of impulsivity.

MacKillop and colleagues (2016) conducted CFA and SEM on several psychometric (BIS-11, UPPS, Monetary Choice Questionnaire (MCQ)) and behavioural measures of impulsivity, including response inhibition (GNGT, SST, continuous performance task (CPT)) and a delay-discounting tasks (DDT). They report three latent constructs: impulsive choice (DDT/MCQ), impulsive action (GNG, CPT, SST) and impulsive personality traits (BIS-11, UPPS). Their results suggest that, on the one hand, response inhibition tasks and questionnaires assessing impulsive personality traits were significantly correlated. This is not consistently reported in previous studies. On the other hand, associations between impulsive choice and impulsive action were non-significant, although theoretically they should be related. It is possible that the second factor (impulsive action) might not have been represented well enough to show a significant association with impulsive choice. This might be because reward sensitivity measures, i.e., BIS/BAS scales, or tasks measuring risk-taking, e.g., Balloon Analog Risk

Task, were not assessed by MacKillop et al. (2016). This means that the impulsive choice construct was only operationalised by reward-directed tasks, such as the DDT and the MCQ.

Current study

Previous studies have used factor analysis and SEM on both self-report and behavioural measures of impulsivity and have provided well fitted models. Most studies have included laboratory tasks which assess reward sensitivity but did not include specific psychometric measures of reward sensitivity (e.g., Cyders & Coskunpinar, 2012; MacKillop et al., 2016).

Here, an exploratory factor analysis is proposed which, in addition to the most widely used measures of impulsivity, also includes venturesomeness-related scales, such as sensation-seeking, and measures of reward-delay sensitivity, such as the BAS subscale of the BIS/BAS scale. Specific measures are: (1) behavioural tasks measuring rapid-response impulsivity, i.e. GNGT and SST; (2) the BIS-11, measuring motor, attention and non-planning impulsivity; (3) the UPPS Impulsive scale, measuring motor impulsivity, lack of perseverance, lack of premeditation and sensation-seeking; (4) Psychoticism and Extroversion subscales from the EPQ, measuring Impulsivity and Venturesomeness, respectively; (5) Delay-Discounting Task, measuring the inability to delay gratification; and (6) the BAS Total score of the BIS/BAS scale, measuring reward sensitivity.

The aim of this study was to test the hypothesis that a three-factor model of impulsivity as reported previously (MacKillop et al., 2016), would benefit from the inclusion of a specific psychometric measure of reward-delay impulsivity, and yet, be differentiated into three latent constructs. To test this hypothesis, a principal axis factoring analysis was conducted. This is the most appropriate method when the main objective is to examine the structure of the data, as it identifies the least number of factors that can explain common variance. The resulting model and its set of relationships were then tested using SEM, with structural path analyses examining the associations between identified factors.

4.3. METHODS

4.3.1. Participants

See section 2.3.2 for details.

4.3.2. Behavioural tasks

See sections 2.5.1, 2.5.2 and 2.5.4 for details.

4.3.3. Self-report measures

See section 2.6 for details.

4.3.4. Data analysis

See section 2.7.2 for details.

4.4. RESULTS

4.4.1. Exploratory Factor Analysis (EFA)

15 variables were submitted to an EFA with robust unweighted least squares (RULS) estimation and promin rotation (Lorenzo-Seva, 1999). Eigenvalues and optimal implementation of parallel analysis (Timmerman & Lorenzo-Seva, 2011) supported a 3-factor solution (Table 4.1). A 2-factor model also seemed reasonable, with factor one comprising psychometric measures of impulsivity and factor two including the behavioural tasks.

In the 3-factor solution, factor one represented the self-report measures of rapid-response impulsivity: BIS-11 Non-Planning, BIS-11 Attention, BIS-11 Motor, UPPS Urgency, UPPS (lack of) Premeditation, UPPS (lack of) Perseverance, EPQ Psychoticism, and DDT proportion of smaller vs. larger reward choices. The DDT, however, showed a weak loading and additionally cross-loaded on factor 3. Factor two comprised the behavioural measures of rapid-response impulsivity: GNGT proportion of commission errors, SST reaction time and SST proportion of commission errors. Factor three comprised psychometric and behavioural measures of reward-delay impulsivity along with sensation seeking: UPPS Sensation Seeking, BAS Total scores, EPQ Extroversion, and DDT proportion of smaller vs. larger reward choices. As before, the DDT showed a weak loading and cross-loaded also on factor 1.

4.4.2. Confirmatory Factor Analysis

Including all variables of impulsivity

The exploratory factor analysis conducted previously favoured a three-factor model. However, a two-factor model also seemed plausible, with factor one comprising psychometric measures of impulsivity and factor two containing the behavioural tasks. Thus, latent structural solutions were tested hierarchically. A one-factor, a two-factor and a three-factor model including all the variables analysed in the exploratory factor analysis were tested using maximum likelihood estimates. Table 4.2 summarises the results of these analyses. Because these are non-nested models, a p-value for the difference cannot be obtained. Instead, standard fit indices, specifically RMSEA, CFI and TLI were used to interpret, in a descriptive manner, which model might fit best.

	Factor 1 (Eigenvalue = 4.28; Variance explained: 33.3%)	**Factor 2** (Eigenvalue = 1.76; Variance explained: 12.4%)	**Factor 3** (Eigenvalue = 1.45; Variance explained: 11.5%)
GNGT Errors	0.14	**0.39**	0.01
SST RT	0.12	**-0.72**	0.09
SST Errors	0.08	**0.77**	0.07
DDT	**0.24**	0.05	0.18
BIS-11 Non-Planning	**0.87**	-0.02	-0.06
BIS-11 Attention	**0.64**	0.07	0.09
BIS-11 Motor	**0.63**	-0.03	0.25
UPPS Urgency	**0.65**	0.02	0.02
UPPS Premeditation	**0.76**	-0.04	-0.05
UPPS Perseverance	**0.74**	-0.04	-0.22
UPPS Sensation	0.08	0.11	**0.43**
BAS Total	-0.08	-0.06	**0.89**
EPQ Psychoticism	**0.33**	0.10	0.04
EPQ Extroversion	-0.05	-0.03	**0.48**

Table 4.1. Exploratory factor analysis of impulsivity variables.

A single latent factor of impulsivity was evaluated which included all the variables analysed in the exploratory factor analysis. This resulted in a poor model fit (Model 1a: C^2 (77) = 241.195, $p < 0.01$; RMSEA = 0.115; CFI = 0.689; TLI = 0.633). A two-factor model which included the same variables as the factor analysis was evaluated. Factor one included the psychometric measures of impulsivity and factor two contained the behavioural tasks. The two-factor model fit was better than the one-factor model (Model 2a: C^2 (76) = 136.715, $p < 0.01$; RMSEA = 0.071; CFI = 0.885; TLI = 0.862). Finally, a three-factor model with the same variables was tested. Factor one included the psychometric measures of rapid-response impulsivity, factor two measured reward-delay impulsivity and factor three consisted of the rapid-response impulsivity behavioural tasks. This three-factor model showed a further improvement in model

fit compared to the two-factor model (Model 3a: C^2 (74) = 112.394, p < 0.01; RMSEA = 0.057; CFI = 0.927; TLI = 0.911). However, although the RMSEA indicates a good model fit, the CFI and TLI indices are below the recommended value for this sample size (>0.95; Hair et al., 2010).

Including all variables except DDT proportion smaller vs. larger reward choices

As reported earlier, the DDT proportion of smaller vs. larger reward choices not only showed a weak loading but also cross-loaded on factors 1 (comprising self-report measures of rapid-response impulsivity) and 3 (comprising self-report measures of reward-delay impulsivity) in the factor analysis. I therefore tested whether the model fit would improve when this variable was removed (see results in Table 4.2).

Model	χ^2 (df)	χ^2 /df	RMSEA	CFI	TLI
Model 1a	241.2 (77) **	3.13	0.12	0.69	0.63
Model 1b	224.3 (65) **	3.45	0.12	0.69	0.63
Model 2a	136.7 (76) **	1.80	0.07	0.86	0.86
Model 2b	119.6 (64) **	1.87	0.07	0.89	0.87
Model 3a	112.4 (74) **	1.52	0.06	0.93	0.91
Model 3b	89.4 (62) *	1.44	0.05	0.95	0.93

Table 4.2. Structural model fit indices. χ^2=chi-square, df=degrees of freedom, RMSEA=root-mean-square error of approximation, CFI=confirmatory fit index, TLI=Tucker-Lewis index. *p < 0.05; **p < 0.01.

A one-factor model, which included the same variables as reported for the factor analysis except the DDT, was evaluated and resulted in a poor model fit (Model 1b: C^2 (65) = 224.276, p < 0.01; RMSEA = 0.124; CFI = 0.693; TLI = 0.631). Similarly, to what was reported before, a two-factor model with factor one including psychometric measures of impulsivity and factor two containing behavioural tasks without DDT, showed a better model fit (Model 2b: C^2 (64) = 119.577, p < 0.01; RMSEA = 0.074; CFI = 0.893; TLI = 0.869). Finally, a three-factor model was tested. Factor one included measures of impulsive action, factor two measured impulsive choice except DDT and factor three comprised RI behavioural tasks. The three-factor model showed an improvement in model fit compared to the two-factor model (Model 3b: C^2(62) = 89.442, p < 0.05; RMSEA = 0.053; CFI = 0.947; TLI = 0.933). It also improved relative to the three-factor model that included the DDT (see model fit indices for models 3a and 3b in Table

4.2). Furthermore, the RMSEA (0.053) and the CFI (0.947) indicate a good model fit. However, the TLI (0.933) remains slightly below the recommended threshold for this sample size (>0.95; Hair et al., 2010). Most variables' standardised loadings were significant and moderate to very large for this sample size. Exceptions were UPPS Sensation Seeking (0.34) and EPQ Psychoticism (0.31), which were below the recommended threshold for this sample size (>0.4; Hair et al., 2010). For factor loadings, see Figure 4.1. Associations between the latent domains comprising psychometric measures and measures of behavioural tasks were low (Reward-Delay Impulsivity and Behavioural Impulsivity, r = 0.03; Rapid-Response Impulsivity and Behavioural Impulsivity, r = 0.10). The association between the two latent domains comprising psychometric measures was higher (Reward-Delay Impulsivity and Rapid-Response Impulsivity, r = 0.24).

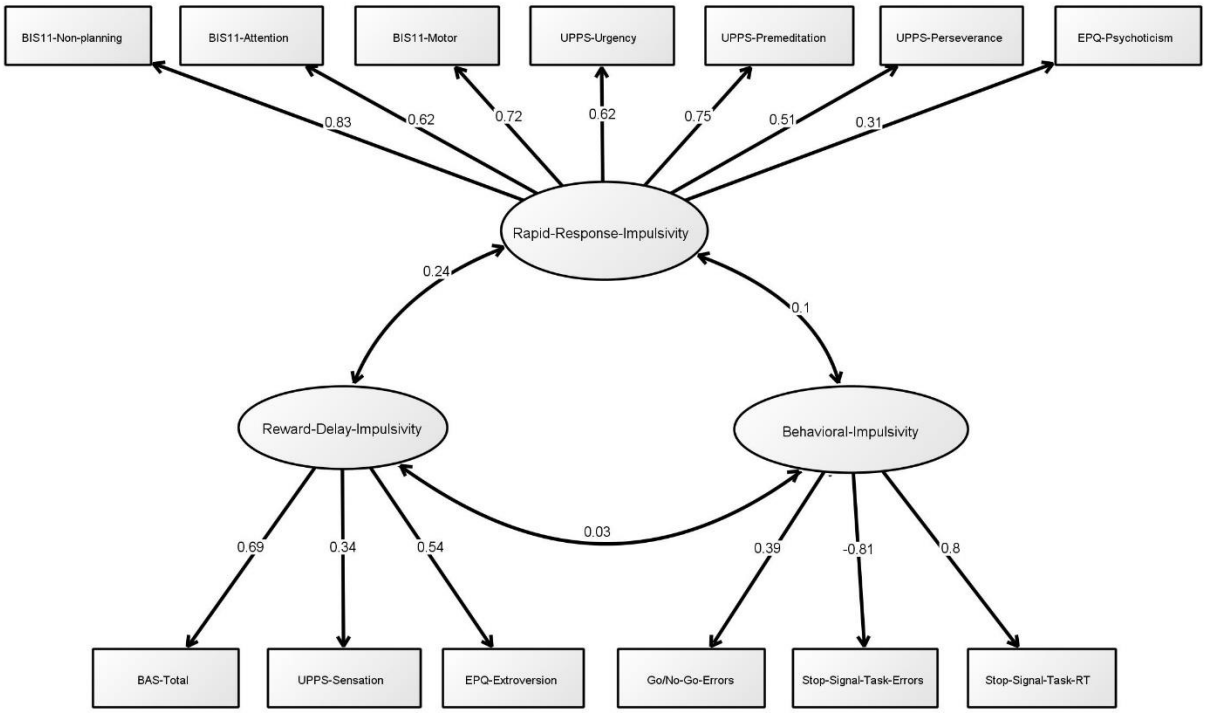

Figure 4.1. Structural model of the three latent constructs: behavioural impulsivity, reward-delay impulsivity and rapid-response impulsivity. Rectangles represent manifest indicator variables, and the ovals represent latent constructs. Single-headed arrows have the standardised factor loadings next to them and double-headed arrows show the correlations between the latent constructs.

4.5. DISCUSSION

The current study tested the hypothesis that a three-factor model of impulsivity consisting of impulsive action, impulsive choice and impulsive personality traits would benefit from the inclusion of a specific psychometric measure of reward-delay impulsivity, the BAS subscale from the BIS/BAS scales. This hypothesis was partly supported. Both, the exploratory factor analysis and the SEM approach supported a three-factor model. However, the structure of our model was different to a model proposed by MacKillop et al., (2016), and including a psychometric measure assessing reward sensitivity did not improve model fit.

The exploratory factor analysis and SEM approach supported a three-factor model comprising psychometric measures of rapid-response impulsivity, measures of reward-delay impulsivity and response inhibition behavioural tasks. Even though these SEM results can only be interpreted in a descriptive manner, the three-factor model showed a better model fit compared to one- and two-factor models. The rapid-response impulsivity latent domain assessed the ability to fully evaluate a stimulus before responding. It comprised three BIS-11 subscales (Non-Planning, Attention and Motor), three of the four UPPS subscales (Urgency, lack of Premeditation and lack of Perseverance) and the EPQ's Psychoticism subscale, which includes items measuring impulsivity. The reward-delay impulsivity latent domain assessed how sensitive an individual is to immediate reward. It includes the BAS Sensitivity subscale (as global measure of reward sensitivity), the BAS Reward Responsiveness, BAS Drive and BAS Fun-Seeking sum score from the BIS/BAS scales and the UPPS Sensation Seeking subscale. In addition, this domain includes items from the EPQ Extroversion subscale, which assesses Eysenck's Venturesomeness construct with questions on sensation seeking and risk-taking. The behavioural impulsivity latent domain included measures of response inhibition performance, i.e., proportion of commission errors of the Go/No-Go and Stop Signal tasks and the Stop-Signal Task's reaction time. The latent constructs and their organisation within this model are different to that from MacKillop and colleagues (2016) which is likely due to the inclusion of reward sensitivity and sensation seeking self-report measures.

Some prior studies assessing response inhibition only included commission errors of the GNGT and SST to measure task performance (e.g., Dougherty et al., 2003; MacKillop et al., 2016). Others only included SST reaction times (e.g., Reynolds et al., 2006), or GNGT commission errors and SST reaction times (Aichert et al., 2012). For example, Aichert et al. (2012) reported that SST reaction time had the weakest loading within the response inhibition latent domain. Interestingly, in our study, SST reaction time showed the largest loading of the three indicators which were included in the behavioural impulsivity latent construct. The differences observed between the GNGT and SST are consistent with other findings,

supporting a distinction between these two tasks. It had previously been suggested that these measures assess different processes, namely action restraint and action cancellation, respectively (Bari & Robbins, 2013; Eagle, Bari, & Robbins, 2008; Schachar et al., 2007; Swick, Ashley, & Turken, 2011).

In line with the findings reported here, previous factor analyses have demonstrated that a one-factor model, in which self-report and behavioural measures of impulsivity load on the same factor, shows a poor model fit (e.g., MacKillop et al., 2016; Smith et al., 2007). Consistent with the study by MacKillop and colleagues (2016) low associations between the latent construct of self-reports (rapid-response impulsivity and reward-delay impulsivity) and the latent construct of behavioural impulsivity were also observed. These findings appear to be in line with numerous other studies reporting inconsistent results of correspondence between behavioural response inhibition tasks and questionnaires in previous studies (e.g., Aichert et al., 2012; Enticott, Ogloff, & Bradshaw, 2006; Horn, Dolan, Elliott, Deakin, & Woodruff, 2003; Keilp, Sackeim, & Mann, 2006; Lijffijt, Lane, Moeller, Steinberg, & Swann, 2004; Malesza & Ostaszewski, 2016; Reynolds et al., 2006; Rodriguez-Fornells, Lorenzo-Seva, & Andrés-Pueyo, 2002; Spinella, 2004). Together, these findings strongly support the idea that impulsivity should not be measured as a single construct.

Importantly, these results corroborate what many researchers have emphasised before: (i) rapid-response impulsivity and reward-delay impulsivity are two separate dimensions which support the idea of a multifaceted construct of impulsivity (Alloy et al., 2009; Dawe & Loxton, 2004; Dawe et al., 2004; MacKillop et al., 2016; Swann, 2010); and (ii) self-report and behavioural measures do not correlate well, possibly because behavioural approaches measure task performance during a limited and at an exact moment in time, whereas questionnaires focus on self-reported trait impulsivity which may be manifested across time and in different situations (Cyders and Coskunpinar, 2011; Reynolds et al., 2006; Swann et al., 2010; Dougherty et al., 2014).

Contrary to our predictions, the exploratory factor analysis conducted here revealed the Delay-Discounting Task to cross-load on two factors, the rapid-response and the reward-delay impulsivity constructs. Furthermore, it showed a weak factor loading on both. The lack of significant loadings of the DDT on any of the factors tested here could be due to the very different nature of this measure. The DDT is neither similar to a typical prepotent response inhibition task, nor is it similar to a classic self-report measure assessing a behaviour across time and in different situations. Therefore, a three-factor model without this indicator was subsequently tested and showed an improved model fit.

Although most indicators' loadings were significant and moderate to very large for this sample size, the UPPS Sensation Seeking and EPQ Psychoticism indicators showed lower loadings than expected. The UPPS Sensation Seeking scale showed a low loading on the reward-delay impulsivity latent domain. However, the BAS Total scale and the EPQ Extroversion subscale, which also include measures of sensation seeking, load onto the same domain, suggesting a possible meaningful association. Furthermore, the EPQ Psychoticism subscale includes items that are not related to impulsivity, for example, aggression. This might explain the low loading on the rapid-response impulsivity latent construct.

4.6. LIMITATIONS

As in similar studies that include both behavioural tasks and self-reports, the different assessments needed for each of these measures can be problematic. Even though all necessary and recommended transformations were made, behavioural tasks assessing prepotent response inhibition, behavioural choices on the delay discounting measure and psychometric scores on the self-report measures, were completely different to each other. Overall, the outcomes of Chapters 3 and 4 show that the two dimensions of impulsivity investigated here are closely related and that both are necessary when assessing impulsivity. However, each of the two rapid-response impulsivity tasks was operationalised by two variables: RT and commission errors, while the DDT only included the proportion of immediate choices. In addition to this, the included self-reports mostly assessed the rapid-response aspect of impulsivity, which can be observed in the larger number of variables included in the Rapid-Response Impulsivity factor compared to the Reward-Delay Impulsivity factor. This is due to the reduced number of psychometric measures of reward-delay impulsivity available and it is possible that the reward-delay impulsivity aspect was underrepresented. As with the DDT, the specific BAS scales (Reward, Drive and Fun), which were designed to assess the inability to delay gratification, were not helpful and were only useful when included as a Total score. Therefore, it could be suggested that even though the objective was to improve previous models of impulsivity by including self-report measures of reward-delay impulsivity, this was not achieved by the lack of specification of current measures of reward-delay impulsivity.

Another limitation was the fact that I could not statistically compare the non-nested models used in this study. Accordingly, the models were compared in a descriptive manner, taking into consideration the theoretical consistency of each model. Standard fit indices were used to compare which model might fit best. However, using such approach requires that results be interpreted with caution. It is also noteworthy that the sample size used in this experiment

(n=167) was smaller than the one in MacKillop et al. (2016; n=1252), which might explain the differences in results observed.

4.7. CONCLUSIONS

The current study tested the hypothesis that a three-factor model of impulsivity, consisting of impulsive action, impulsive choice and impulsive personality traits, would benefit from the inclusion of a psychometric measure of reward-delay impulsivity. This hypothesis was partly supported. Results favoured a three-factor model but including a reward sensitivity psychometric measure did not improve the model fit. Furthermore, the model structure reported here was different to that proposed in previous studies (e.g., MacKillop et al., 2016). These findings strengthened what researchers have emphasised before, namely that rapid-response impulsivity and reward-delay impulsivity are two major yet different dimensions which contribute independently to the multifaceted nature of the impulsivity construct.

CHAPTER 5: MEG Experiment 1: Sensor- and source-level analyses of event-related fields and time-frequency representations in response inhibition.

5.1. CHAPTER AIMS

As seen in previous chapters, impulsivity can be examined using questionnaire measures, such as the BIS-11 and the UPPS Impulsiveness Scales, but also using behavioural laboratory tasks, which are designed to assess specific cognitive processes during a limited and exact moment in time (Cyders & Coskunpinar, 2011). Tasks are regarded as more objective measures of these behaviours than questionnaires, as they do not depend on the accurate recollection of one's behaviour, honest answers and introspection. Neuroimaging studies using behavioural tasks have provided much of the evidence found in the literature regarding the neural correlates associated with inhibitory processes (e.g., Aron & Poldrack, 2006; Falkenstein, Hoormann, & Hohnsbein, 1999; Huster et al., 2013; 2019; Johnstone et al., 2007; Kreusch et al., 2014; Luijten et al., 2014; Sehlmeyer et al., 2010; Simmonds et al., 2008). To better understand how response inhibition is processed in the brain, the most widely used tasks to measure this dimension of impulsivity along with its neural findings will be explained in the following sections.

In Chapter 3, the relationship between behavioural and self-report measures of impulsivity was investigated, by examining how sensitive behavioural measures of impulsivity are to differences between low and high impulsivity groups, as assessed by self-reports. This experiment demonstrated that the behavioural measures used are sensitive enough to pick up differences between these groups. However, the impulsivity paradigms were more sensitive to differences between groups defined by the combination of two impulsivity factors, rapid-response and reward-delay impulsivity, than groups of each dimension alone (Jauregi et al., 2018, see section 3.5). The impulsivity construct was further explored in Chapter 4, by conducting an exploratory factor analysis on all the impulsivity measures tested in Chapter 3, both behavioural and self-report measures. Results suggested that rapid-response impulsivity and reward-delay impulsivity are two major yet different dimensions which contribute independently to the multifaceted nature of the impulsivity construct, see section 4.5 for details.

Findings from the two analyses of the behavioural data show that the measures of impulsivity tested here are adequate to investigate impulsivity further. Considering the behavioural differences found between high and low impulsivity groups, the primary aim of the current chapter was to examine these behavioural differences in response inhibition using MEG. This neuroimaging method offers several advantages over other techniques such as fMRI, as it can measure brain activity directly (Gross, 2019), with a millisecond temporal resolution, allows tracking of real-time variations in cortical activity underlying the signal processing happening in the brain. The current MEG experiment was conducted in a healthy undergraduate student

population, divided by their level of impulsivity as measured by questionnaires, see section 2.4.4 for details. By investigating the macroscopic neural mechanisms of response inhibition (Chapter 5) and intertemporal choice (Chapter 6), a better understanding of the neurocognitive deficits observed in impulsive individuals might be achieved. The functional significance of oscillations is unknown in its entirety and thus, studies like the ones presented here, which compare the oscillatory activity in non-clinical subjects, might also aid in providing novel findings on the cognitive functionalities of these oscillations. In this Chapter, the MEG experiment on response inhibition is presented and the ensuing sensor- and source-level analyses in terms of event-related fields (ERFs) as well as oscillatory activity (time-frequency representations, TFR), followed by a brief discussion of the results.

5.2. INTRODUCTION

The behavioural measures of rapid-response impulsivity, such as the Go/No-Go (GNGNT) and Stop-Signal (SST) tasks, allow the examination of the neural mechanisms produced during inhibitory processes. Neuroimaging studies using these tasks have benefited from techniques such as functional magnetic resonance imaging (fMRI), electroencephalography (EEG) and magnetoencephalography (MEG) (e.g., Boehler et al., 2009; Falkenstein et al., 1999; Luijten et al., 2014; Simmonds et al., 2008). Using the GNGT alone during fMRI showed an activation in the anterior portion of the supplementary motor area (pre-SMA), anterior cingulate cortex (ACC), orbitofrontal cortex (OFC), right inferior frontal gyrus (rIFG) and middle frontal gyrus (MFG) (e.g., Braver, Barch, Gray, Molfese, & Snyder, 2001; Luijten et al., 2014; Simmonds et al., 2008). During the SST, it has been suggested that a network of cortical and sub-cortical regions, the rIFG, pre-SMA, and the subthalamic nucleus, are critical for inhibiting a response (Aron, Robbins, & Poldrack, 2014). Other fMRI studies have also observed greater activation in the right pre-SMA on successful stop trials compared to failed stop trials (e.g., Aron & Poldrack 2006; Boecker et al. 2011; Cai et al., 2012). See section 1.3.2.1 for more details on previous fMRI findings. Here, previous neuroimaging findings on the spectral and spatio-temporal dynamics of response inhibition are presented.

5.2.1. Event-Related Fields (ERFs)

Previous studies have examined the ERFs generated during the inhibitory process by employing the GNGT or the SST separately, or by combining both in the same study. Both NOGO and STOP conditions have been reported to elicit two particular ERP effects when compared to GO trials (no response inhibition required). Firstly, a frontocentral negativity

reported between 140 and 300 after stimulus onset, the N2, and a frontocentral positive response peak between 300 and 450 after stimulus onset (e.g., Falkenstein et al., 2002; Gao et al., 2019; Huster et al., 2013; Johnstone et al., 2007; Kreusch et al., 2014; Sehlmeyer et al., 2010). Differences in responses evoked by these tasks between high and low impulsivity individuals, might indicate a potential inhibitory deficit

NOGO condition

A stronger negative deflection detected during NOGO trials than during GO trials, has been reported between 140 and 300 ms after stimulus onset, and is considered an indicator of response inhibition (Gao et al., 2019). The neural sources of the NOGO-N2 effect have been reported in EEG to be the mid-cingulate cortex (Huster et al., 2019), the orbitofrontal area and the ACC (Luijten et al., 2014). Previous studies have reported differences in individuals with high trait anxiety or behavioural issues related to impulsivity, such as alcohol and/or tobacco use, social networking use and excessive smartphone use, compared to controls (e.g., Detandt et al., 2017; Gao et al., 2019; Kreusch et al., 2014; Sehlmeyer et al., 2010). They suggested larger NOGO-N2 amplitude or shorter latencies compared to controls, might reflect inhibitory deficits (Detandt et al., 2017; Gao et al., 2019; Kreusch et al., 2014; Sehlmeyer et al., 2010). However, others have found contradictory results, such as reduced NOGO-N2 amplitude in impulsive-violent offenders than in controls (Chen et al., 2005) or no differences in NOGO-N2 between high and low impulsivity individuals, as measured by their individual reaction times (Ruchsow et al., 2008). The contradictory results reported (e.g., Chen et al., 2005; Detandt et al., 2017; Gao et al., 2019; Kreusch et al., 2014; Sehlmeyer et al., 2010) raise the question of whether it is larger NOGO-M2 amplitudes that characterises impulsive individuals or smaller NOGO-M2 amplitudes compared to low impulsivity individuals.

Larger NOGO-P3 amplitudes reported in frontocentral regions (e.g., Huster et al., 2013; Luijten et al., 2014; Wessel & Aron, 2015), have been suggested to serve as an index of response inhibition (Huster et al., 2013; 2019; Wessel & Aron, 2015). NOGO-P3 amplitude has been found to be reduced in high impulsivity individuals, as assessed by the BIS-11 Motor (Benvenuti et al., 2015) and as assessed by individual reaction times (Ruchsow et al., 2008a), compared to low impulsivity individuals. Furthermore, a negative association between the BIS-10 (Barratt, 1985) and the NOGO-P3 amplitude has also previously been reported (Ruchsow et al., 2008b). Previous studies have observed a reduced NOGO-P3 amplitude in individuals with behavioural problems, such as alcohol (Kamarajan et al., 2005), cocaine (Sokhadze et al., 2008) or internet addiction (Dong et al., 2010), as well as internet gambling disorder (Gao et al., 2019) and excessive social networking users (Li et al., 2019), compared with controls. However, two studies have reported larger NOGO-P3 amplitudes in non-clinical populations

with impulsive characteristics, such as smokers and excessive internet users compared to controls (Detandt et al., 2017; Dong et al., 2010). These findings are hard to reconcile with the remainder of the literature, casting some doubt on a simplistic interpretation of the NOGO-P3 as an indicator of response suppression. Nonetheless, the majority of studies favour the notion that reduced NOGO-P3 amplitudes are associated with impaired response inhibition (Huster et al., 2019).

A stronger negative deflection has also been reported (NOGO-N1) compared to GO trials, between 100 and 200 ms after the stimulus onset (De Jong et al., 1990; Filipovic et al., 2000). However, results on the NOGO-N1 are even less clear as those on the NOGO-N2 and NOGO-P3. The lack of studies focusing or reporting differences on this effect in individuals with impulsive characteristics, makes the analysis of potential differences necessary. Larger NOGO-N1 amplitudes to certain cues compared to neutral stimuli might reflect the amount of attention towards the stimuli (Gao et al., 2019). Potential differences might explain what contributes to reduced task performance in impulsive individuals.

In keeping with the majority of the reviewed studies, it is hypothesised that high impulsivity individuals, as assessed by both rapid-response and reward-delay impulsivity self-report measures, will show significantly reduced task performance, larger NOGO-N2 and reduced NOGO-P3 amplitude compared to low impulsivity individuals, in line with previous literature.

STOP condition

The electrophysiological results on the SST are very contradictory. Since both, SST and GNGT are used to measure motor inhibition, similarities between findings on NOGO and STOP conditions would be expected. The larger STOP-N1 amplitude observed in high impulsivity compared to low impulsivity individuals previously reported (Dimoska & Johnstone, 2007) suggests a similar trend might be observed in the current study. Precious studies have reported reduced STOP-N2 amplitude in clinical populations, such as children with ADHD (Dimoska et al. 2003). While studies on the STOP-P3 have reported both smaller and larger amplitudes (e.g., Dimoska & Johnstone, 2007; Lansbergen et al., 2007; Shen et al., 2014), the NOGO-P3 counterpart has been found to be predominantly reduced in high impulsivity compared to low impulsivity individuals (e.g., Benvenuti et al., 2015; Ruchsow et al., 2008a).

Differences between high and low impulsivity individuals on the NOGO-P3 and STOP-P3 effects have been investigated using each task separately and on different samples; mostly resulting in contradictory evidence. I believe the literature would benefit from the examination of the neural mechanisms involved in both NOGO and STOP conditions in the same task and in the same sample. This approach allows a fair comparison between the two inhibition

paradigms, while disentangling the differences on the STOP-P3 amplitude between high and low impulsivity individuals.

In the current study, the spatio-temporal distribution of the specific ERF components generated during response inhibition was investigated, in high and low impulsivity groups. The NOGO/STOP-N1, NOGO/STOP-N2 and NOGO/STOP-P3 effects were specifically examined further by simultaneously testing the Go/No-Go and Stop-Signal tasks using MEG in a healthy undergraduate population.

5.2.2. Time-frequency representations (TFRs)

Electrophysiological studies have investigated the oscillatory activity generated during the GNGT and SST by conducting time frequency analyses (Hermann et al., 2005). Compared to the number of studies investigating ERFs using the GNGT and SST, few studies have examined the oscillatory activity generated by the SST. Thus, previous findings on both the NOGO and STOP conditions in each of the four frequency bands of interest, delta, theta, alpha and beta, are described.

NOGO and STOP conditions

Studies conducting time-frequency analyses during the GNGT have shown that theta band (4-8 Hz) oscillations play a fundamental role in response inhibition. Specifically, higher theta power has been reported in NOGO trials compared to GO trials (Beste et al., 2011; Harper et al., 2014; Huster et al., 2013; Isabella et al., 2015; Kirmizi-Alsan et al., 2006; Mückschel et al., 2017; Nakata et al., 2013; Quetscher et al., 2015). This NOGO theta activity has been found in medial-frontal sensors (Harper et al., 2014; Kirmizi-Alsan et al., 2006), which source imaging studies found to be mainly generated in the anterior cingulate cortex (ACC; Bokura et al., 2001; Cohen, 2011; Luu & Tucker, 2001; Luu et al., 2003; Pandey et al., 2012; Wang et al., 2005).

An increase in delta (1-4 Hz) power has been observed in NOGO trials compared to GO trials (Harper et al., 2014) and is thought to be involved in response inhibition and to serve as an index of motor inhibition (Kaiser et al., 2019). Previous studies have found that during the GNGT, oscillatory activity in delta and theta bands and inhibitory processes are strongly related (e.g., Harper et al., 2014; Lavallee et al., 2014). Event-related oscillations in the delta band have been reported to be associated with attentional, signal detection and decision-making processes (Basar, 1999; Basar-Eroglu et al., 1992). Even though the literature on spectral analysis of the SST is very limited, a recent EEG study using scalp-wide current source density (CSD) transformation, which attenuates the influence of problematic volume

conductance, found increased midfrontocentral theta and frontal delta during the STOP condition (Lockhart et al., 2019).

Increased alpha band (8–12 Hz) power has been associated with response inhibition during NOGO trials (Nakata et al., 2013). Specifically, significantly decreased alpha power in young individuals at risk for alcoholism (Kamarajan et al., 2006) and in abstinent alcohol-dependent adults (Pandey et al., 2016) compared to control subjects, has also been reported. During a flanker task, adolescents diagnosed with ADHD have been found to show less alpha suppression than typical developed adolescents (Mazaheri et al., 2014). This decrease in alpha power has been suggested to reflect an early attentional deficit that might affect the inhibition process (Pandey et al., 2016). To our knowledge, no significant result in alpha band activity has been reported during the stop-signal task, but it could be argued that individuals with impulsive characteristics might show a similar decrease in alpha power to those seen in previous studies (Kamarajan et al., 2006; Pandey et al., 2016).

Several studies have reported decreased delta, theta and/or alpha band power specifically during the GNGT in individuals diagnosed with ADHD (Krämer et al., 2009; Mazaheri et al., 2014), in abstinent alcohol-dependent individuals (Colrain et al., 2011; Kamarajan et al., 2004; Pandey et al., 2016), in young adults classified as binge drinkers (Lopez-Caneda et al., 2017) and in young individuals at risk for alcoholism (Kamarajan et al., 2006), compared to healthy controls. In some studies, this reduction was significantly different only during NOGO trials (e.g., Krämer et al., 2009; Kamajaran et al., 2006) and found in frontal regions, which has been suggested to reflect a deficit in fronto-parietal networks recruited during inhibitory processing (Colrain et al., 2011; Kamarajan et al., 2004; Lopez-Caneda et al., 2017). Some of these authors have proposed that weaker low-frequency oscillatory activity related to inhibitory processing, such as delta and theta, might lead to a predisposition to develop disorders characterised by disinhibition or alcohol-related disorders, and might serve as a vulnerability marker for developing alcoholism (Lopez-Caneda et al., 2017).

To our knowledge, no study has investigated the oscillatory activity in these frequency bands in healthy individuals scoring high and low on impulsivity, which has previously been done when examining ERPs (e.g., Benvenuti et al., 2015; Chen et al., 2005; Dimoska & Johnstone, 2007; Lansbergen et al., 2007; Ruchsow et al. 2008; Shen et al., 2014).

In NOGO and STOP trials, beta band (12-30 Hz) power has been seen to increase in frontal areas (Alegre et al., 2004) or central areas (Krämer et al., 2011). Previous studies have shown motor activity, such as motor preparation and execution, to be associated with beta oscillatory activity (Krämer et al., 2011; Marco-Pallares et al., 2008; Swann et al., 2009). Marco-Pallares and colleagues (2008) using EEG, reported an increase in beta during successful STOP trials

compared to GO trials and unsuccessful STOP trials. Swann et al. (2012), also reported increased beta activity in successful stop trials compared to unsuccessful trials. Consequently, fronto-central beta activity has been suggested to serve as a potential marker of inhibition in electrophysiological studies (Huster et al., 2013). Previous studies have shown changes in beta oscillatory activity in the IFC and preSMA to be related to motor execution and successful inhibition (Huster et al., 2013).

However, in those studies in which beta band power has been specifically investigated (e.g., Kamarajan et al., 2006), no significant differences were found, for example, between young individuals at risk for alcoholism and controls. Contrary to findings in theta and delta band activity, which predominantly come from experiments using the GNGT, results in beta band power have more prominently been reported in studies using the SST. I believe that investigating both GNGT and SST simultaneously and in the same sample, will provide novel findings while clarifying the role of each of the frequency bands examined here.

5.2.3. Current study

There is limited data on the spectral and spatio-temporal dynamics of response inhibition and particularly, how the behavioural measures of response inhibition, in which high impulsivity individuals have been found to show an impaired performance, are related to those neural mechanisms. The present experiment investigated the neural correlates of response inhibition using MEG in a healthy undergraduate student population, divided by their level of impulsivity as assessed by self-report measures.

I believe that the literature on this topic would benefit from the use of MEG, as this technique could effectively determine what the differences in the spatio-temporal neural dynamics of response inhibition are between individuals scoring high and low on impulsivity. This study also aimed to investigate the conflicting results from previous studies. Specifically, results on the NOGO-N2, STOP-N1, and the contradictory differences reported between the NOGO-P3 and STOP-P3 components, in high and low impulsivity individuals were examined. This was achieved by investigating these potential differences in both NOGO and STOP trials in the same task and in the same sample.

Regarding ERFs during response inhibition, it was hypothesised that (1) high impulsivity individuals would show reduced NOGO-N2 and NOGO-P3 amplitudes compared to low impulsivity individuals, (2) high impulsivity individuals would show larger STOP-N2 and reduced STOP-P3 amplitude compared to low impulsivity individuals, and (3) the HI group would show a reduced M1 amplitude in both conditions in visual areas compared to the LI group. Regarding oscillatory activity during response inhibition, decreased power in delta,

theta, alpha and beta bands was hypothesised in high impulsivity individuals compared to low impulsivity individuals.

5.3. METHODS

5.3.1. Participants

See section 2.3.2 for details.

5.3.2. Procedure

See section 2.4.2 for details on the MEG and MRI sessions.

5.3.3. Group assignment

See section 2.4.4 for details.

5.3.4. Stimuli

See section 2.5.3 for details on the task used.

5.3.5. Questionnaires

See sections 2.6.1 and 2.6.4 for details.

5.3.6. MEG data acquisition and analysis.

See section 2.8 for full details on how the MEG analysis was conducted at sensor- and source-level for ERFs and TFRs.

5.4. RESULTS

5.4.1. Behavioural results.

The mean reaction times (RT) for the correct GO trials were 532.44 (±54.0) and 515.11 (±71.4) ms for the high (HI) and low (LI) impulsivity groups, respectively. There was no significant difference between these groups ($F(1,32) = 0.64$; $p = 0.431$). Accuracy for the GO trials was 97.7% (±0.6) in the HI group and 98.1% (±0.7) for the LI group, showing no significant difference between them ($F(1,32) = 2.42$; $p = 0.130$). Accuracy for NOGO trials was 94.9% (±1.6) in the HI group and 96.1% (±0.8) in the LI group, showing a significant difference between them ($F(1,32) = 6.723$; $p = 0.014$). On STOP trials, overall accuracy was 72.1% (±8.2) and 78.9% (±7.4) in the HI and LI groups, respectively, revealing a significant difference between groups ($F(1,32) = 6.38$; $p = 0.017$). Although mean SSRT was higher in the HI group

(259.56 ms; ±58.0) than in the LI group (239.58 ms; ±55.6), this difference was not significant ($F(1,32) = 1.09$; $p = 0.302$).

5.4.2. Event-related fields (ERFs) results.

High vs Low - ERFs in the NOGO condition: sensor- and source-level analyses

The cluster-based permutation testing performed on ERFs in the NOGO condition revealed significant differences between the HI and LI groups. As shown in Figure 5.1.A, a significant difference between groups was found between 125 and 190 ms after the presentation of the NOGO cue (NOGO-M1, M denotes magnetic field; $p = 0.002$) in parieto-occipital sensors. The HI group showed significantly smaller amplitude than the LI group. Another significant difference between groups was found between 210 and 265 ms post-cue (NOGO-M2; $p = 0.012$), during the N2 component, localised in parieto-occipital sensors. The HI group showed significantly smaller amplitude, see Figure 5.1.A. A significant difference between groups was also found between 270 and 410 ms post-cue (NOGO-M2b; $p = 0.002$) in left parieto-occipital sensors. The HI group showed significantly smaller amplitudes than the LI group, see Figure 5.1.A The N2b has previously been reported in an EEG study during an oddball task, as a negativity which is preceded by the NOGO-N2, with smaller amplitudes in both the NOGO-N2 and -N2b in depressed individuals compared to controls (Ogura et al., 1993)

The group-averaged ERFs and the differences between groups observed in sensor-level analysis during NOGO trials, were reconstructed and localised in source-space using an LCMV beamformer, see section 2.8.3.2 for details. The mean source activity in each group was calculated for the time intervals of interest, corresponding to NOGO-M1, -M2 and M2b components. HI and LI group means were compared using cluster-based permutations tests to control for multiple comparisons (Maris & Oostenveld, 2007), for each NOGO component, see section 2.8.3.2 for details.

The comparison of HI and LI groups of the NOGO-M1 component, HI > LI, showed significant negative clusters located in the right inferior parietal region, right inferior and middle frontal gyrus, right middle and inferior parts of the frontal gyrus, right inferior and middle temporal regions, right superior temporal pole and left orbital part of medial frontal gyrus; $p < .05$, see Figure 5.1.C. The comparison of HI and LI groups of the NOGO-M2 component showed significant negative clusters located in the right inferior parietal, right inferior occipital and right superior temporal regions, bilateral superior and medial frontal gyrus, right orbital part of superior frontal gyrus, left middle occipital and left middle temporal region regions; $p < .05$, see Figure 5.1.C. The comparison of HI and LI groups of the NOGO-M2b component revealed a significant negative cluster for the HI group in the right inferior and superior parietal region,

bilaterally in the supplementary motor area, the right superior and middle temporal pole and right inferior temporal region; $p < .05$, see Figure 5.1.C.

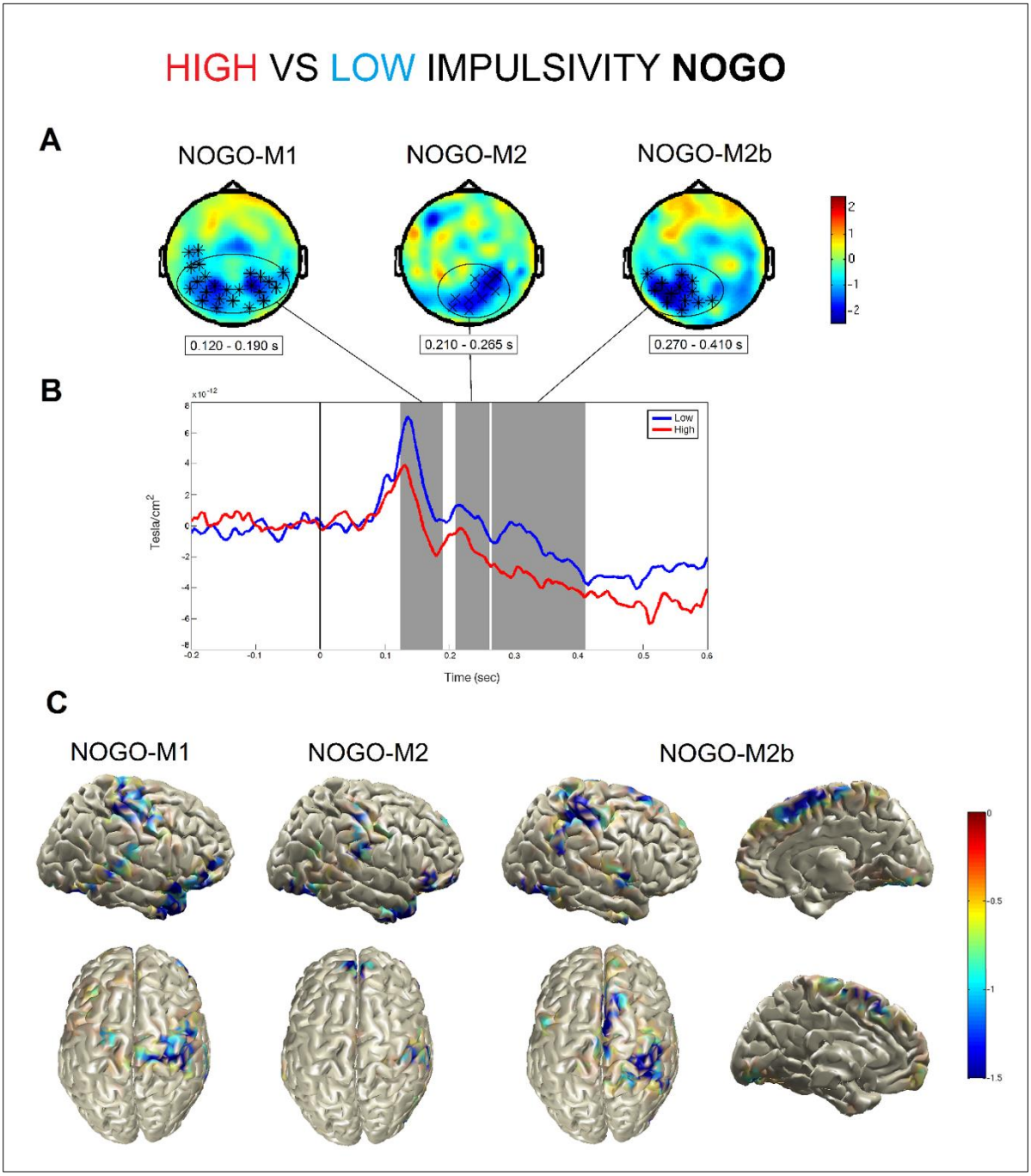

Figure 5.1. (A) Results from sensor-level analysis: topography of the statistical difference in amplitude between HI and LI groups during the NOGO condition, between 0.105-0.170 (NOGO-M1), 0.210-265 (NOGO-M2) and 0.270-0.410 (NOGO M2b) seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$. The colour scale represents the *t*-values, hot for positive and cool colours for negative values; x indicates significant clusters with $p < .05$; * indicates significant clusters with $p < .01$. (B) Group averages of ERFs during NOGO trials for the HI (red line) and LI (blue line) groups, the group averages were plotted from significant sensors ($p < .05$). The grey columns represent the statistical difference between groups from the cluster-based permutations, see section 2.8.2.1 for details. Time point 0, marked with a

black line, denotes the onset of target stimulus presentation. (C) Results from source-level analysis: the statistical differences observed in amplitude between HI and LI groups during NOGO trials, were localised in source-space, see section 2.8.3.2 for details. The colour scale represents the t-values, from 0 to -1.5.

High vs Low - ERFs in the STOP condition: sensor- and source-level analyses

The cluster-based permutation testing performed on ERFs in the STOP condition revealed significant differences between the HI and LI groups, see section 2.8.2.1 for details. As seen in Figure 5.2.A, a significant difference between groups between 105 and 170 ms after the presentation of the STOP cue (M1; p = 0.004) was found during the standard time window of the N1 component in parieto-occipital sensors. The HI group showed a significantly smaller amplitude than the LI group, see Figure 5.2.A. A significant difference between groups was also found between 230 and 255 ms post-cue. The HI group showed a significant decrease in amplitude compared to the LI group (M2-Low impulsivity group, M2L; p = 0.006) in posterior sensors, also affecting more central anterior sensors, see Figure 5.3.A. However, a significant difference between groups was also found between 280–330 ms post-cue, in which the HI group showed a significant increase in amplitude (M2-High impulsivity group, STOP- M2H; p = 0.002) in anterior and right posterior sensors, while the amplitude in the LI group was already decreasing (from their STOP-M2 peak), see Figure 5.4.A. This indicates a delayed STOP-M2 in the HI group. The amplitudes of the STOP-M2L and STOP-M2H were compared statistically, which showed they were not significantly different (peak amplitude of NOGO-M2L = 3.6x10e-13; peak amplitude of STOP-M2H = 2.8x10e-13; t(1) = 8.00; p > 0.05). Finally, a significant difference between groups was also found between 340–385 ms post-cue in a time window typical for the M3 (p = 0.020), in which the HI group showed a significant increase in amplitude compared to the LI group, see Figure 5.5.A.

The group-averaged ERFs and the differences between groups observed in sensor-level analysis during STOP trials, were reconstructed and localised in source-space using an LCMV beamformer, see section 2.8.3.2 for details. The mean source activity in each group was calculated for the time intervals of interest, corresponding to STOP-M1, -M2H, -M2L and -M3 components. HI and LI group means were compared using cluster-based permutations tests to control for multiple comparisons (Maris & Oostenveld, 2007), see section 2.8.3.2 for details.

For the STOP-M1 component, the comparison between HI and LI groups, HI > LI, showed a significant negative cluster located in the right inferior parietal area; p < .05, see Figure 5.2.C. During the STOP-M2L, the comparison of HI and LI groups showed significant negative clusters in the right supplementary motor area and bilaterally in medial superior frontal gyrus; p < .05, see Figure 5.3.C. For the STOP-M2H component, the comparison of HI and LI groups showed significant positive clusters in the right fronto-temporal region and left parietal and

frontal regions. Specifically, the positive clusters were located in the right inferior frontal gyrus, bilateral superior parietal, bilateral precuneus, left superior frontal gyrus, left middle frontal gyrus, left orbital part of superior frontal gyrus, left anterior cingulate and left middle cingulate; $p < .05$, see Figure 5.4.C. During the STOP-M3 component, the comparison of HI and LI groups showed significant positive clusters located in the right anterior cingulate, bilateral supplementary motor area, bilateral superior frontal gyrus and left temporal regions; $p < .05$, see Figure 5.5.C.

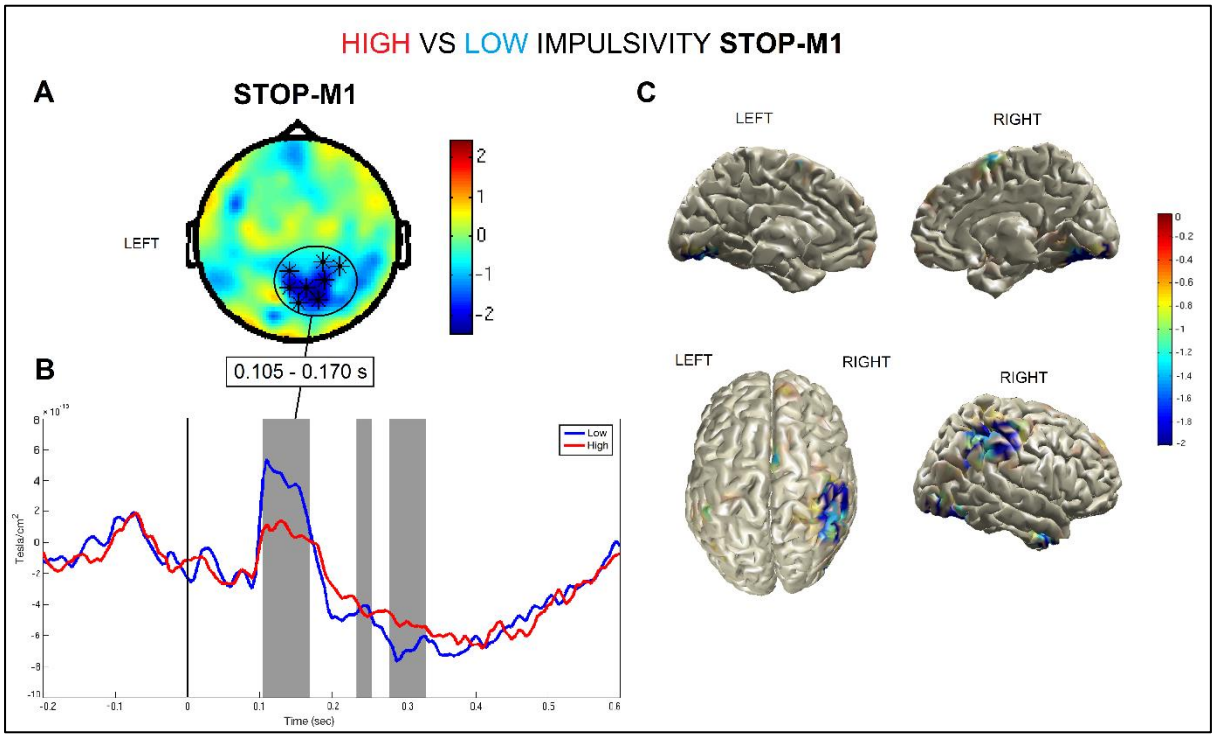

Figure 5.2. (A) Sensor-level analysis: topography of the statistical difference in amplitude between HI and LI groups during the STOP condition, between 0.105 and 0.170 seconds after stimulus onset. *T*-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$. The colour scale represents the *t*-values, * indicates significant clusters with $p < .01$. (B) Group averages of ERFs during STOP trials for the HI (red line) and LI (blue line) groups, the group averages were plotted from significant sensors ($p < .01$) only. The grey columns represent the statistical difference between groups from the cluster-based permutations, see section 2.8.2.1 for details. Time point 0, marked with a black line, denotes the onset of target stimulus presentation. (C) Source-level analysis: the statistical differences observed in amplitude between HI and LI groups during STOP trials, were localised in source-space, see section 2.8.3.2 for details. The colour scale represents the *t*-values, from 0 to -2.

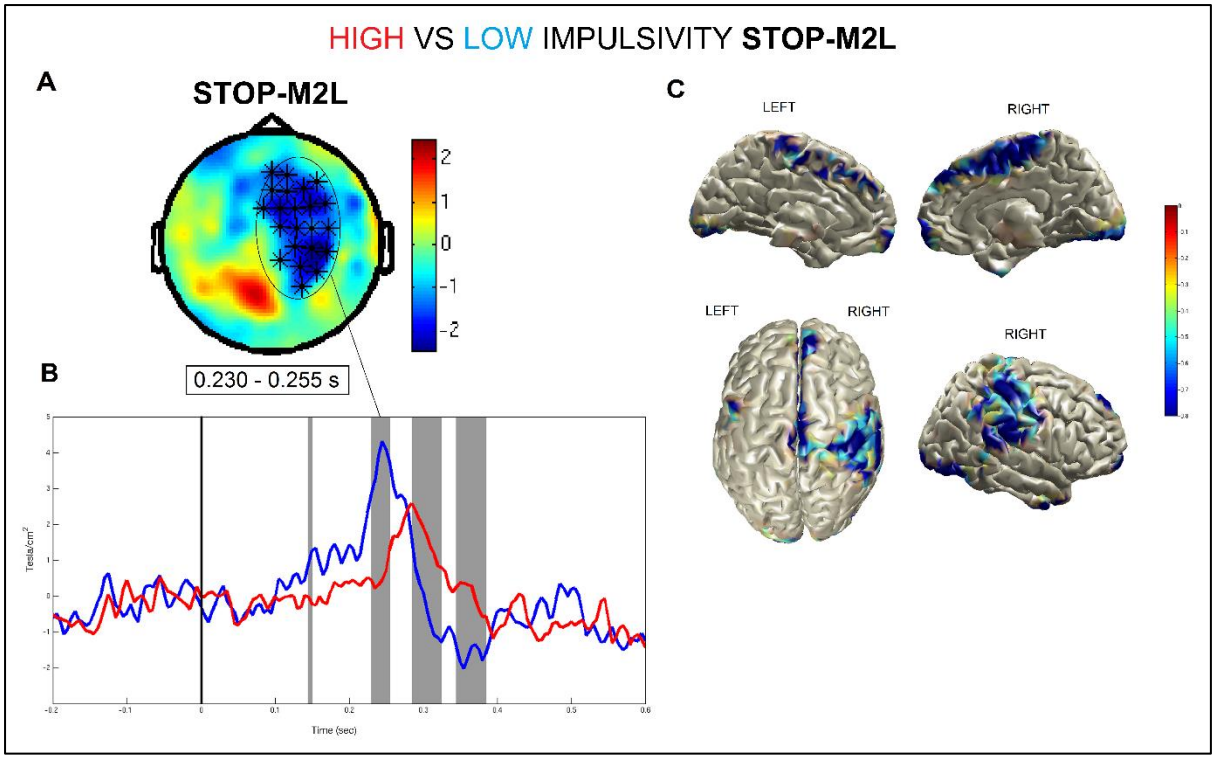

Figure 5.3. (A) Results from sensor-level analysis: topography of the statistical difference in amplitude between HI and LI groups during the STOP condition, between 0.230 and 0.255 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$. The colour scale represents the t-values, * indicates significant clusters with $p < .01$. (B) Group average topo-plots, baseline corrected, during STOP trials, between 0.230 and 0.255 seconds after stimulus onset, in HI and LI groups. The colour scale denotes the intensity of the magnetic field, which is measured using units of Tesla/cm2. (C) Group averages of ERFs during STOP trials for the HI (red line) and LI (blue line) groups, the group averages were plotted from significant sensors ($p < .01$) only. The grey columns represent the statistical difference between groups from the cluster-based permutations, see section 2.8.2.1 for details. Time point 0, marked with a black line, denotes the onset of target stimulus presentation. (D) Source-level analysis: the statistical differences observed in amplitude between HI and LI groups during STOP trials, were localised in source-space, see section 2.8.3.2 for details. The colour scale represents the t-values, from 0 to -0.8.

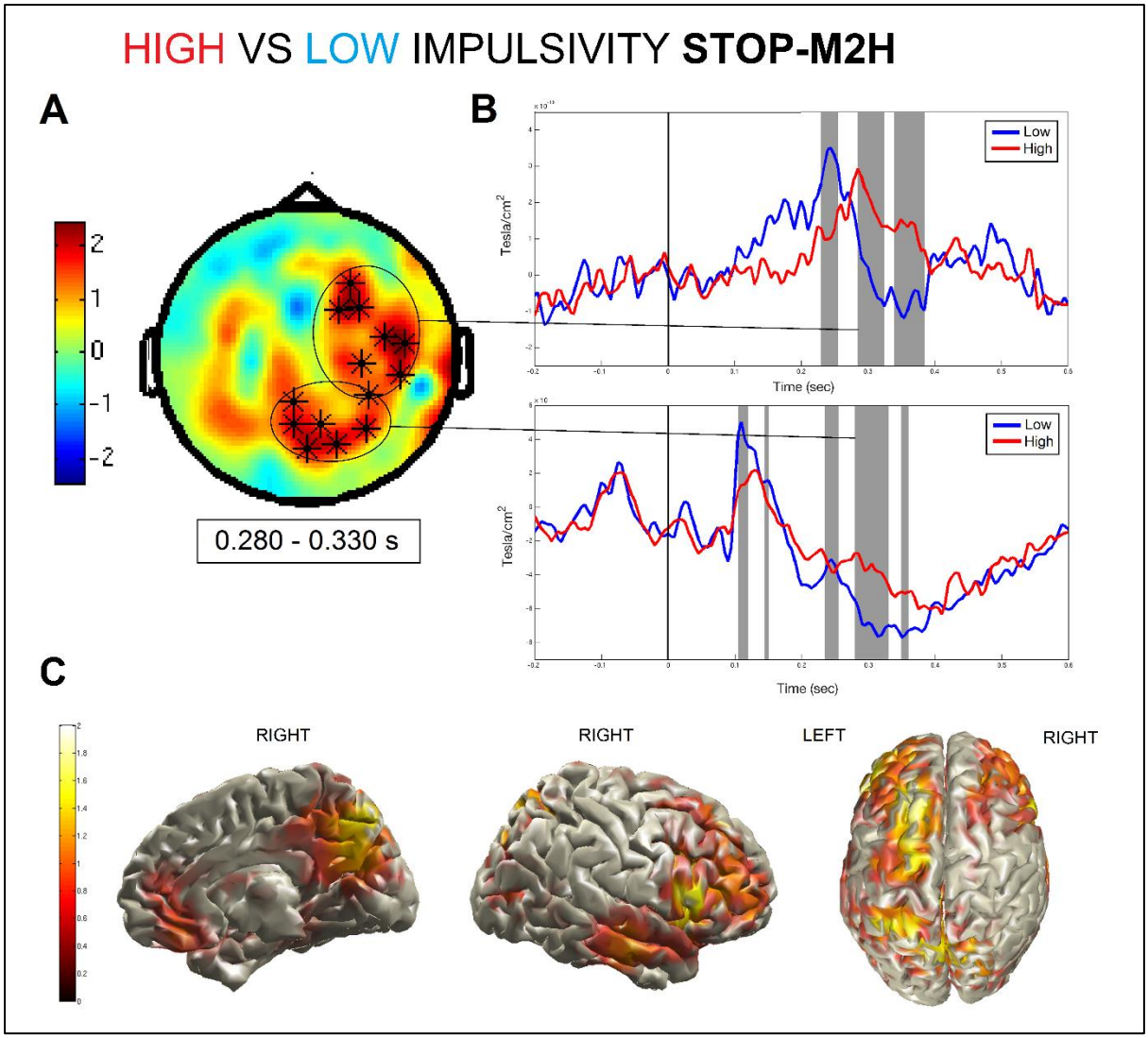

Figure 5.4. (A) Sensor-level analysis: topography of the statistical difference in amplitude between HI and LI groups during the STOP condition, between 0.280 and 0.330 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$. The colour scale represents the t-values, * indicates significant clusters with $p < .01$. (B) Group averages of ERFs during STOP trials for the HI (red line) and LI (blue line) groups, the group averages were plotted from significant sensors ($p < .01$) only. The grey columns represent the statistical difference between groups from the cluster-based permutations, see section 2.8.2.1 for details. Time point 0, marked with a black line, denotes the onset of target stimulus presentation. (C) Source-level analysis: the statistical differences observed in amplitude between HI and LI groups during STOP trials, were localised in source-space, see section 2.8.3.2 for details. The colour scale represents the t-values, from 0 to 2.

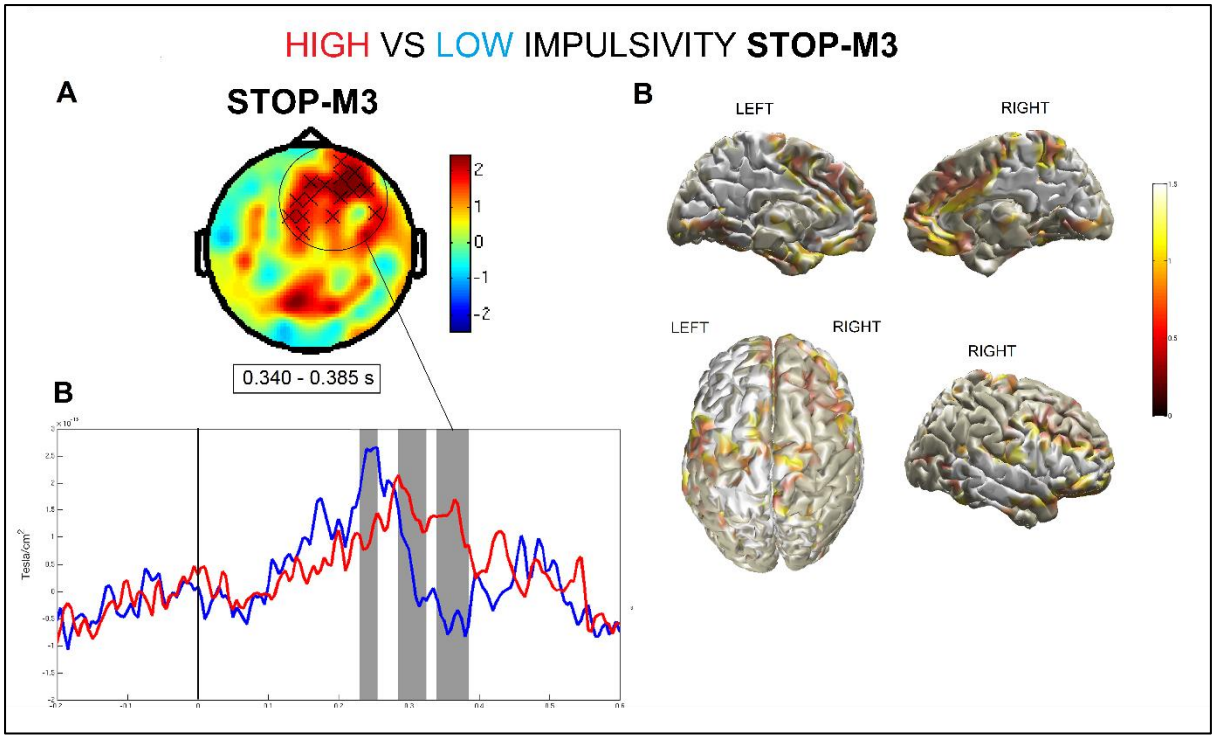

Figure 5.5. (A) Sensor-level analysis: topography of the statistical difference in amplitude between HI and LI groups during the STOP condition, between 0.340 and 0.385 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$. The colour scale represents the t-values, x indicates significant clusters with $p < .05$. (B) Group averages of ERFs during STOP trials for the HI (red line) and LI (blue line) groups, the group averages were plotted from significant sensors ($p < .05$) only. The grey columns represent the statistical difference between groups from the cluster-based permutations, see section 2.8.2.1 for details. Time point 0, marked with a black line, denotes the onset of target stimulus presentation (C) Source-level analysis: the statistical differences observed in amplitude between HI and LI groups during STOP trials, were localised in source-space, see section 2.8.3.2 for details. The colour scale represents the t-values, from 0 to 1.5.

5.4.3. Frequency and time-frequency results

High vs Low - NOGO condition: Sensor-level and source-level analyses

Delta band activity

Cluster-based permutation tests, which contrasted the HI and LI groups in the NOGO condition with respect to time-averaged power for the frequency range of 1-4 Hz, revealed significant differences between groups. As shown in Figure 5.6.A, two significant differences between groups were observed in this frequency range. Firstly, a significant difference was observed between 0 and 352 ms after the stimulus onset ($p = 0.028$), where the HI group showed significantly reduced delta power relative to the LI group in frontal sensors. Secondly, a significant difference was found between 0 and 500 ms after the presentation of the NOGO

cue (p = 0.001), where HI participants showed significantly higher delta power than LI participants in posterior sensors.

At source-level, the statistical comparison of the HI group against the LI group, during NOGO trials in the delta band (1-4 Hz), showed significant negative clusters located in frontal, temporal and occipital regions, specifically in left dorsal, lateral, orbital, and medial prefrontal cortex (PFC) extending into right orbital and medial PFC, incl. anterior cingulate cortex (ACC), as well as including posterior regions such as occipital and parietal areas; p < .05, see Figure 5.6.C. The same cluster-based permutation test also revealed significant positive clusters in the left superior parietal cortex, in the left inferior and bilateral middle occipital cortex, and right inferior and middle temporal regions; p < .05, see Figure 5.6.C.

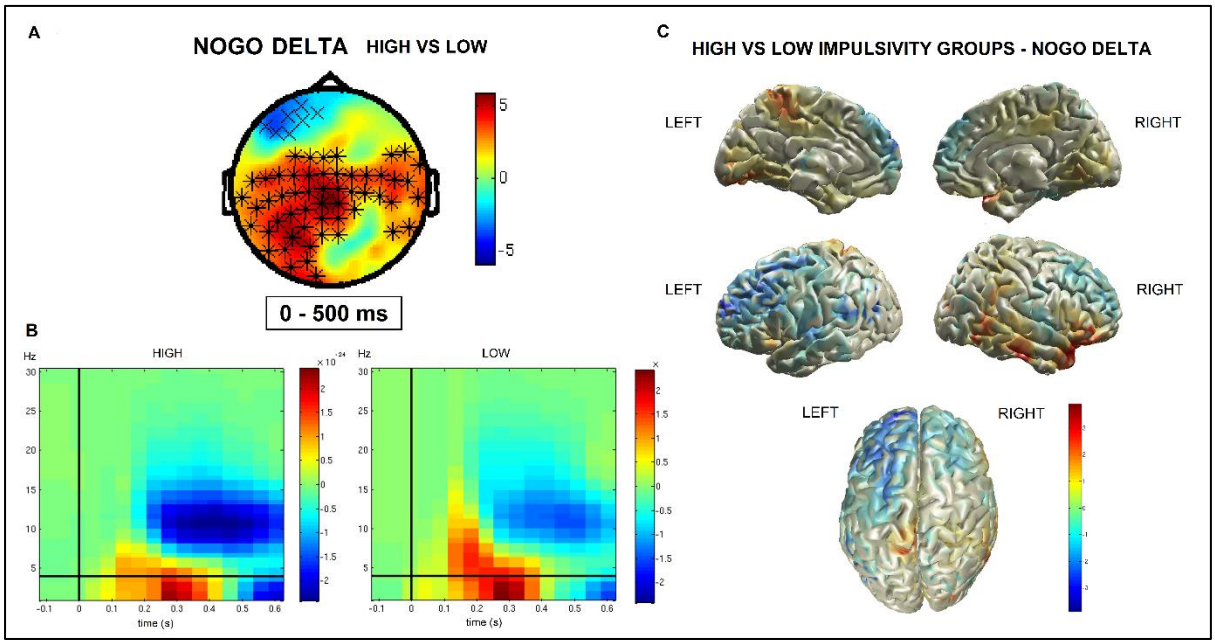

Figure 5.6. (A) Results from sensor-level analysis: topography of the statistical difference in delta power between HI and LI groups during the NOGO condition, between 0 and 0.500 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and p < .05; see section 2.8.2.2 for details. As a result of contrasting the HI against the LI group, hot colours denote positive power difference, cool colours negative power difference relative to the HI group. The colour scale represents the *t*-values, hot for positive and cool colours for negative values; * indicates significant clusters with p < .01. **(B)** Baseline corrected TFR power plots for NOGO trials in the HI and LI groups, showing delta, theta, alpha and beta bands. The y-axis denotes frequency, the x-axis denotes time in ms, t=0 and vertical black lines represent the presentation of the NOGO cue, horizontal black lines limit delta band. The colour scale represents power in relation to the baseline period, power values from -2 to 2 ($x10^{-24}$). **(C)** Results from source-level analysis: the statistical differences observed in delta band power between HI and LI groups during NOGO trials, were localised in source-space, see section 2.8.3.3 for details. The colour scale represents the t-values, from -3.5 to 3.5.

Theta band activity

Cluster-based permutation tests, which contrasted the HI and LI groups in the NOGO condition with respect to time-averaged power for the frequency range of 4-8 Hz, revealed significant differences between groups. As shown in Figure 5.7.A, two significant differences between groups were observed in this frequency range. A significant negative cluster was observed between 0 and 352 ms after the stimulus onset ($p = 0.031$) where the HI group showed significantly reduced theta power relative to the LI group in frontal sensors. Furthermore, a significant positive difference between groups was found between 0 and 500 ms after the presentation of the NOGO cue ($p = 0.001$) individuals showed significantly higher theta power than LI participants in posterior sensors.

In source-space, the statistical comparison of the HI group against the LI group, during NOGO trials in theta band (4-8 Hz), showed significant negative clusters located bilaterally in the supplementary motor area (SMA), bilateral medial, incl. ACC, and superior frontal regions, incl. the frontal eye fields (FEF), bilateral middle and lateral frontal gyrus, orbital PFC, bilateral middle and posterior cingulate cortex, bilateral inferior parietal regions, left superior parietal regions and right superior temporal regions ($p < .05$, see Figure 5.7.C). The same cluster-based permutation test also revealed significant positive clusters in right middle and superior occipital cortex, bilateral superior temporal gyrus, and bilateral ventromedial PFC ($p < .05$, see Figure 5.7.C).

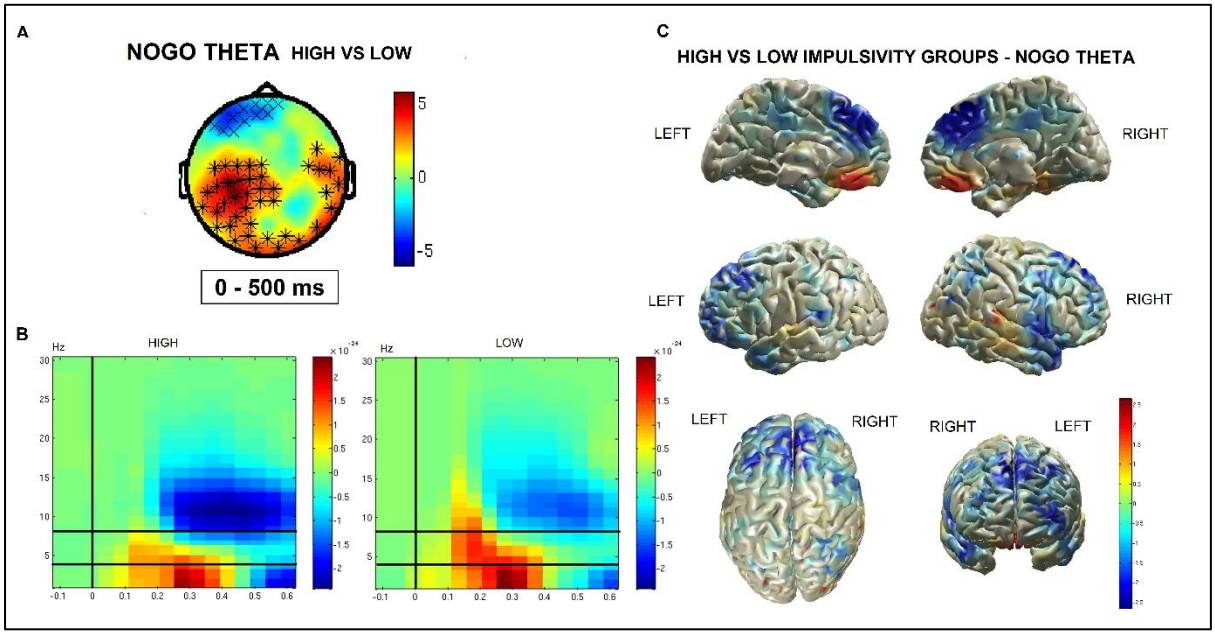

Figure 5.7. (A) Results from sensor-level analysis: topography of the statistical difference in theta power between HI and LI groups during the NOGO condition, between 0 and 0.500 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$; see section 2.8.2.2 for details. As a result of contrasting the HI against the LI group, hot colours denote positive power difference, cool colours negative power difference relative to the HI group. The colour scale

represents the *t*-values, hot for positive and cool colours for negative values; x indicates significant clusters with $p < .05$, * indicates significant clusters with $p < .01$. **(B)** Baseline corrected TFR power plots for NOGO trials in the HI and LI groups, showing delta, theta, alpha and beta bands. The y-axis denotes frequency, the x-axis denotes time in ms, t=0 and vertical black lines represent the presentation of the NOGO cue, horizontal black lines limit theta band. The colour scale represents power in relation to the baseline period, power values from -2 to 2 ($x10^{-24}$). **(C)** Results from source-level analysis: the statistical differences observed in theta band power between HI and LI groups during NOGO trials, were localised in source-space, see section 2.8.3.3 for details. The colour scale represents the t-values, from -2.5 to 2.5.

Alpha band activity

Cluster-based permutation tests, which contrasted the HI and LI groups in the NOGO condition with respect to time-averaged power for the frequency range of 8-12 Hz, revealed significant differences between groups. As shown in Figure 5.8.A, two significant differences between groups were observed in this frequency range. A significant negative difference was observed between 0 and 500 ms after the stimulus onset ($p = 0.001$), where the HI group showed significantly reduced alpha power relative to the HI group in frontal sensors. A significant positive difference was found between 0 and 500 ms after the presentation of the NOGO cue ($p = 0.001$), where HI participants showed significantly increased alpha power than LI in posterior sensors.

At source-level, the statistical comparison of the HI group against the LI group, during NOGO trials in alpha band (8-12 Hz), showed significant negative clusters located bilaterally in the medial part of the superior frontal gyrus, incl. SMA, left middle and superior frontal gyrus, incl. FEF, and right superior parietal regions ($p < .05$, see Figure 5.8.C). The same cluster-based permutation test also revealed significant positive clusters in the right superior parietal region, left inferior and superior temporal regions, right inferior and middle temporal regions, and ventromedial PFC ($p < .05$, see Figure 5.8.C).

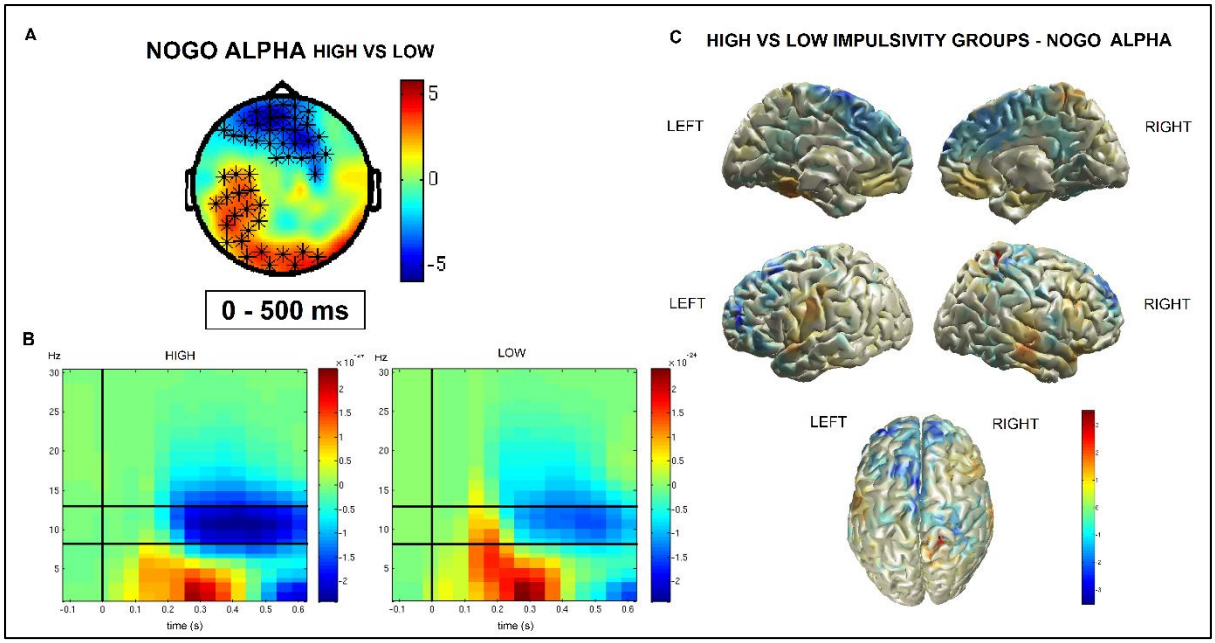

Figure 5.8. (A) Results from sensor-level analysis: topography of the statistical difference in alpha band power between HI and LI groups during the NOGO condition, between 0 and 0.500 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$; see section 2.8.2.2 for details. As a result of contrasting the HI against the LI group, hot colours denote positive power difference, cool colours negative power difference relative to the HI group. The colour scale represents the *t*-values, hot for positive and cool colours for negative values; * indicates significant clusters with $p < .01$. **(B)** Baseline corrected TFR power plots for NOGO trials in the HI and LI groups, showing delta, theta, alpha and beta bands. The y-axis denotes frequency, the x-axis denotes time in ms, t=0 and vertical black lines represent the presentation of the NOGO cue, horizontal black lines limit alpha band. The colour scale represents power in relation to the baseline period, power values from -2 to 2 ($x10^{-24}$). **(C)** Results from source-level analysis: the statistical differences observed in alpha band power between HI and LI groups during NOGO trials, were localised in source-space, see section 2.8.3.3 for details. The colour scale represents the t-values, from -3.5 to 3.5.

Beta band activity

Cluster-based permutation tests, which contrasted the HI and LI groups in the NOGO condition with respect to time-averaged power for the frequency range of 12-30 Hz, revealed significant differences between groups. As shown in Figure 5.9.A, two significant differences between groups were observed in this frequency range, similar to the previously reported frequency bands. A significant negative difference was observed between 0 and 500 ms after the stimulus onset ($p = 0.001$), where the HI group showed significantly lower beta power than the HI group in anterior regions. A significant positive difference was also found between 0 and 500 ms after the presentation of the NOGO cue ($p = 0.001$), where HI participants showed significantly increased beta power than LI in posterior sensors.

At source-level, the statistical comparison of the HI group against the LI group, during NOGO trials in beta band (13-30 Hz), showed significant negative clusters located in the bilateral medial, including SMA and dorsal ACC, frontal region, orbital part of middle frontal gyrus, left middle temporal, right inferior and middle occipital regions ($p < .05$, see Figure 5.9.C). The same cluster-based permutation test also revealed significant positive clusters in right inferior, middle and superior temporal regions, left SMA, left medial part of superior frontal gyrus, left superior frontal gyrus, bilateral middle and medial temporal cortex, left inferior and bilateral superior parietal regions, and bilateral middle occipital cortex ($p < .05$, see Figure 5.9.C).

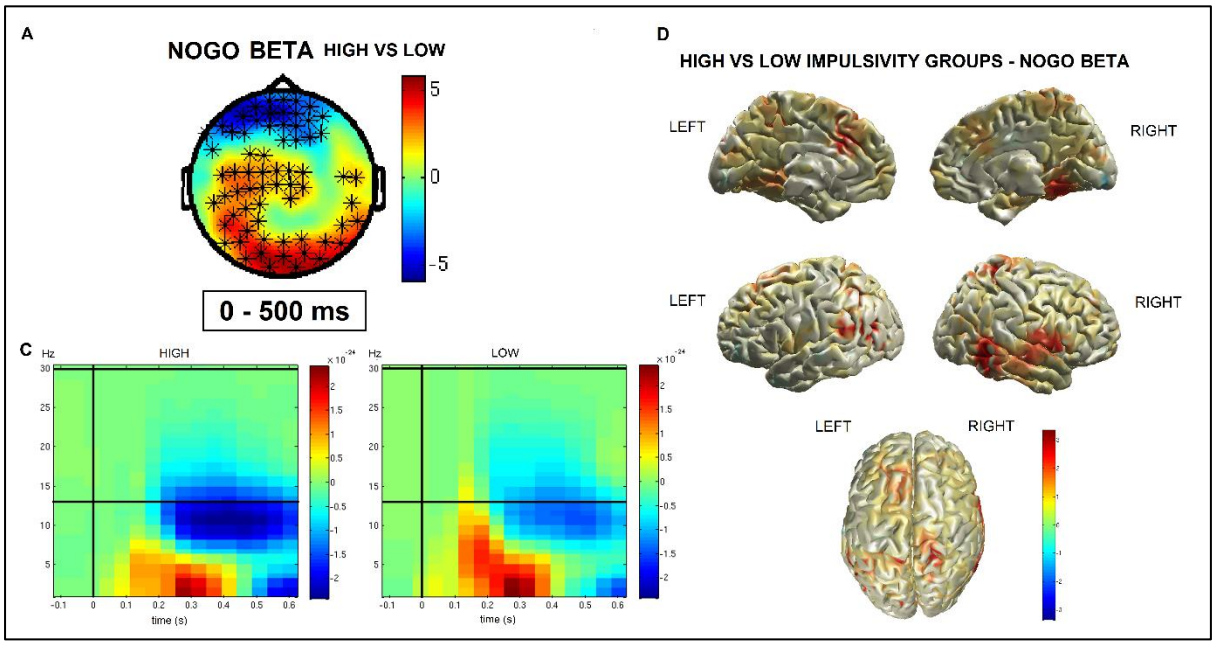

Figure 5.9. (A) Results from sensor-level analysis: topography of the statistical difference in beta power between HI and LI groups during the NOGO condition, between 0 and 0.500 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$; see section 2.8.2.2 for details. As a result of contrasting the HI against the LI group, hot colours denote positive power difference, cool colours negative power difference relative to the HI group. The colour scale represents the *t*-values, hot for positive and cool colours for negative values; * indicates significant clusters with $p < .01$ **(B)** Baseline corrected TFR power plots for NOGO trials in the HI and LI groups, showing delta, theta, alpha and beta bands. The y-axis denotes frequency, the x-axis denotes time in ms, t=0 and vertical black lines represent the presentation of the NOGO cue, horizontal black lines limit beta band. The colour scale represents power in relation to the baseline period, power values from -2 to 2 ($x10^{-24}$). **(C)** Results from source-level analysis: the statistical differences observed in beta band power between HI and LI groups during NOGO trials, were localised in source-space, see section 2.8.3.3 for details. The colour scale represents the t-values, from -3.5 to 3.5.

High vs Low – STOP condition: Sensor-level and source-level analyses

Delta band activity

Cluster-based permutation tests, which contrasted the HI and LI groups in the STOP condition with respect to time-averaged power for the frequency range of 1-4 Hz, revealed significant differences between groups. As shown in Figure 5.10.A, two significant differences between groups were observed in this frequency range. A significant difference was observed between 0 and 500 ms after stimulus onset ($p = 0.001$) in frontal regions, where the LI group showed significantly higher delta power relative to the HI group in anterior sensors. A second significant difference was found between 0 and 500 ms after the presentation of the STOP cue ($p = 0.001$), where HI participants showed significantly higher delta power than LI participants in significantly decreased delta power in anterior sensors and posterior regions.

At source-level, the statistical comparison of the HI group against the LI group, during STOP trials in delta band (1-4 Hz), showed significant negative clusters located bilaterally in the SMA, ACC, right middle frontal gyrus, left inferior and middle temporal regions, bilateral inferior parietal regions; $p < .05$, see Figure 5.10.C. The same cluster-based permutation testing also revealed significant positive clusters in the left middle and superior frontal gyrus, bilateral inferior frontal and right superior frontal regions, insula, fusiform, bilaterally in the middle temporal region and left superior parietal region; $p < .05$, see Figure 5.10.C.

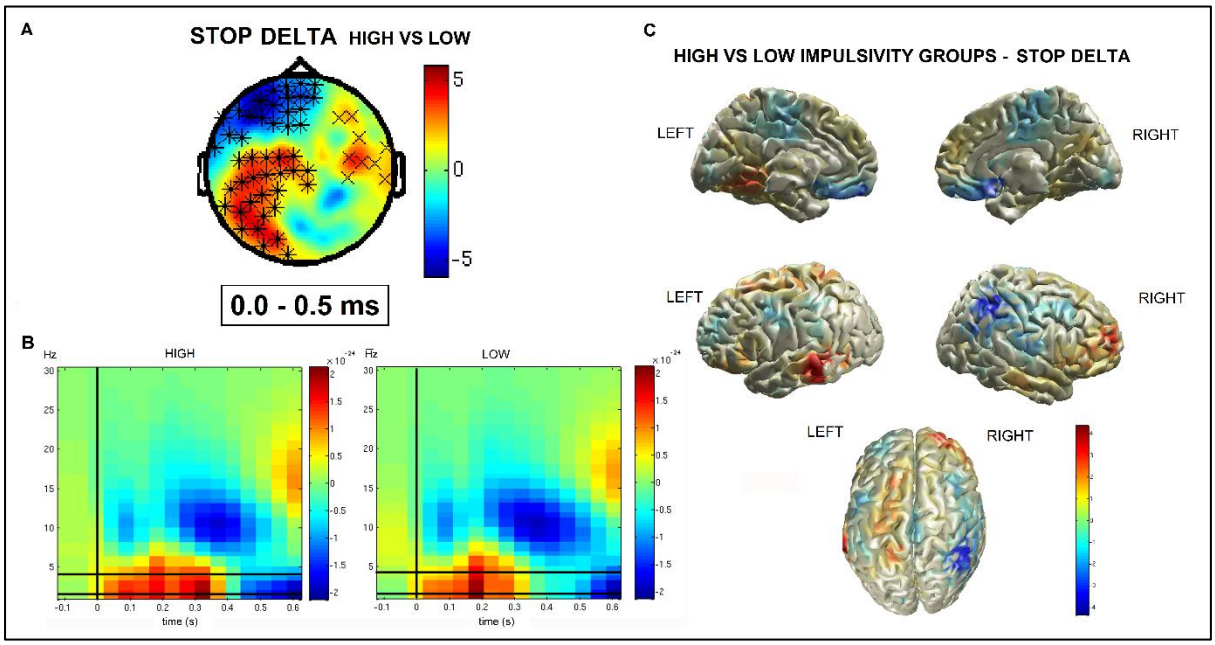

Figure 5.10. (A) Results from sensor-level analysis: topography of the statistical difference in beta power between HI and LI groups during the STOP condition, between 0 and 0.500 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$; see section 2.8.2.2 for details. As a result of contrasting the HI against the LI group, hot colours denote positive power difference, cool colours negative power difference relative to the HI group. The colour scale

represents the t-values, hot for positive and cool colours for negative values; x indicates significant clusters with $p < .05$, * indicates significant clusters with $p < .01$. **(B)** Baseline corrected TFR power plots for STOP trials in the HI and LI groups, showing delta, theta, alpha and beta bands. The y-axis denotes frequency, the x-axis denotes time in ms, t=0 and vertical black lines represent the presentation of the STOP cue, horizontal black lines limit delta band. The colour scale represents power in relation to the baseline period, power values from -2 to 2 ($x10^{-24}$). **(C)** Results from source-level analysis: the statistical differences observed in delta band power between HI and LI groups during STOP trials, were localised in source-space, see section 2.8.3.3 for details. The colour scale represents the t-values, from -4.5 to 4.5.

Theta band activity

Cluster-based permutation tests, which contrasted the HI and LI groups in the STOP condition with respect to time-averaged power for the frequency range of 4-8 Hz, revealed significant differences between groups. As shown in Figure 5.11.A, two significant differences between groups were observed in this frequency range. A significant difference was observed between 0 and 500 ms after stimulus onset ($p = 0.001$) in frontal regions, where the HI group showed significantly reduced theta power relative to the LI group in anterior sensors. A significant positive difference was found between 0 and 500 ms after the presentation of the STOP cue ($p = 0.001$), where HI participants showed significantly increased theta power than LI participants in posterior sensors.

At source-level, the statistical comparison of the HI group against the LI group, during STOP trials in theta band (4-8 Hz), showed significant negative clusters located in the left superior frontal gyrus, SMA, fusiform, left inferior parietal and right inferior occipital regions; $p < .05$, see Figure 5.11.C. The same cluster-based permutation testing also revealed significant positive clusters in the right middle and superior frontal gyrus, left inferior frontal regions, right middle temporal regions, left inferior and middle temporal regions, right middle and superior occipital regions; $p < .05$, see Figure 5.11.C.

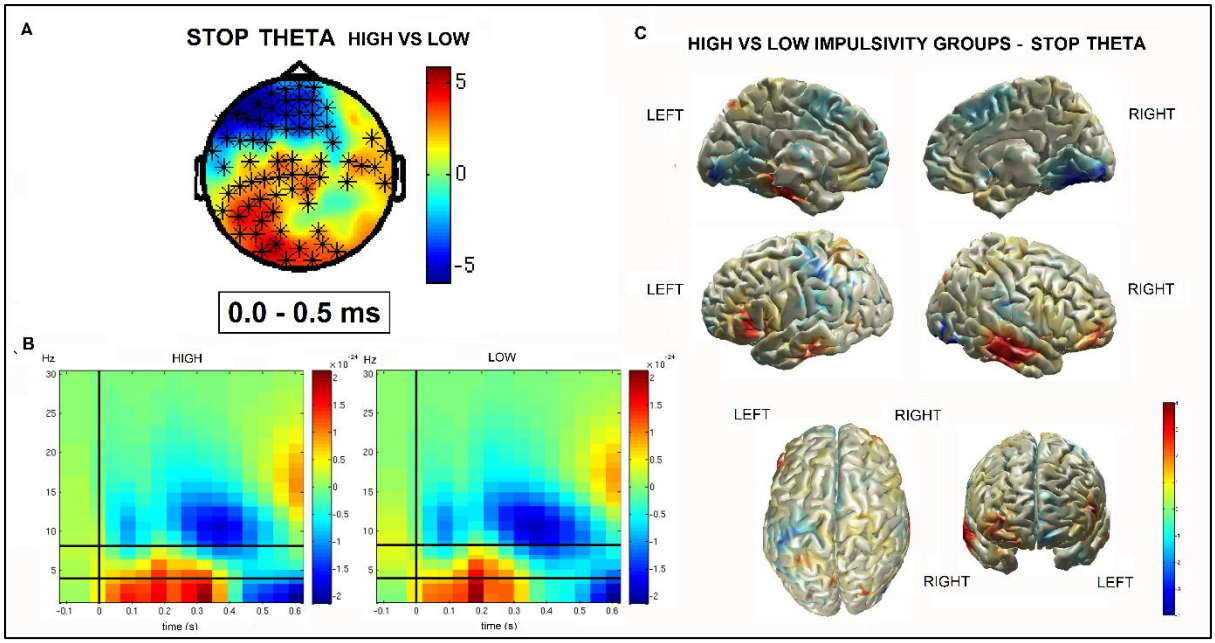

Figure 5.11. (A) Results from sensor-level analysis: topography of the statistical difference in theta power between HI and LI groups during the STOP condition, between 0 and 0.500 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$; see section 2.8.2.2 for details. As a result of contrasting the HI against the LI group, hot colours denote positive power difference, cool colours negative power difference relative to the HI group. The colour scale represents the t-values, hot for positive and cool colours for negative values; * indicates significant clusters with $p < .01$. **(B)** Baseline corrected TFR power plots for STOP trials in the HI and LI groups, showing delta, theta, alpha and beta bands. The y-axis denotes frequency, the x-axis denotes time in ms, t=0 and vertical black lines represent the presentation of the STOP cue, horizontal black lines limit theta band. The colour scale represents power in relation to the baseline period, power values from -2 to 2 ($x10^{-24}$). **(C)** Results from source-level analysis: the statistical differences observed in theta band power between HI and LI groups during STOP trials, were localised in source-space, see section 2.8.3.3 for details. The colour scale represents the t-values, from -4 to 4.

Alpha band activity

Cluster-based permutation tests, which contrasted the HI and LI groups in the STOP condition with respect to time-averaged power for the frequency range of 8-12 Hz, revealed significant differences between groups. As shown in Figure 5.12.A, two significant differences between groups were observed in this frequency range. A significant difference was observed between 0 and 500 ms after stimulus onset ($p = 0.001$) in frontal regions, where the HI group showed significantly higher alpha power relative to the LI group in anterior sensors. A second significant difference was found between 0 and 500 ms after the presentation of the STOP cue ($p = 0.012$) in posterior sensors, where the HI group showed significantly lower alpha power than the LI group.

At source-level, the statistical comparison of the HI group against the LI group, during STOP trials in alpha band (8-12 Hz), showed significant negative clusters located bilaterally in medial part of the superior frontal gyrus, right fusiform, right superior parietal, left inferior parietal and right inferior occipital regions; $p < .05$, see Figure 5.12.C. The same cluster-based permutation testing also revealed significant positive clusters in the orbital part of the inferior frontal gyrus, right superior frontal gyrus, middle cingulate, left fusiform, middle temporal, right superior parietals and left superior temporal regions; $p < .05$, see Figure 5.12.C.

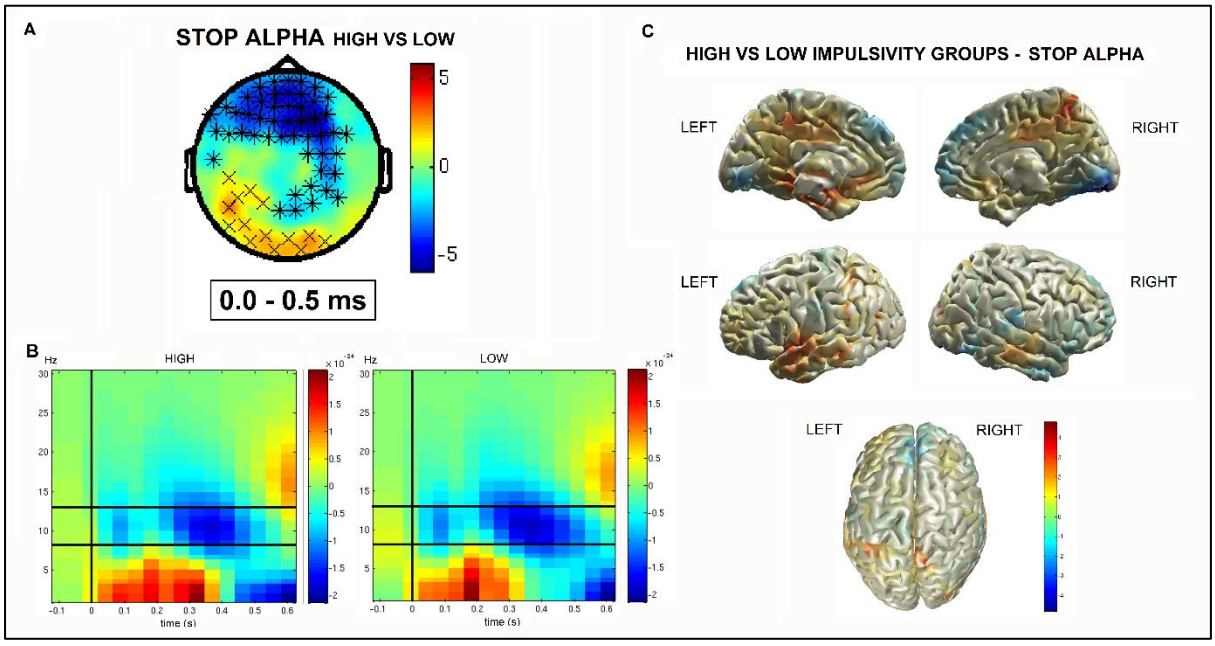

Figure 5.12. (A) Results from sensor-level analysis: topography of the statistical difference in alpha power between HI and LI groups during the STOP condition, between 0 and 0.500 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$; see section 2.8.2.2 for details. As a result of contrasting the HI against the LI group, hot colours denote positive power difference, cool colours negative power difference relative to the HI group. The colour scale represents the t-values, hot for positive and cool colours for negative values; x indicates significant clusters with $p < .05$; * indicates significant clusters with $p < .01$. **(B)** Baseline corrected TFR power plots for STOP trials in the HI and LI groups, showing delta, theta, alpha and beta bands. The y-axis denotes frequency, the x-axis denotes time in ms, t=0 and vertical black lines represent the presentation of the STOP cue, horizontal black lines limit alpha band. The colour scale represents power in relation to the baseline period, power values from -2 to 2 ($x10^{-24}$). **(C)** Results from source-level analysis: the statistical differences observed in alpha band power between HI and LI groups during STOP trials, were localised in source-space, see section 2.8.3.3 for details. The colour scale represents the t-values, from -4.5 to 4.5.

Beta band activity

Cluster-based permutation tests, which contrasted the HI and LI groups in the STOP condition with respect to time-averaged power for the frequency range of 12-30 Hz, revealed significant differences between groups. As shown in Figure 5.13.A, two significant differences between

groups were observed in this frequency range. A significant difference was observed between 0 and 500 ms after the stimulus onset (p = 0.001) in frontal regions, where the HI group showed significantly higher beta power relative to the LI group in anterior sensors**.** A second significant difference was found between 0 and 500 ms after the presentation of the STOP cue (p = 0.004), where LI participants showed significantly lower beta power than HI participants in posterior sensors.

At source-level, the statistical comparison of the HI group against the LI group, during STOP trials in beta band (13-30Hz), showed significant negative clusters in the right orbital part of medial frontal gyrus, fusiform, right middle temporal, right inferior temporal, bilateral inferior occipital and left middle occipital regions; p < .05, see Figure 5.13.C. The same cluster-based permutation testing also revealed significant positive clusters bilaterally in inferior, middle and superior frontal gyrus, including SMA and ACC, left inferior and middle frontal gyrus, middle cingulate, left inferior parietal, right middle and superior occipital, left inferior and middle occipital regions; p < .05, see Figure 5.13.C.

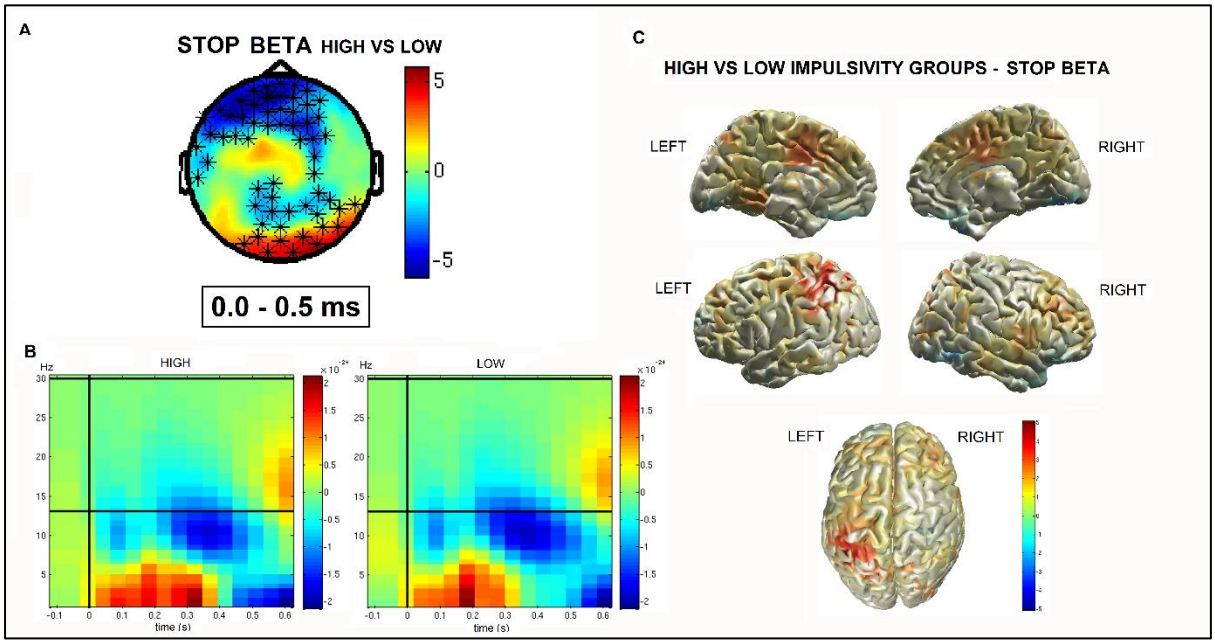

Figure 5.13. (A) Results from sensor-level analysis: topography of the statistical difference in beta power between HI and LI groups during the STOP condition, between 0 and 0.500 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and p < .05; see section 2.8.2.2 for details. As a result of contrasting the HI against the LI group, hot colours denote positive power difference, cool colours negative power difference relative to the HI group. The colour scale represents the t-values, hot for positive and cool colours for negative values; * indicates significant clusters with p < .01. **(B)** Baseline corrected TFR power plots for STOP trials in the HI and LI groups, showing delta, theta, alpha and beta bands. The y-axis denotes frequency, the x-axis denotes time in ms, t=0 and vertical black lines represent the presentation of the STOP cue, horizontal black lines limit beta band. The colour scale represents power in relation to the baseline period, power values from -2 to 2 ($x10^{-24}$). **(C)** Results from source-level analysis: the statistical differences observed in beta band power between HI and LI groups

during STOP trials, were localised in source-space, see section 2.8.3.3 for details. The colour scale represents the t-values, from -5 to 5.

5.5. DISCUSSION

In Chapter 3, the relationship between behavioural and self-report measures of impulsivity was investigated by examining how sensitive behavioural measures of impulsivity are to differences between low and high impulsivity groups, as assessed by self-reports. This experiment demonstrated that the behavioural measures used are sensitive enough to pick up differences between groups. Results also showed that these two paradigms were more sensitive to differences between groups defined by the combination of two impulsivity factors, rapid-response and reward-delay impulsivity, than groups of each dimension alone (Jauregi et al., 2018). These results were further explored in Chapter 4, by conducting an exploratory factor analysis on all the impulsivity measures tested in Chapter 3, behavioural and self-report measures (Jauregi et al., *under review*). Results suggested that rapid-response impulsivity and reward-delay impulsivity are two major yet different dimensions which contribute independently to the multifaceted nature of the impulsivity construct. Findings from the two analyses of the behavioural study showed that the measures of impulsivity tested here are adequate to investigate impulsivity further.

Considering the behavioural differences found between high and low impulsivity groups, the primary aim of the current experiment was to examine these behavioural differences in response inhibition using MEG. This was conducted in a healthy undergraduate student population, divided by their level of impulsivity as measured by questionnaires, see section 2.4.4 for details.

ERP hypotheses

Firstly, it was hypothesised that because impulsive individuals have been found to be impaired in both NOGO and STOP conditions than controls (Bari & Robbins, 2013), the HI group would show reduced task performance in the response inhibition task than the LI group. As expected, the HI group showed reduced performance in both NOGO and STOP trials than the LI group, but reaction times to the STOP cue were not significantly different between the groups.

Secondly, regarding both NOGO and STOP trials, it was hypothesised that the HI group would show a reduced M1 amplitude in both conditions in visual areas. Results from sensor-level analysis indicated that the amplitudes of the NOGO-M1 and STOP-M1 components in visual areas during were significantly smaller in the HI than in the LI group. Source-level analysis of the NOGO-M1 component revealed a significant negative cluster located in the right inferior

parietal region, see Figure 5.1. Source analysis of the STOP-M1 component also showed a significant negative cluster located in the right inferior parietal region, see Figure 5.2.

Reduced NOGO-M2 and STOP-M2 amplitudes were expected in the HI group compared to the LI group. Sensor-level analysis showed that the amplitude of the NOGO-M2 component was significantly smaller in the HI group in posterior sensors. Source-level analysis of the NOGO-M2 component revealed significant negative clusters located in right parietal, frontal and temporal regions. Source-level analysis of the NOGO-M2b component revealed significant negative clusters located bilaterally in the frontal region, along with right parietal and temporal regions, see Figure 5.1.

The peak amplitude of the STOP-M2 component happened earlier in the LI (STOP-M2L) than in the HI group (STOP-M2H) in central anterior and posterior sensors. Source analysis of the STOP-M2L component showed significant negative clusters located in bilateral fronto-parietal and right inferior parietal regions, see Figure 5.3. The STOP-M2H showed significant clusters located in right fronto-temporal, right inferior parietal, left superior parietal and left frontal regions, see Figure 5.4.

Regarding the M3 component, significantly smaller amplitudes in the HI group were predicted. Contrary to our expectations, differences between groups in the NOGO-M3 component were not found, while the amplitude in the STOP-M3 was significantly larger in the HI group in posterior sensors. Source-level analysis of the STOP-M3 showed significant positive clusters located in bilateral frontal, parietal and temporal regions, see Figure 5.5. These results favour the compensatory strategy proposed in previous studies (Dimoska & Johnstone, 2007; Lansbergen et al., 2007).

TFR hypotheses

It was hypothesised that during NOGO trials, the HI group would show decreased low frequency (delta and theta) power in frontal regions, reflecting a deficit in executive control, i.e. inhibitory processing, compared to the LI group. It was also hypothesised that high impulsivity individuals would show decreased alpha, suggesting poor attentional processing of the stimulus which might affect the inhibition process, and decreased beta power, reflecting less motor inhibition, compared to the low impulsivity group. As predicted, the HI group showed significantly lower delta, theta, alpha and beta band power in anterior sensors, conversely, this group also showed significantly increased power across the four frequency bands in posterior sensors, than the LI group. These findings were partially confirmed at source-level, where the HI group showed significant negative clusters compared to the LI group in frontal regions, see Figures 5.6, 5.7, 5.8 and 5.9.

Based on results from studies examining the SST, decreased delta, theta and beta power were hypothesised to be found in the HI group. Although less is known regarding the oscillatory activity during the SST compared to the GNGT in other frequency bands, because both paradigms are believed to measure response inhibition, similar differences between groups were expected during STOP trials: decreased power in the HI group in the four frequency ranges. The HI group showed significantly lower delta and theta power in frontal sensors, and significantly higher delta and theta band power in posterior sensors, compared to the LI group. Significantly decreased alpha and beta power was also observed in the HI group in frontal and central sensors, and significantly increased alpha and beta power in posterior sensors, compared to the LI group. These findings were confirmed at source-level, where the HI group showed significant negative clusters compared to the LI group in frontal regions, along with significant positive clusters in posterior regions, see Figures 5.10, 5.11, 5.12 and 5.13.

5.5.1. Differences in response inhibition: Event-Related Fields (ERFs)

NOGO-M1

The sensor-level results showed that the amplitude of the NOGO-M1 component (M denotes magnetic field), between 125 and 190 ms after stimulus onset, was significantly smaller in the HI group than in the LI group in parieto-occipital sensors. The NOGO-M1 component found here resembles the NOGO-N1 effect reported in previous MEG and EEG studies, occurring between approximately 100 and 200 ms after the stimulus onset. This component has been suggested to reflect the visual detection (Boehler et al., 2009) and attentional processing of stimuli (Vogel & Luck 2000) or of infrequent events (Kenemans, 2015). Specifically, researchers have suggested that larger NOGO-N1 amplitudes to certain cues compared to neutral stimuli might reflect the amount of attention towards the stimuli (Gao et al., 2019). Here, it was hypothesised that those with enhanced attention towards the NOGO stimulus would show better task performance, as a better attention might facilitate the following process, in this case, the inhibition of the motor response. As expected, the HI group committed significantly more errors during NOGO trials than the LI group, hence, exhibited reduced task performance. The significantly smaller NOGO-M1 amplitude found in the HI group suggests that the visual detection of the NOGO stimulus might be reduced in HI individuals than in LI individuals.

Source-level analysis revealed a significant negative cluster for the HI-LI group comparison in parietal regions, which is in line with the results at sensor-level, see Figure 5.1. The comparison of HI and LI groups further showed significant negative clusters located in the

frontal gyrus, right inferior and middle temporal, right superior temporal pole and right inferior parietal regions. These findings suggest that high impulsivity individuals show diminished attentional processing of the stimuli, as reflected by their significantly smaller M1 amplitudes compared to low impulsivity individuals.

STOP-M1

The analysis of the STOP-M1 component showed a significant difference between groups in parieto-occipital areas, between 105 and 170 ms after the presentation of the STOP cue, see Figure 5.2. The amplitude of the STOP-M1 was smaller in the HI group compared to the LI group, as hypothesised. This component resembles the STOP-N1 effect reported in some studies (e.g., Bekker et al. 2005; Boehler et al., 2009), found to happen between 140 and 160 ms in occipito-temporal visual areas, but which has not been found in other studies (e.g., Castro-Meneses et al., 2016; Kenemans, 2015).

The STOP-N1 amplitude has been previously reported to be larger in high impulsivity individuals compared to low impulsivity individuals, as measured by the Eysenck's Impulsiveness/ Venturesomeness/Empathy Questionnaire (Dimoska & Johnstone, 2007). The authors suggested that the enhanced STOP-N1 reflected sensation seeking behaviour in HI individuals (Dimoska & Johnstone, 2007). Although the current findings contrast with these two studies, results here are in line with other studies, in which the NOGO-M1 component has been suggested to reflect visual detection (Boehler et al., 2009) and attentional processing of stimuli (Vogel & Luck 2000); which could mean these visual and attentional processes might be diminished in HI individuals compared to LI individuals.

One of the few MEG studies that has investigated the Stop-Signal Task in healthy individuals showed that failure or success on stop trials depended on the amplitude of the STOP-N1 (Boehler et al., 2009). Specifically, the negative deflection generated by the STOP cue, the STOP-N1, showed a larger amplitude for successful than for unsuccessful trials, suggesting that heightened processing of the STOP cue is also related to success in stopping (Boehler et al., 2009). Here, the reduced STOP-M1 amplitudes found in the HI group are in accordance with those findings, as a reduced processing of the STOP cue in HI individuals might partly explain their impaired performance in response inhibition tasks compared to LI individuals. Source-level analysis of the STOP-M1 revealed a significant negative cluster located in the right inferior parietal region, which is in line with the results from sensor-level analysis. Overall, the present results are in line with previous EEG and MEG studies using response inhibition paradigms (e.g.., Boehler et al., 2009; Dimoska & Johnstone, 2007).

Conclusions from the analyses of NOGO-M1 and STOP-M1

As hypothesised, results from sensor-level analysis indicated that the amplitudes of the NOGO-M1 and STOP-M1 components in visual areas were significantly smaller in the HI than in the LI group. In line with these results, source-level analysis of the NOGO-M1 component revealed a significant negative cluster located in the right inferior parietal region among other regions. Results here are in line with previous studies, in which the NOGO-M1 component has been suggested to reflect attentional processing of stimuli (Vogel & Luck 2000). Current findings suggest that attentional processes might be diminished in HI individuals compared to LI individuals.

NOGO-M2

The amplitude of the NOGO-M2 component found here between 210 and 265 ms after stimulus onset, was significantly reduced in the HI group relative to the LI group, in posterior sensors, see Figure 5.1. These results are in line with previous studies (e.g., Falkenstein et al., 1999; van Boxtel et al., 2001), which reported a similar NOGO-N2 component between 140 and 300 ms after stimulus onset. A larger NOGO-N2 amplitude in participants with better task performance compared to those with reduced performance has been reported (Falkenstein et al., 1999; van Boxtel et al., 2001). Therefore, the HI group was expected to show a significantly reduced amplitude. Our current results add further evidence to those from previous studies, which have suggested the NOGO-N2 component might serve as a measure of response inhibition efficiency (Schmiedt-Fehr & Basar-Eroglu, 2011). Source-level analysis of the NOGO-M2 component revealed significant negative clusters located bilaterally in the superior and medial frontal gyrus, right inferior parietal, left temporal and bilateral occipital regions. Considering that the HI group showed reduced task performance and a reduced NOGO-M1 component, results suggest high impulsivity individuals are less efficient in the (pre-) motor inhibition process than the low impulsivity individuals.

NOGO-M2b

At sensor-level, a significant difference between groups was also found in the continuation of the NOGO-M2 component in posterior sensors, see Figure 5.1. This NOGO-M2b component (between 270 and 410 ms after stimulus onset) continued to decrease in both the LI and HI groups. As in the NOGO-M2 effect the amplitude of this component in the HI group was significantly smaller than in the LI group in parieto-occipital sensors. Source-level analysis of the NOGO-M2b component revealed a significant negative cluster for the HI group in the SMA,

right inferior and superior parietal region, right temporal pole and right inferior temporal region, which is in line with the results from sensor-level analysis.

Not many studies have investigated the differences in the neural correlates of the GNGT between individuals scoring high or low on impulsivity questionnaires. Studies in which differences within the NOGO-N2 component have been reported have shown that in impulsive-violent offenders, for example, the NOGO-N2 amplitudes were significantly smaller than those reported for controls (Chen et al., 2015). In a study by Ruchsow et al. (2008a), however, no significant differences were found in the NOGO-N2 amplitudes between HI and LI individuals, as measured by reaction time residual scores. Current results are in line with those of Chen and colleagues (2015), as the NOGO-N2 amplitudes were significantly smaller in the HI group.

STOP-M2

The STOP-M2 component in the present study happened earlier in the LI group (STOP-M2L, between 230 and 255 ms post-cue) than in the HI group (STOP-M2H, between 280–330 ms post-cue), located in central anterior and posterior sensors in both groups, see Figures 5.3 and 5.4. Source-level analysis of the STOP-M2L component revealed significant negative clusters located in the right the supplementary motor area and bilaterally in medial superior frontal gyrus. For the STOP-M2H component, the source-level comparison of HI and LI groups showed significant positive clusters in the right fronto-temporal region and left frontal and parietal regions. Specifically, the positive clusters were located bilaterally in the superior frontal gyrus, including the left orbital part, left middle frontal gyrus, left anterior cingulate and left middle cingulate, bilateral precuneus and bilateral superior parietal regions.

Previous studies have shown the N2 peaks earlier in successful trials compared to unsuccessful trials (Ramautar et al., 2004; 2006), which suggests that an early N2 component might be associated with successful inhibition. The current findings are in line with these studies, as low impulsivity individuals showed an earlier STOP-M2 component than HI individuals.

As a larger amplitude in the STOP-N2 component has been reported in successful inhibition compared to failed inhibition, the STOP-N2 component has been suggested to represent increased inhibitory activity (Schmajuk et al., 2006). However, in children with ADHD, the STOP-N2 amplitude was found to be reduced compared to the same component in healthy children (Dimoska et al. 2003). A larger STOP-N2 amplitude has also been reported in participants that display better inhibition performance (van Boxtel et al. 2001). Considering these findings, reduced STOP-N2 amplitudes were predicted in the HI group, however, the amplitudes were not significantly different.

Taking into consideration the differences between groups in the timing of the STOP-M2, it is possible that in individuals scoring high on impulsivity, the network involved in the inhibition process is engaged later. The reduced processing of the STOP cue, as reflected by the smaller STOP-M1 amplitude reported here in the HI group, might explain why impulsive individuals' performance is impaired in response inhibition tasks.

Conclusions from the analyses of NOGO-M2 and STOP-M2

Reduced NOGO-M2 and STOP-M2 amplitudes were expected in the HI group compared to the LI group. Sensor- and source-level analyses showed that the amplitudes of the two components were significantly smaller in the HI group in posterior sensors. Our current results add further evidence to those from previous studies, which have suggested the NOGO-N2 component might serve as a measure of response inhibition efficiency (Schmiedt-Fehr & Basar-Eroglu, 2011). Interestingly, the peak amplitude of the STOP-M2 component happened earlier in the LI (STOP-M2L) than in the HI group (STOP-M2H) in central anterior and posterior sensors. This difference was not observed in NOGO trials. Results from source-level analyses were in line with those from sensor-level, showing negative clusters in STOP-M2L, in mid-frontal and parietal regions, and in NOGO-M2, in frontal and parietal regions. Considering the evidence found for the role of the M2 component in successful motor inhibition, it could be argued that in high impulsivity individuals, there is a delay in inhibitory processing of the STOP condition.

NOGO-M3

Contrary to our expectations, a NOGO-M3 component was not found. Instead, the NOGO-M2 and -M2b components occurred between 0.210 to 0.410 seconds, overlapping the timing of the NOGO-P3. The NOGO-P3 component has been suggested to represent the evaluation of the outcome of response inhibition (Righi, Mecacci, & Viggiano, 2009; Schmajuk et al., 2006; Sehlmeyer et al., 2010) or the monitoring of successful motor inhibition (Schmajuk et al., 2006). According to Schmiedt-Fehr & Basar-Eroglu (2011), increased monitoring of the inhibition process involves a larger amplitude in the NOGO-P3 component.

HI individuals were expected to show a smaller amplitude in the NOGO-P3 component, reflecting poor monitoring or evaluation of the inhibitory process. The lack of significant differences around the NOGO-P3 component suggests the HI and LI groups were monitoring successful motor inhibition to similar degrees.

Overall, the results presented here suggest that the impairment in the Go/No-Go Task found in impulsive individuals might be specifically related to the attentional processing of the stimuli

and of the (pre-)motor inhibition process. The amplitudes of NOGO-M1, -M2 and -M2b were significantly smaller in the HI than in the LI group, while the HI group also showed significantly reduced task performance.

STOP-M3

The amplitude of the STOP-M3 component found here, between 340 and 385 ms after stimulus onset, was significantly larger in the HI group than in the LI group in posterior sensors, see Figure 5.5. It is unclear what cognitive processes the STOP-P3 is related to (Kramer et al., 2013). Some authors have suggested it might be related to the evaluation of stimuli and to the response selection process (Falkenstein, Hohnsbein, & Hoormann, 1994). This effect has previously been reported to correlate with the SSRTs in failed trials (Kok et al., 2004). Because the modulation continues after a response to the stop signal is given, it has been suggested that it might not be related to the stopping process but to the evaluation of the inhibitory process (Kok et al., 2004).

However, results from studies comparing the STOP-P3 amplitude in HI and LI individuals suggest differently. Individuals scoring high on self-report measures of impulsivity have been found to have larger STOP-P3 amplitudes than those scoring low (Dimoska & Johnstone, 2007; Lansbergen et al., 2007). The authors of these two previous studies suggested the larger amplitude in STOP-P3 in HI individuals reflects the demand for increased inhibitory effort in those scoring high as a compensatory strategy (Dimoska & Johnstone, 2007; Lansbergen et al., 2007). In contrast, Shen et al. (2014) reported the STOP-P3 amplitude to be significantly smaller in HI individuals than in those scoring low on impulsivity. Shen et al., (2014) argued that the compensatory strategy might only work in the auditory modality of the Stop-Signal Task (as in Dimoska & Johnstone, 2007; Lansbergen et al., 2007), but not in the visual modality (as in Shen et al., 2014), as the characteristics of the pre-cue attentional processing are more crucial for auditory than for visual processing.

In addition to previous findings, it was expected that because of the similarities between the Go/No-Go and Stop-Signal tasks as measures of response inhibition, the differences in NOGO-P3 and STOP-P3 amplitudes between individuals scoring high and low on impulsivity questionnaires, would follow a similar pattern. Although results on the differences in amplitude of STOP-P3 are contradictory (e.g., Dimoska & Johnstone, 2007; Lansbergen et al., 2007; Shen et al., 2014), those on the NOGO-P3 have shown the amplitude to be reduced in HI individuals compared to LI individuals (e.g., Benvenuti et al., 2015; Ruchsow et al., 2008a). Moreover, there appears to be more consensus on what the NOGO-P3 reflects, that being the evaluation/monitoring of the outcome of response inhibition (Beste, Dziobek, Hielscher, Willemssen, & Falkenstein, 2009; Righi et al., 2009; Schmajuk et al., 2006; Sehlmeyer et al.,

2010); which corresponds to what Kok and colleagues' (2004) suggest the STOP-P3 component might reflect as well.

Contrary to our expectations, the amplitude of the STOP-M3 component was significantly larger in the HI group in anterior sensors, suggesting this group engaged frontal networks significantly more than the LI group. Source-level analysis of the STOP-M3 component revealed significant positive clusters located bilaterally in the superior frontal gyrus, right anterior cingulate, SMA and left temporal regions, which is in line with the results from sensor-level analysis.

These results favour the compensatory strategy proposed in previous studies (Dimoska & Johnstone, 2007; Lansbergen et al., 2007), while refuting the possibility that this only happens in auditory stop-signal tasks, as suggested by Shen et al. (2014). These findings might also dispute whether the STOP-P3 reflects the evaluation of the inhibitory process (as suggested by Kok et al., 2004) or not. Here, no differences between groups were observed in the NOGO-P3 amplitude, in contrast to differences seen in the STOP-P3 amplitude. This finding suggests the STOP condition might be more sensitive to differences in response inhibition in HI and LI individuals than the NOGO condition.

Conclusions from the analyses of NOGO-M3 and STOP-M3

In the current experiment, significantly smaller NOGO-M3 and STOP-M3 amplitudes in the HI group were predicted. However, differences between groups in the NOGO-M3 component were not found, while the amplitude in the STOP-M3 was significantly larger in the HI group in anterior sensors. These findings also provide further evidence for task-related differences described in Chapter 1, see section 1.3.2 for details, and favour the STOP condition as a more efficient measure of response inhibition.

I believe that investigating both the NOGO and STOP conditions in the same experiment and in the same sample yielded more clarifying results, such as task-related differences, than when these have been examined separately in the past. Results regarding the debated P3 component showed differences between NOGO-P3 and STOP-P3, along with the possible dysfunctionalities in inhibitory processes in HI individuals.

Conclusions on the group differences in NOGO and STOP ERFs

In summary, from the results presented here it can be concluded that HI individuals showed reduced processing of the STOP cue, as reflected by a reduced STOP-M1 component, compared to LI individuals. Considering the differences between groups in the timing of the STOP-M2, it is suggested that in individuals scoring high on impulsivity, the network involved in the inhibition process is engaged later, resulting in more commission errors than in LI

individuals. Finally, as reflected by the larger amplitude of the STOP-M3 component in the HI group, it could be argued that this group engaged frontal networks significantly more than the LI group, possibly as a compensatory strategy.

Results from source-level analysis were consistent with previous fMRI studies, which reported activation during the NOGO condition in parietal regions (Rubia et al., 2001; Sebastian et al., 2013; Swick et al., 2011), pre-SMA (Dambacher et al., 2014; Sebastian et al., 2013; Swick et al., 2011), right superior frontal gyrus (Dambacher et al., 2014), orbitofrontal cortex (Luijten et al., 2014), rIFG and MFG (Luijten et al., 2014; Simmonds et al., 2008). Yet, no significant differences were found in the pre-SMA or ACC as in previous fMRI studies using the GNGT (e.g., Luijten et al., 2014; Simmonds et al., 2008). Regarding the STOP condition, these are also consistent, showing significant activations in the pre-SMA (Dambacher et al., 204; Sebastian et al., 2013; Swick et al., 2011), ACC (Dambacher et al., 2014), parietal regions (Rubia et al., 2001; Sebastian et al., 2013) and in the inferior parietal lobule (Swick et al., 2011). Researchers have suggested that a network of cortical and sub-cortical regions, the rIFG, pre-SMA, and the subthalamic nucleus, to be critical for successful motor inhibition (Aron, Robbins, & Poldrack, 2014), which is in line with current findings.

5.5.2. Differences in response inhibition: oscillatory activity

Delta and theta band activity

NOGO condition

An increase in delta power has been suggested to be involved in response inhibition, as it has been found to be larger in NOGO trials than in GO trials (Harper et al., 2014). Others have suggested it reflects both motor inhibition (Smith et al., 2008) and context updating (Polich, 2007). On the other hand, an increase in theta power has also been suggested to be essential for response inhibition as an integrative mechanism of early stimulus detection and response selection processes (Cavanagh & Frank, 2014; Mückschel et al., 2017). This mechanism is necessary for cognitive control processes (Cavanagh & Frank, 2014), with a greater demand during response inhibition (Mückschel et al., 2017). Previous studies comparing clinical and non-clinical populations during NOGO trials have reported a significant decrease in delta and/or theta power in individuals diagnosed with ADHD (Krämer et al., 2009), in abstinent alcohol-dependent individuals (Colrain et al., 2011; Kamarajan et al., 2004; Pandey et al., 2016), in young adults classified as binge drinkers (Lopez-Caneda et al., 2017) and in young individuals at risk for alcoholism (Kamarajan et al., 2006), compared to healthy controls. Although these studies compared individuals with ADHD and alcohol-related issues to

controls, these disorders and behaviours are also characterised by impulsivity (Pandey et al., 2016).

Thus, those with better performance in the response inhibition task, the LI group, were expected to show an increase in delta and theta power relative to the HI group. Frequency analysis at sensor-level indeed showed significantly decreased delta and theta power in anterior sensors, along with significantly increased delta and theta power in central and posterior sensors in the HI compared to the LI group. Consistent with results from sensor-level analysis, source localisation of the power difference in delta and theta bands between the HI and LI group showed a significant negative cluster in frontal regions, see Figures 5.6 and 5.7. Specifically, delta band power was significantly decreased in the superior and medial frontal gyrus in the HI group, while theta was decreased in the pre-SMA and middle, medial and superior frontal gyrus. These findings are in line with previous studies which have reported a decrease in delta and/or theta power in frontal regions, possibly reflecting a deficit in fronto-parietal networks recruited during the suppression of a motor response (Colrain et al., 2011; Kamarajan et al., 2004; Lopez-Caneda et al., 2017).

This deficit in frontal pathways might be present in impulsive individuals, who engage in maladaptive behaviours such as alcohol abuse, might predisposes them to these maladaptive behaviours (Lopez-Caneda et al., 2017). This is consistent with other studies which have proposed that weaker low-frequency oscillatory activity related to inhibitory processing, such as delta and theta, might lead to a predisposition to develop disorders characterised by disinhibition or alcohol-related disorders. This might serve as a vulnerability marker for developing certain impulsivity-related disorders (Kamarajan et al., 2006; Lopez-Caneda et al., 2017).

STOP condition

Frequency analysis at sensor-level during NOGO trials showed delta and theta band power to be significantly decreased in frontal areas in the HI group compared to the LI group, which, conforming to our hypotheses, is likely to reflect reduced inhibitory processing. Source reconstruction confirmed this finding in both delta and theta bands. In the delta band specifically, significant negative clusters relative to the HI group were found in the ACC, pre-SMA, right middle frontal gyrus, left parietal and left temporal regions, see Figure 5.10. A recent EEG study using CSD found increased mid-frontocentral theta and increased delta in posterior sensors during the STOP condition (Lockhart et al., 2019), which is in line with results from source-level.

NOGO and STOP delta and theta activity: Conclusions

Power decrease in delta and theta band power was observed in high impulsivity individuals compared to low impulsivity individuals in frontal regions during both NOGO and STOP conditions. This finding is consistent with previous studies and provides further support for a possible deficit in frontal pathways involved in the suppression of a motor response in individuals scoring high on impulsivity.

Alpha band activity

NOGO condition

Increased alpha activity has been associated with response inhibition during NOGO trials (Nakata et al., 2013). As in delta and theta oscillatory activity, alpha band power has previously been found to be significantly reduced in the GNGT in young individuals at risk for alcoholism (Kamarajan et al., 2006) and in abstinent alcohol-dependent adults (Pandey et al., 2016). Thus, similar findings were expected here. Current findings show that alpha power significantly decreased in the HI group compared to the LI group in frontal areas, see Figure 5.8. A strong increase in alpha power is observed in LI participants, which is minimal in HI individuals.
Alpha activity has been associated with attentional processing (Basar et al 1997; Klimesch et al 1998). Thus, lower alpha power previously found in alcohol-dependent individuals and in those at risk for alcoholism has been suggested to be related to poor early attentional processing of the stimulus which might affect the inhibition process (Pandey et al., 2016).

Frequency analysis of alpha band power in source-space revealed significant negative clusters in the superior frontal gyrus and right superior parietal region in the HI group, compared to the LI group. The same cluster-based permutation testing also revealed significant positive clusters in the orbital part of medial frontal gyrus, right parietal and temporal regions in the HI group, suggesting more posterior attentional processing.

STOP condition

Although little is known about alpha band in the STOP condition, other response inhibition tasks such as the GNGT, have shown alpha power to be significantly reduced in young individuals at risk for alcoholism (Kamarajan et al., 2006) and in abstinent alcohol-dependent adults (Pandey et al., 2016). Moreover, results in alpha band during NOGO trials presented here are also consistent with such findings. This decrease in alpha power has been suggested to reflect an early attentional deficit that might affect the inhibition process (Pandey et al., 2016).

Here, alpha power was found to be significantly lower in the HI group compared to the LI group in anterior sensors, reflecting poor attentional processing in HI participants, as hypothesised,

see Figure 5.12. However, increased alpha power was also observed in posterior sensors in HI participants, while in LI participants alpha power decreased in the same region, as in the NOGO condition. In an EEG study, adolescents diagnosed with ADHD showed less posterior alpha suppression over occipital electrodes, as reflected by alpha power increase, than typically developed adolescents in another response inhibition task, a flanker task (Mazaheri et al., 2014). This increase in alpha power has been associated with reduced cue processing (Mazaheri et al., 2014), which suggests high impulsivity individuals might also show reduced processing of the NOGO and STOP cues.

Results from source-level analysis were in line with sensor-level findings, as decreased alpha power in frontal regions and increased power in temporal and parietal regions in HI compared to LI group were observed. These findings are consistent with results from sensor-level analyses and with previous literature (Kamarajan et al., 2006; Mazaheri et al., 2014; Pandey et al., 2016), strengthening the possibility of an attentional or cue processing deficit.

NOGO and STOP alpha activity: Conclusion

The results presented here suggest that in individuals scoring high on impulsivity, impairment in response inhibition might be a consequence of a deficit in the inhibitory process itself. This is reflected by decreased delta and theta power, along with reduced alpha suppression, which suggests diminished processing of the NOGO and STOP cues. Although comparisons between failed and successful stopping trials were not possible in the current experiment, see section 2.8.1 for details, results are in line with previous literature, providing further evidence for a possible attentional processing deficit. Current findings indicate that decreased anterior and increased posterior alpha activity during STOP and NOGO trials might characterise high impulsivity individuals.

Beta band activity

NOGO condition

Little is known about potential differences in beta band activity between those with behaviours or disorders characterised by impulsivity and controls, as in those studies in which it has been investigated, no significant differences were found during NOGO trials (e.g., Kamarajan et al., 2006; Krämer et al., 2009). As NOGO trials have shown beta band power to increase over frontal areas in healthy individuals, it has been suggested it might also represent inhibitory processes (Alegre et al., 2004). Considering this finding, it was expected that those with better task performance, LI participants, would show increased beta power relative to HI participants in frontal sensors and associated brain areas. As hypothesised, the HI group showed

significantly lower beta power than the LI group in frontal sensors during NOGO trials, see Figure 5.9. To our knowledge, this is the first study that has reported a significant decrease in frontal beta power in individuals characterised by high impulsivity compared to those scoring low on impulsivity measures. This result is consistent with Alegre et al. (2004), in that an increase in beta power in frontal areas during NOGO trials reflects motor inhibition. It is however noteworthy that this lack of frontal increase was accompanied by a stronger positive cluster in posterior sensors.

Source-level analysis of the differences in beta band power between HI and LI groups showed significant negative clusters located in middle occipital regions, right anterior cingulate, right medial and middle frontal gyrus. The same cluster-based permutation test also revealed significant positive clusters in right inferior, middle and superior temporal regions, left pre-supplementary motor area, left medial part of superior frontal gyrus, left temporal, left occipital and left parietal regions. In contrast to results from sensor-level analysis, beta band power in source-space was predominantly increased in high impulsivity compared to low impulsivity individuals. Although this finding was not expected, similar results were found during both conditions in all four frequency bands, and beta band is no exception. The increased beta power in high compared to low impulsivity individuals found during the NOGO and STOP conditions suggests that beta might be involved in response inhibition in a different way to delta, theta and alpha bands.

STOP condition

As discussed for NOGO, previous studies have shown changes in beta oscillatory activity to be related to motor execution and inhibition (Neuper et al., 2006). Specifically, an increase in beta during successful STOP trials compared to GO trials and unsuccessful STOP trials has been reported (Marco-Pallares et al., 2008). In other EEG studies, this increase in beta power was reported in frontal electrodes in successful STOP trials (Alegre et al., 2008), while others have found this difference in central areas (Krämer et al., 2011). As these results suggest inhibitory control might be associated with increased beta power, decreased beta power was expected in the HI group compared to the LI group, reflecting impaired motor inhibition. At sensor-level, HI participants showed significantly decreased beta power in anterior and central-posterior sensors relative to LI participants during STOP trials, in line with results during NOGO trials and with what was hypothesised.

Source-level analysis of differences in beta band power showed significant negative clusters in the right orbital part of medial frontal gyrus, fusiform, right inferior and middle temporal, and bilateral occipital regions in the HI group, see Figure 5.13. The same cluster-based permutation testing also revealed significant positive clusters in the inferior, middle and

superior frontal gyrus, including the pre-SMA and ACC, left parietal and bilateral occipital regions. As in the NOGO beta power analysis and in contrast to results from sensor-level analysis, beta band power in source-space was predominantly increased in high impulsivity compared to low impulsivity individuals.

Current findings in source-space contrast with previous studies which suggest there is an association between increased beta power and successful stopping, when compared to beta power in unsuccessful STOP trials (Swann et al., 2009; 2012). However, other authors (e.g., Engel & Fries, 2010) have concluded that increased beta band activity in inferior frontal and primary motor regions in STOP trials compared to GO reflects the prolongation of the oscillatory activity present before the display of the STOP cue; which the GO trial changes (Swann et al., 2009; 2012). These studies also reported increased beta band activity in inferior frontal and primary motor regions in STOP trials compared to GO trials (Swann et al., 2009; 2012). Other neuroimaging studies have also reported changes in beta oscillatory activity in the IFC and preSMA to be related to motor execution and successful inhibition (Huster et al., 2013). In the current experiment, changes were found in these areas, specifically, an increase in beta power was found in the inferior frontal gyrus and pre-SMA in high impulsivity individuals.

NOGO and STOP beta activity: Conclusions

As hypothesised, sensor-level frequency analysis revealed significantly decreased beta band power in frontal sensors in the HI group compared to the LI group. Source localisation of the power difference between the HI and LI groups showed in both conditions a decrease in delta, theta and alpha bands predominantly in left frontal regions in high impulsivity individuals, which is in line with what was observed in sensor-space. These findings are consistent with previous studies, which suggested an increase in beta power in frontal areas during NOGO trials to reflect motor inhibition (Alegre et al., 2004), as an increase in beta power in low impulsivity individuals was expected. Sensor-level analysis of beta band power also showed significant positive clusters in the HI group compared to the LI group, in central and posterior sensors during NOGO trials and in posterior sensors during STOP trials. However, results at source-level showed that the difference of beta power during NOGO and STOP conditions was predominantly positive for the HI group. Although this finding was not expected, similar results were found during both conditions in all four frequency bands, and beta band is no exception. The increased beta power in high compared to low impulsivity individuals found during the NOGO and STOP conditions suggests that beta might be involved in response inhibition in a different way to delta, theta and alpha bands. More research on beta band power during response inhibition tasks is required to clarify what characterises impulsive individuals in beta band during inhibitory processing.

5.6. CONCLUSIONS

This experiment investigated the neural correlates of response inhibition using MEG in a healthy undergraduate student population, divided by their level of impulsivity as assessed by self-report measures. Current results indicate that the impairment found in these two tasks in impulsive individuals might be related to reduced attentional processing of NOGO and STOP stimuli. During NOGO trials, results suggest that the (pre-)motor inhibition process might be less efficient in impulsive individuals. Meanwhile in STOP trials, the network involved in the stopping process is engaged later in high impulsivity than in low impulsivity individuals. Findings regarding the late positive component commonly found in both NOGO and STOP trials, suggest the high impulsivity group engaged frontal networks significantly more than the low impulsivity group, possibly as a compensatory strategy, during STOP trials only; no differences were found in this component during NOGO trials.

Current findings suggest that the decreased oscillatory activity observed in delta and theta frequency bands during the Go/No-Go and Stop-Signal tasks in impulsive individuals, may indicate that the neural mechanisms underlying cognitive control processes might be less efficient, especially during response inhibition, compared to low impulsivity individuals. High impulsivity individuals also showed significantly reduced alpha suppression compared to low impulsivity individuals, which suggests diminished processing of the NOGO and STOP cues. Although this study is based in a young adult non-clinical population, these results illustrate how personality traits such as impulsivity relate to differences in the neural correlates of cognitive processes.

CHAPTER 6: MEG EXPERIMENT 2: Sensor- and source-level analyses of event-related fields in delay discounting.

6.1. CHAPTER AIMS

Findings from the behavioural and MEG studies described here show that the measures of impulsivity tested are adequate to investigate impulsivity further. Considering the behavioural (Chapter 3) and neural (Chapter 5) differences found between high and low impulsivity groups, the primary aim of the current chapter was to examine these differences in reward delay-discounting using MEG. This was conducted in a healthy undergraduate student population, which was divided by their level of impulsivity as measured by questionnaires, see section 2.4.4 for details. Here, the MEG data of sensor- and source-level analyses of event-related fields and oscillatory activity in delay discounting is presented, followed by a brief discussion of the results.

6.2. INTRODUCTION

Researchers have suggested that an increase in reward-seeking actions and poor cognitive control might serve as a marker of risk for addictive behaviours in teenagers (Li et al., 2019). It could be argued that inhibitory deficits and excessive reward-delay sensitivity represent major contributors towards impulsivity, as both are necessary to precisely conceptualise impulsivity, as seen in Chapter 4. Yet, compared to the number of neuroimaging studies investigating response inhibition, studies specifically examining reward-delay impulsivity are very few.

Reward-delay impulsivity can be measured using reward-directed tasks or decision-making tasks, but more precisely, using delay discounting paradigms, which require participants to choose between either smaller but immediate or larger, but delayed rewards. As the delay until the reward is obtained increases, the value of a future reward given by an individual, decreases. This is known as temporal discounting (Ainslie, 1975). The delay discounting task (DDT) by Kirby and colleagues (1999) for example, consists of hypothetical choices between smaller immediate rewards and larger but delayed rewards, which allows the examination of temporal discounting, see section 1.2.3.1 for details

During the DDT, activity in the anterior mPFC has been found to inversely correlate with trait-impulsivity as measured by BIS-11 scores (Sripada et al., 2011). Similarly, a study by Luhmann et al. (2008) reported a negative correlation between participants' higher rates of impulsive choices on the DDT and activity in the mPFC. Correlations between activity in the ventral striatum and trait-impulsivity, as measured by BIS scores (Forbes et a., 2009), and temporal discounting rates have also been previously reported (Hariri et al., 2006). These

findings suggest that the ventral striatum (VS) and medial prefrontal cortex (mPFC) might be directly related to trait impulsivity and reward-delay impulsivity, along with other active areas.

Analysing each of the three DDT conditions, immediate reward, future reward magnitude and future reward delay, has produced interesting results. Specifically, these studies have reported the mPFC, PCC and VS to be active during the presentation of the immediate reward condition (Tanaka et al., 2004; Wittmann et al., 2007) and during the presentation of the magnitude of the future reward (Ballard & Knutson, 2009). The future reward magnitude condition has also implicated the nucleus accumbens (NAcc; Sripada et al., 2011), while brain activity during future reward delay condition remains unspecified.

Although a major part of the literature on this topic has come from studies using fMRI, MEG has been proven to be a useful technique for both clinical (Stufflebeam, 2011) and research (e.g., Boehler et al., 2009; Takei et al., 2010; Vidal et al., 2012) objectives. Considering that the objective here is to investigate the spectral and spatio-temporal dynamics of response inhibition and delay discounting, the better spatial resolution of MEG when coupled with individual MRI data and the millisecond temporal resolution, make MEG ideally suited for this purpose.

6.2.1. Event-Related Fields (ERFs)

Electrophysiological studies have reported larger evoked responses in occipital regions during the presentation of reward cues compared to the presentation of non-reward cues between 100ms and 155ms after stimulus onset (M100 component, e.g., peaking at 100ms in Apitz & Bunzeck, 2012; M150 component, peaking at 155ms in Thomas, Vanni-Mercier, & Derher, 2013). The amplitude of this early component during early visual processing has been suggested to be influenced by the magnitude of the reward presented (Doñamayor et al., 2012).

A later effect on reward cues compared to non-reward cues, at approximately 170ms after stimulus onset, has also been observed in the fusiform gyrus (M170 component, Apitz & Bunzeck, 2012). Interestingly, the amplitude of this later response has also been found to be larger in the context of reward compared to non-reward contexts (Apitz & Bunzeck, 2012). Considering results from Chapter 5, in which the M1 amplitudes were significantly reduced during both NOGO and STOP conditions in the HI compared to the LI group, reflecting a potential attentional deficit, I would expect to find a similar result here.

ERP studies using monetary gambling tasks to investigate reward processing, have generally found two main components: a negative component at around 200-250 ms, usually referred to as feedback-related negativity (FRN) or N2, and a positive component at around 300-500

ms, usually referred to as feedback-related positivity (FRP) or P3 (Kamarajan et al., 2009; 2015b). The N2 component has been reported to index an initial response to primary stimulus attributes (Bernat et al., 2015). During the delay discounting task, a P2 component has also been reported (e.g., Gui et al., 2016; He et al., 2012) and is considered to represent the first evaluation of reward magnitude and reward delay (Gui et al., 2016). It has also been suggested that it represents the evaluation and assessment of the stimulus (Potts et al., 2006).

Depending on the delay of the future magnitude, increasing from 2 weeks to 50 years, the amplitudes of P2 and P3 have been found to also change (He et al., 2012). Together with the P2, a frontal N2 component has also been reported to be associated with preference for immediate rewards, some authors consider it to be the key component in temporal dynamics of the interaction between reward and time valuation (Gui et al., 2016; 2018).

Regarding the P3 component, Kamarajan et al. (2015b) using a monetary gambling task found that individuals at high risk for alcoholism showed significantly lower P3 amplitudes than the low risk group during reward processing. Similar results have been reported during the presentation of reward-related cues in alcoholics (Kamarajan et al., 2010). Altogether, the P3 attenuation has been suggested to serve as an index of reward processing, representing dysfunctional reward processing (Kamarajan et al., 2015b). Using surface Laplacian (current source density maps; CSD), weaker current density in the high-risk for alcoholism group at the frontal sources has been reported, which has been suggested it might explain the decreased P3 amplitudes found in that group (Kamarajan et al., 2015b).

As described, previous studies have found M1, N2, P2 and P3 components during the delay discounting task (e.g., Gui et al., 2016; He et al., 2012). The DDT can be used to examine the sensitivity to an immediate reward, the sensitivity to a delay of future rewards and the sensitivity to the magnitude of a reward. These specific processes cannot be examined when using other reward-delay tasks, mainly because of their task design. Although other reward-related paradigms, such as the MID or card guessing paradigm described in section 1.2.3.1, have been used to examine the reward process, their objective is to examine the neural processes produced by the decision and feedback phases (Nusslock et al., 2014). It could be argued that individuals who prefer immediate rewards generally know that the consequences of their immediate choices are often negative, but they might not be able to avoid choosing the immediate option in many occasions. That is why the focus here is on intertemporal choice in impulsive individuals, see section 1.2.3.1 for details. Consequently, in the current work the neural correlates involved in temporal discounting were specifically examined, that being the processing of the three conditions separately: immediate reward, the magnitude of the future reward and the delay of the future reward. Because the DDT allows these examinations, it

might be a paradigm with great potential to measure reward-delay impulsivity with MEG, a technique with much better temporal resolution than fMRI, which allows tracking of real-time variations in cortical activity underlying the signal processing happening in the brain.

Considering previous findings, differences between high and low impulsivity individuals in temporal discounting are expected. Reduced M1 amplitudes in the HI group compared to the LI group were observed in Chapter 5, reflecting a potential attentional deficit, which I expect to find here too. The P2 component, has been reported to reflect the first valuation of the reward magnitude and delay (Gui et al., 2016). This suggests the reward evaluation process might occur differently in high impulsivity individuals and be reflected by larger P2 amplitudes compared to low impulsivity individuals. The frontal N2 component previously found to correlate with immediate rewards (Gui et al., 2016) is also a potential candidate for group differences, as a stronger frontal N2 component can be expected in high impulsivity individuals, reflecting an increased preference for immediacy. Finally, the reduced P3 amplitudes reported in previous studies in individuals with impulsive characteristics (Kamarajan et al., 2010; 2015b) suggests that individuals scoring high on impulsivity would show reduced P3 amplitudes compared to those scoring low.

6.2.2. Time-frequency representations (TFRs)

As previously described (see section 6.2.1), electrophysiological studies on reward processing have shown ERPs to be characterised by two components, a negative one between 200 and 250 ms after stimulus onset, referred to as N2 or feedback-related negativity, and a positive one between 300 and 500 ms after stimulus onset, referred to as P3 or feedback-related positivity. While some studies have reported theta oscillations to predominantly contribute to both N2 and P3 components (e.g., Kamarajan et al., 2008; 2015; Cohen et al., 2007), others have reported theta to be associated with N2 and delta with P3 (Bernat et al., 2011; 2015; Harper et al., 2014).

The N2 component, specifically, has been reported to index an initial response to primary stimulus attributes (Bernat et al., 2015) and to exhibit an increase in theta power following the presentation of reward cues compared to the presentation of non-reward cues (Doñamayor et al., 2012). The P3 component on the other hand, has been found be sensitive to reward magnitude, where a larger amplitude in delta was associated with larger magnitudes (Bernat et al., 2015). In this regard, Doñamayor et al. (2012) reported a decrease in beta power to be more prominent the more the magnitude of a reward decreased. Decreases in beta power have previously been associated with motor control (e.g., Neuper et al., 2006; Zhang et al., 2008). Furthermore, in Doñamayor et al. (2012), this decrease in beta power was found near sensorimotor areas and it was related to between-subject variability in reaction times and thus,

the authors of the study suggested the decrease in beta power could be related to motor preparation.

In a study by Kamarajan and colleagues (2015a), adolescents and young adults at high risk for alcoholism were compared with aged-matched participants at low risk during a monetary gambling task. They found subjects at high risk to show lower theta power than subjects at low risk during reward processing, specifically between 200 and 500 ms after stimulus onset, which included both N2 and P3 components (Kamarajan et al., 2015a). Individuals at high risk also showed increased impulsivity, as measured by the BIS-11, compared to individuals at low risk (Kamarajan et al., 2015a). The authors of this study suggested that, because the lower theta power was found in both gain and loss conditions of the monetary gambling task, it could be possible that individuals at high risk might struggle to evaluate the consequences of their choices (Kamarajan et al., 2015a).

Researchers agree that ADHD is associated with trait-impulsivity (e.g., BIS-11 scores, Malloy-Diniz et al., 2007), exhibiting impaired response inhibition (e.g., longer stop-signal traction time, Lijffijt et al., 2005) and higher delay discounting (e.g., Bitsakou et al., 2009; Paloyelis et al., 2010; Solanto et al., 2001). Past studies have shown an increase in theta and a decrease in beta band activity in individuals diagnosed with ADHD compared to controls during intertemporal choice (Gui et al., 2018; Loo et al., 2013; Monastra et al., 2001). The authors suggest that increased impulsivity and increased theta band activity are related, whereas inhibitory control is associated with stronger beta band activity (Gui et al., 2018).

Although there is limited knowledge of alpha band power during delay discounting, alpha power is considered to represent the inhibition of neural activity (Jensen & Mazaheri, 2010) and stronger alpha suppression has been previously reported following monetary-reward cues (Hughes et al., 2013). A previous study investigating response preparation and cue processing in adolescents diagnosed with ADHD using a cued flanker task, found significantly less alpha suppression in the visual cortex in this group compared to controls, which suggests reduced processing of the cue (Mazaheri et al., 2014). In non-clinical populations, it has been reported that after the presentation of reward-related cues in a Stroop task, individuals with increased alpha suppression over occipital regions displayed better behavioural performance on reward trials (van den Berg et al., 2014). Altogether, it has been suggested that alpha power suppression reflects increased attentional processing during the expectation of reward-related cues (Pornpattananangkul & Nusslock, 2016).

Given the inconsistent findings reported during the delay discounting task (as in theta band, e.g., Gui et al., 2018; Kamarajan et al., 2015a), I believe that the literature on this topic would benefit from the use of MEG. This technique could effectively determine what the differences

in the spatio-temporal neural dynamics of delay-discounting are between individuals scoring high and low on impulsivity. Furthermore, it might also aid in clarifying what processes each frequency band are reflecting during the DDT.

6.2.3. Current study

Investigating reward-delay impulsivity using the delay-discounting task (DDT) allows the specific examination of the oscillatory pattern generated by the presence of an immediate reward, the magnitude of a future reward and the delay to receive that future reward. I believe the DDT is the optimal choice for examining reward-delay sensitivity and that potential differences in power between high and low impulsivity individuals might explain what contributes to impulsive temporal discounting. The present experiment investigated the neural correlates of delay-discounting using MEG in a healthy undergraduate student population, divided by their level of impulsivity as measured by self-report measures. I expect to find differences between high and low impulsivity individuals during the DDT, adding novel MEG insights to the limited existing literature on the neural correlates of temporal discounting.

Regarding ERFs in the DDT, it was hypothesised that (1) high impulsivity individuals would show reduced M1 amplitudes compared to low impulsivity individuals, (2) in the Immediate condition, high impulsivity individuals would show larger M2 amplitudes in frontal sensors than low impulsivity individuals, (3) in the future Magnitude and Delay conditions, high impulsivity individuals would show larger M2 amplitudes compared to low impulsivity individuals, (4) high impulsivity individuals would show reduced M3 amplitudes than low impulsivity individuals.

Regarding oscillatory activity, it was hypothesised that (1) high impulsivity individuals would show more delta band power than low impulsivity individuals, (2) high impulsivity individuals would show either more or less theta band power compared to low impulsivity individuals, (3) high impulsivity individuals would show less alpha suppression than low impulsivity individuals, (4) high impulsivity individuals would show less beta band power compared to low impulsivity individuals.

6.3. METHODS

6.3.1. Participants

See section 2.3.2 for details.

6.3.2. Procedure

See section 2.4.2 for details on the MEG and MRI sessions.

6.3.3. Group assignment

See section 2.4.4 for details.

6.3.4. Stimuli

See section 2.5.4 for details on the task used.

6.3.5. Questionnaires

See sections 2.6.1 and 2.6.4 for details.

6.3.6. MEG data acquisition and analysis.

See section 2.8 for full details on how the MEG analysis was conducted at sensor- and source-level for ERFs and TFRs.

6.4. RESULTS

6.4.1. Behavioural results.

The HI group temporally discounted in a more impulsive manner than the LI group, but not significantly so (HI: Mean proportion of smaller immediate vs. larger delayed reward choices = 0.54, SD = 0.14; LI: Mean proportion of smaller immediate vs. larger delayed reward choices = 0.48, SD = 0.17; $F(32) = 1.23$; $p = 0.230$).

6.4.2. Event-related fields (ERFs) results.

High vs Low Impulsivity groups - DDT Immediate condition

Sensor-level analyses

The cluster-based permutation testing performed on ERFs revealed significant differences between HI and LI groups. The differences between groups in the immediate reward condition showed a significant negative cluster (= larger amplitudes in the LI group) in central sensors between 1.14 and 1.19 seconds after fixation (Immediate-M1; $p < .05$). A significant positive cluster (= larger amplitudes in the HI group) was also observed between 1.21 and 1.24 seconds after stimulus onset in parietal, left temporal and frontal sensors (Immediate-M2; $p < .01$). A further significant positive cluster was observed later, between 1.28 and 1.37 seconds after fixation in frontal sensors (Immediate-M3; $p < .05$). See Figure 6.1.A for details.

The group-averaged ERFs and the differences between groups observed in sensor-level analysis during the DDT Immediate condition were reconstructed and localised in source-space using an LCMV beamformer, see section 2.8.3.2 for details. The mean source activity in each group was calculated for the time intervals of interest, corresponding to Immediate-

M1, -M2 and -M3 components. HI and LI group means were compared using cluster-based permutations tests to control for multiple comparisons (Maris & Oostenveld, 2007), for each component, see section 2.8.3.2 for details.

Source-Level Analyses

The comparison of HI and LI groups of the Immediate-M1 component, HI > LI, showed significant negative clusters located bilaterally in the middle cingulate, bilateral inferior parietal, right superior parietal, right temporal and left occipital regions; $p < .05$, see Figure 6.1.C. The comparison of HI and LI groups of the Immediate-M2 component showed significant positive clusters located in the left middle and superior, right inferior and middle frontal regions, right fusiform, left temporal, bilateral occipital regions; $p < .05$, see Figure 6.1.C. The comparison of HI and LI groups of the Immediate-M3 component revealed significant positive clusters (= larger amplitudes for the HI group) bilaterally in the superior frontal gyrus, left inferior and middle frontal regions, left middle and bilateral superior temporal regions, and bilateral occipital regions,; $p < .05$, see Figure 6.1.C. Source-analysis of the Immediate-M3 component also revealed significant negative clusters (= larger amplitudes in the LI group) bilaterally in the medial part of the superior frontal gyrus, including the supplementary motor area (SMA), middle cingulate, right precentral and postcentral regions, right temporal region; $p < .05$, see Figure 6.1.C.

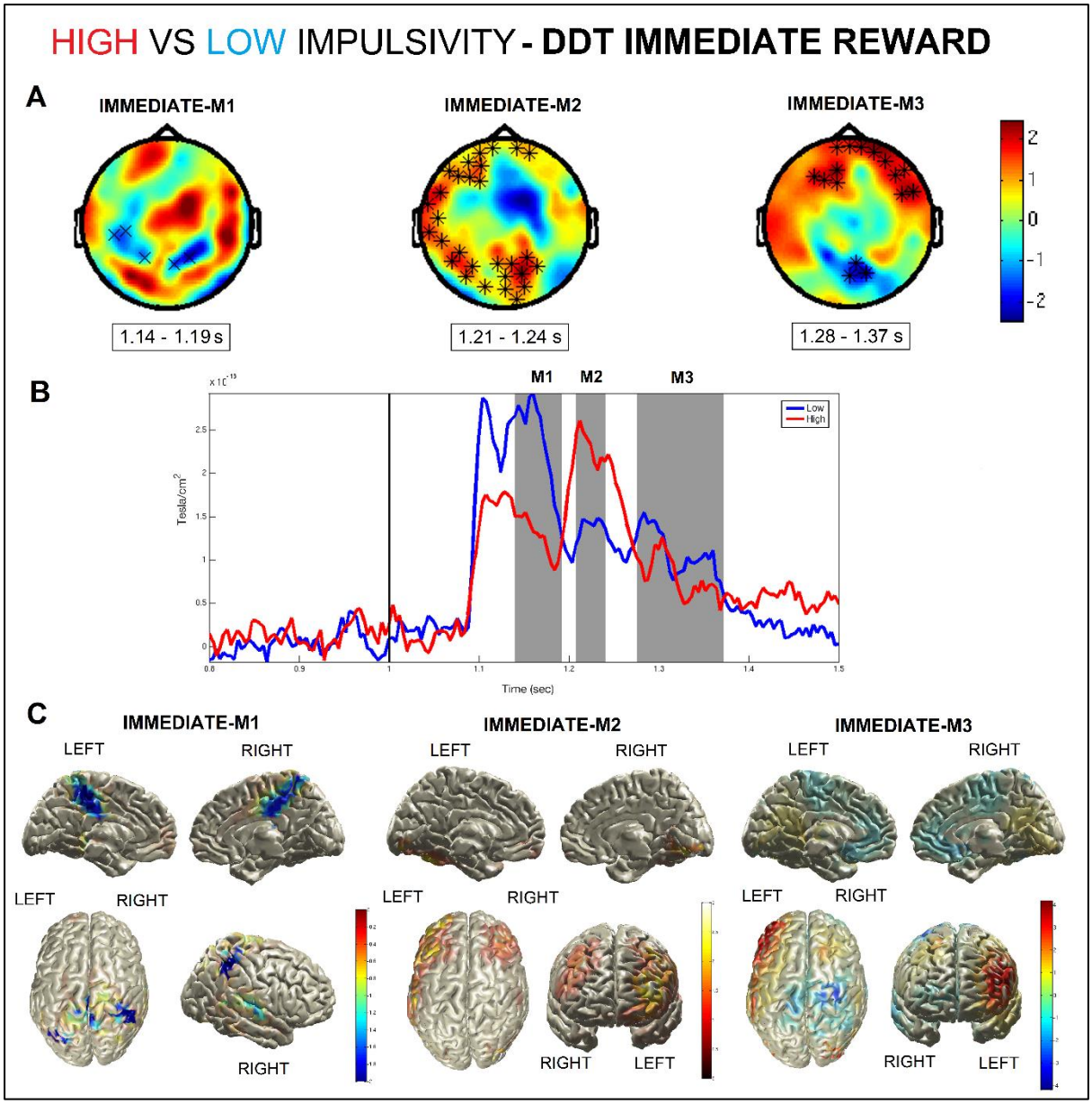

Figure 6.1. (A) Sensor-level analysis: topography of the statistical difference in amplitude between HI and LI groups during the Immediate condition, between 1 and 3 seconds after stimulus onset. *T*-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$. The colour scale represents the *t*-values, * indicates significant clusters with $p < .01$. (B) Group averages of ERFs during Immediate trials for the HI (red line) and LI (blue line) groups, the group averages were plotted from significant sensors ($p < .01$) only. The grey columns represent the statistical difference between groups from the cluster-based permutations, see section 2.8.2.1 for details. Time point 0, marked with a black line, denotes the onset of target stimulus presentation. (C) Source-level analysis: the statistical differences observed in amplitude between HI and LI groups during the Immediate condition, were localised in source-space, see section 2.8.3.2 for details. The colour scale represents the *t*-values.

High vs Low Impulsivity groups - DDT Future Magnitude condition

Sensor-level analysis

The cluster-based permutation testing performed on ERFs revealed significant differences between HI and LI groups. The differences between groups in the future reward magnitude condition showed a significant negative cluster (= larger amplitude in the LI group) in parietal sensors between 3.09 and 3.21 seconds after fixation (Magnitude-M1; $p < .01$). A significant negative cluster was also observed between 3.27 and 3.35 seconds after stimulus onset in the HI compared to the LI group, in parietal sensors (Magnitude-M2; $p < .01$). An additional significant negative cluster (= larger amplitude in the LI group) was observed later, between 3.35 and 3.48 seconds after fixation in fronto-temporal sensors (Magnitude-M3; $p < .01$). See Figure 6.2.A for details.

Source-level analysis

The group-averaged ERFs and the differences between groups observed in sensor-level analysis during the DDT future reward magnitude condition, were reconstructed and localised in source-space using an LCMV beamformer, see section 2.8.3.2 for details. The comparison of HI and LI groups of the Magnitude -M1 component, HI > LI, showed significant negative clusters located in right superior frontal gyrus, SMA, middle cingulate, left middle occipital, right middle and superior temporal, right inferior parietal and bilateral parietal regions; $p < .05$, see Figure 6.2.C. The comparison of HI and LI groups of the Magnitude-M2 component showed significant negative clusters in left middle occipital region, precentral and postcentral regions, middle temporal and right superior temporal regions; $p < .05$, see Figure 6.2.C. The comparison of HI and LI groups of the Magnitude-M3 component revealed significant negative clusters in the right middle and superior frontal gyrus, paracentral lobule, right superior temporal regions, bilateral parietal, and left occipital regions; $p < .05$, see Figure 6.2.C.

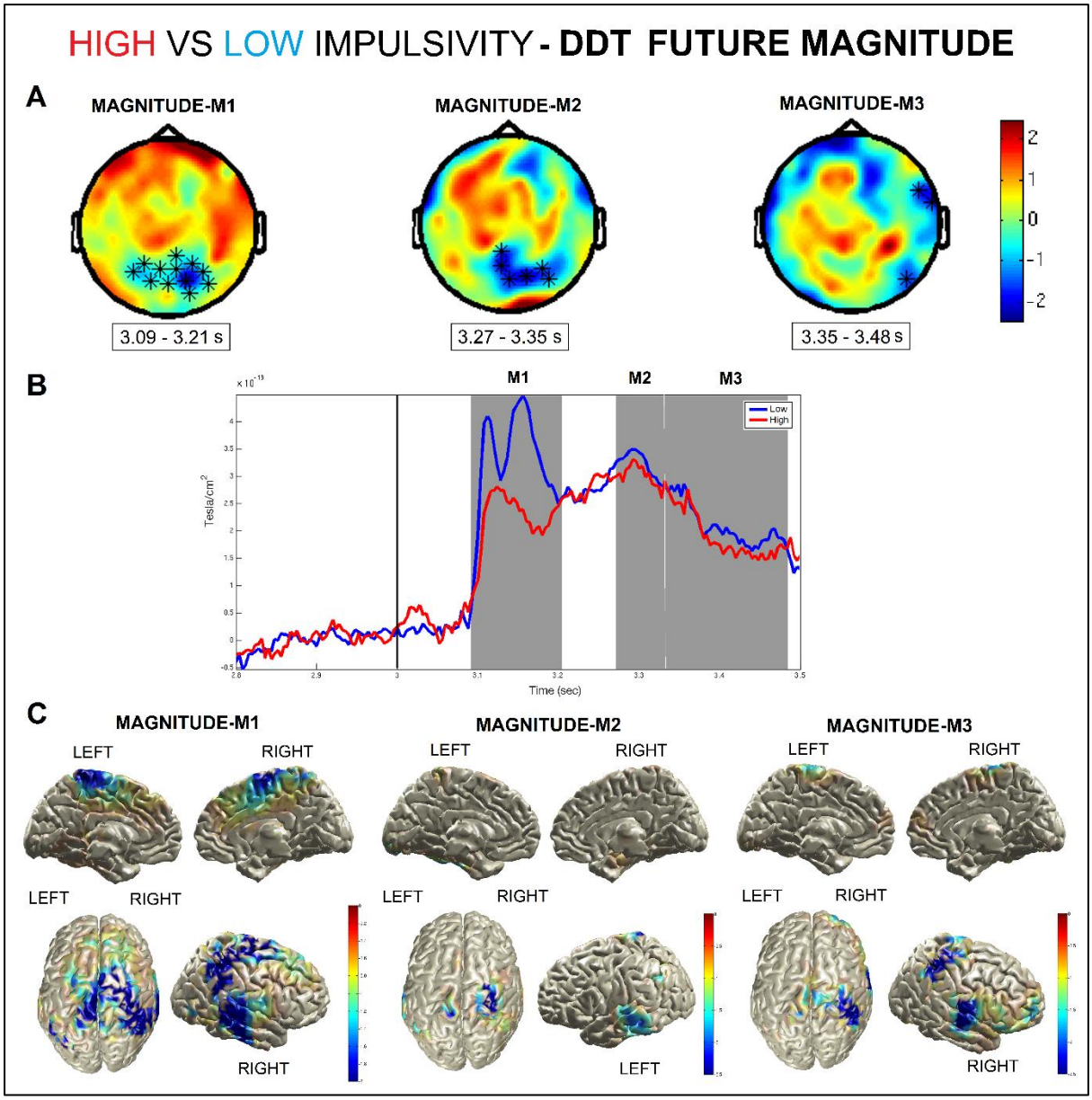

Figure 6.2. (A) Sensor-level analysis: topography of the statistical difference in amplitude between HI and LI groups during the Magnitude condition, between 3 and 5 seconds after stimulus onset. *T*-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$. The colour scale represents the *t*-values, * indicates significant clusters with $p < .01$. (B) Group averages of ERFs during Immediate trials for the HI (red line) and LI (blue line) groups, the group averages were plotted from significant sensors ($p < .01$) only. The grey columns represent the statistical difference between groups from the cluster-based permutations, see section 2.8.2.1 for details. Time point 0, marked with a black line, denotes the onset of target stimulus presentation. (C) Source-level analysis: the statistical differences observed in amplitude between HI and LI groups during the Magnitude condition, were localised in source-space, see section 2.8.3.2 for details. The colour scale represents the *t*-values.

High vs Low Impulsivity groups - DDT Future Delay condition:

Sensor-level analysis

The cluster-based permutation testing performed on ERFs revealed significant differences between HI and LI groups. The differences between groups in the future reward delay condition showed a significant negative cluster (= larger amplitude in the LI group) in parietal sensors between 5.13 and 5.21 seconds after fixation (Delay-M1; $p < .01$). Another significant negative cluster was also observed between 5.29 and 5.31 seconds after stimulus onset (= larger amplitude in the LI group), in right parietal sensors (Delay -M2; $p < .05$). Significant positive clusters (= larger amplitude in the HI group) were observed later, between 5.33 and 5.37 seconds after fixation in frontal, right temporal and occipital sensors (Delay-M3; $p < .01$). See Figure 6.3.A for details.

Source-level analysis

The group-averaged ERFs and the differences between groups observed in sensor-level analysis during the DDT future reward delay condition, were reconstructed and localised in source-space using an LCMV beamformer, see section 2.8.3.2 for details. The comparison of HI and LI groups of the Delay-M1 component, HI > LI, showed significant negative clusters located in the right middle frontal gyrus, left middle cingulate, thalamus, left fusiform, bilateral temporal regions, right parietal and right occipital regions; $p < .05$, see Figure 6.3.C. The comparison of HI and LI groups of the Delay-M2 component showed significant negative clusters in the right middle frontal gyrus, left hippocampus, left amygdala, right thalamus, right precentral and postcentral regions, fusiform, bilateral temporal regions, right parietal and bilateral occipital regions; $p < .05$, see Figure 6.3.C. The comparison of HI and LI groups for the Delay-M3 component revealed significant positive clusters (= larger amplitude in the HI group) bilaterally in the medial part of the superior frontal gyrus, left middle and superior frontal gyrus, including the thalamus and left insula, as well as right temporal, bilateral occipital regions; $p < .05$, see Figure 6.3.C.

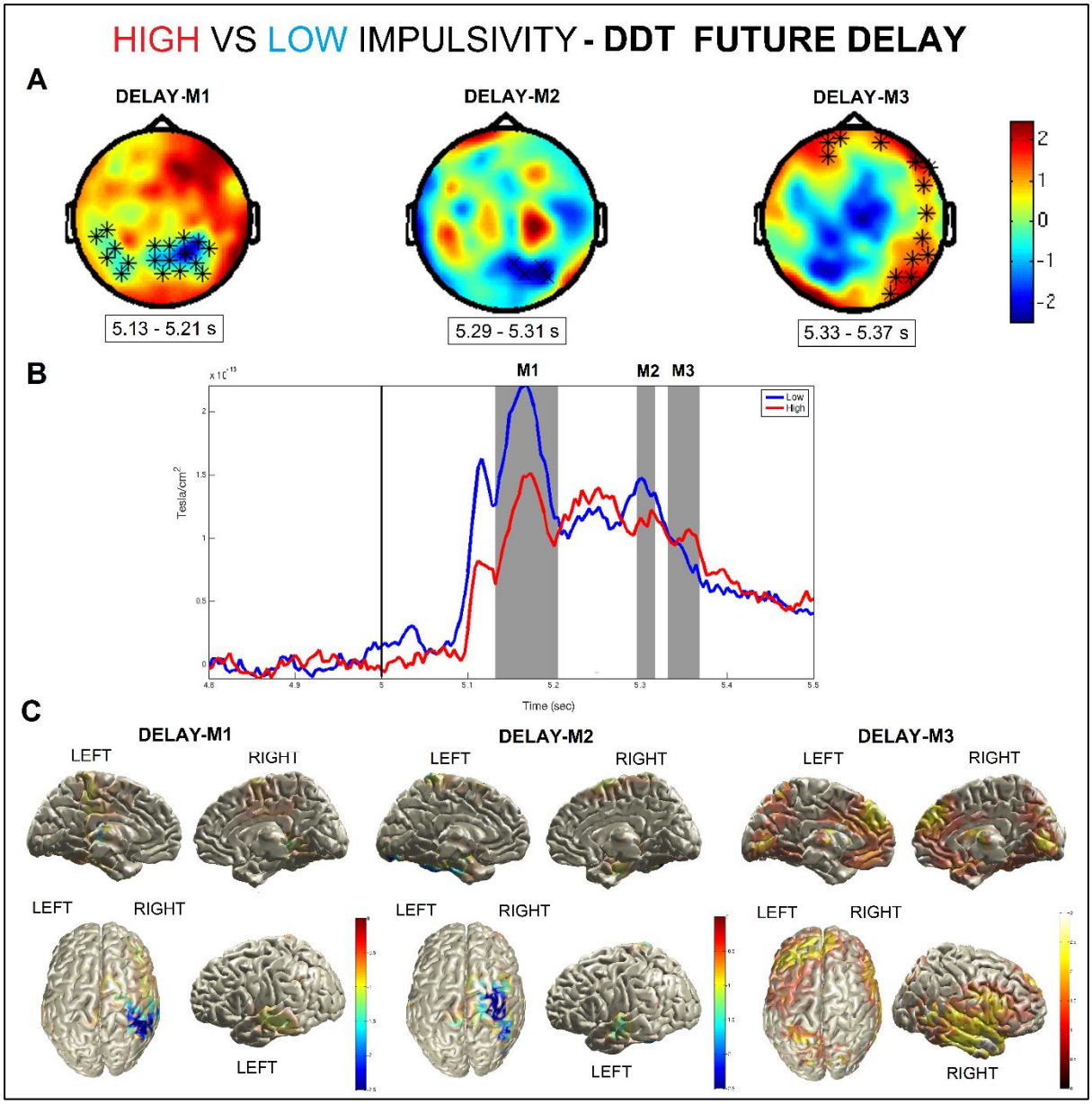

Figure 6.3. (A) Sensor-level analysis: topography of the statistical difference in amplitude between HI and LI groups during the Delay condition, between 5 and 7 seconds after stimulus onset. *T*-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$. The colour scale represents the *t*-values, * indicates significant clusters with $p < .01$. (B) Group averages of ERFs during Immediate trials for the HI (red line) and LI (blue line) groups, the group averages were plotted from significant sensors ($p < .01$) only. The grey columns represent the statistical difference between groups from the cluster-based permutations, see section 2.8.2.1 for details. Time point 0, marked with a black line, denotes the onset of target stimulus presentation. (C) Source-level analysis: the statistical differences observed in amplitude between HI and LI groups during the Delay condition, were localised in source-space, see section 2.8.3.2 for details. The colour scale represents the *t*-values.

6.4.3. Frequency and time-frequency analysis.

High vs Low Impulsivity groups – DDT Immediate reward condition

Delta band activity

Cluster-based permutation tests, which contrasted the HI and LI groups in the DDT Immediate reward condition with respect to time-averaged power for the frequency range of 1-4 Hz, revealed significant differences between groups. As shown in Figure 6.4.A, a significant difference was found between 1 and 3 s after the presentation of the immediate reward cue ($p < .01$). Here, the HI group showed significantly higher delta power relative to the LI group in posterior and anterior sensors.

At source-level, the statistical comparison of the HI group against the LI group in the delta band (1-4 Hz), showed significant positive clusters located in frontal, temporal and occipital regions, specifically bilaterally in the middle and right superior frontal regions, right cuneus, precuneus, fusiform, bilateral temporal, parietal and occipital regions; $p < .05$, see Figure 6.4.C.

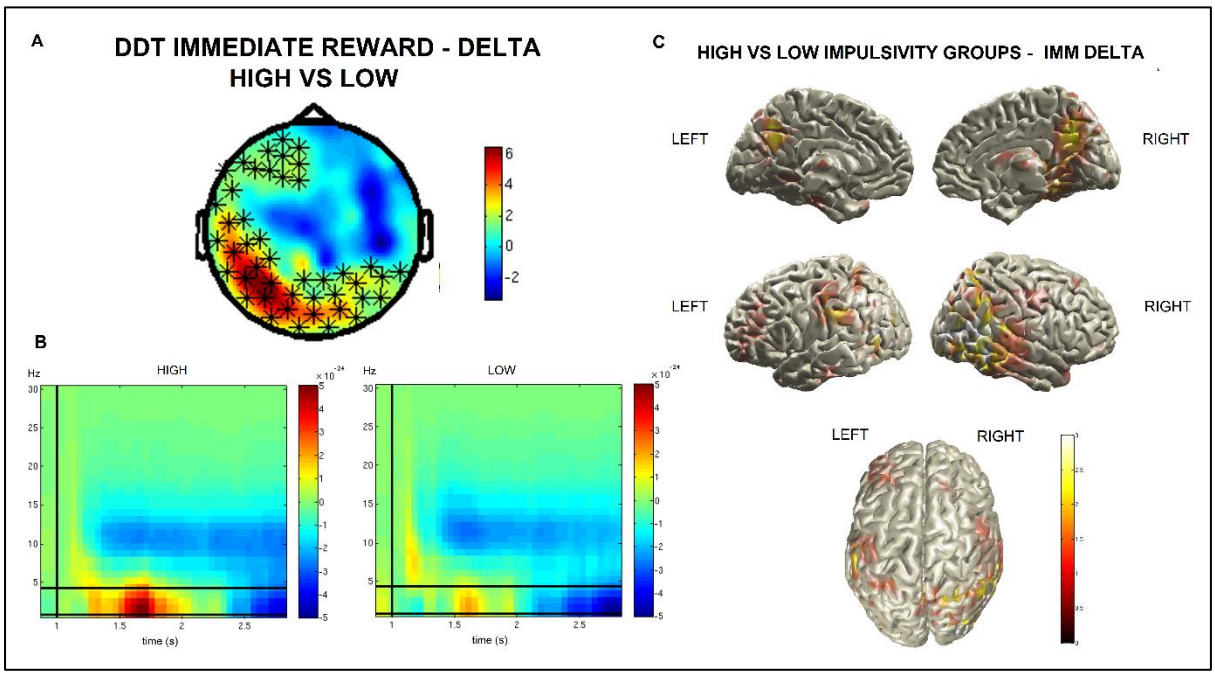

Figure 6.4. **(A)** Results from sensor-level analysis: topography of the statistical difference in delta power between HI and LI groups during the Immediate condition, between 1 and 3 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$; see section 2.8.2.2 for details. As a result of contrasting the HI against the LI group, hot colours denote positive power differences (larger power in the HI group), while cool colours denote negative power differences (larger power in the LI group). The colour scale represents the *t*-values, hot for positive and cool colours for negative values; * indicates significant clusters with $p < .01$. **(B)** Baseline corrected TFR mean power plots of all sensors in the HI and LI groups, showing delta, theta, alpha and beta bands. The y-axis denotes frequency, the x-axis denotes time in

ms, t=0 and vertical black lines represent the presentation of the Immediate reward cue, horizontal black lines limit delta band. The colour scale represents power in relation to the baseline period, power values from -5 to 5 ($x10^{-24}$). **(C)** Results from source-level analysis: the statistical differences observed in delta band power between HI and LI groups during the Immediate condition, were localised in source-space, see section 2.8.3.3 for details. The colour scale represents the t-values.

Theta band activity

Cluster-based permutation tests, which contrasted the HI and LI groups in the DDT Immediate reward condition with respect to time-averaged power for the frequency range of 4-8 Hz, revealed significant differences between groups. As shown in Figure 6.5.A, a significant difference was observed between 1.052 and 2.052 s after the stimulus onset ($p < .01$). The HI group showed significantly higher theta power than LI subjects in posterior sensors.

At source-level, the statistical comparison of the HI group against the LI group in theta band (4-8 Hz), showed significant positive clusters located bilaterally in the middle frontal gyrus, right inferior and superior frontal gyrus, left SMA, left hippocampus, bilateral parietal, temporal and occipital regions; $p < .05$, see Figure 6.5.C.

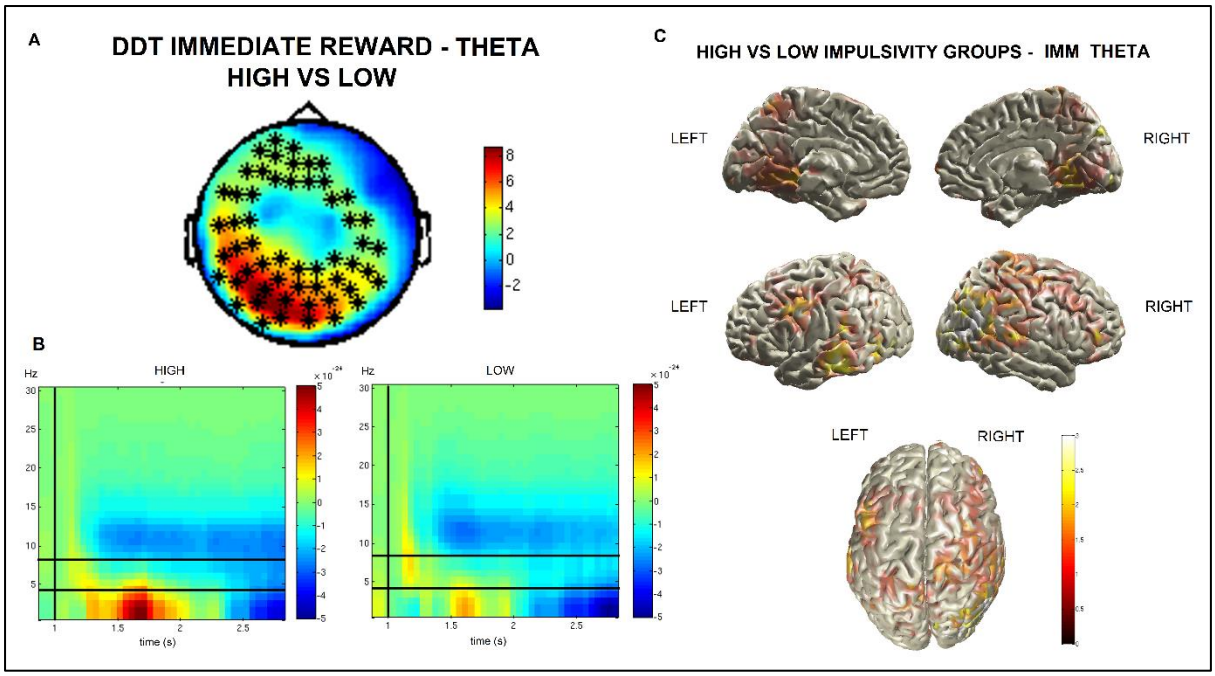

Figure 6.5. **(A)** Results from sensor-level analysis: topography of the statistical difference in theta power between HI and LI groups during the Immediate condition, between 1 and 3 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$; see section 2.8.2.2 for details. As a result of contrasting the HI against the LI group, hot colours denote positive power differences (larger power in the HI group), while cool colours denote negative power differences (larger power in the LI group). The colour scale represents the *t*-values, hot for positive and cool colours for negative values; * indicates significant clusters with $p < .01$. **(B)**

Baseline corrected TFR mean power plots of all sensors in the HI and LI groups, showing delta, theta, alpha and beta bands. The y-axis denotes frequency, the x-axis denotes time in ms, t=0 and vertical black lines represent the presentation of the Immediate reward cue, horizontal black lines limit theta band. The colour scale represents power in relation to the baseline period, power values from -5 to 5 ($x10^{-24}$). **(C)** Results from source-level analysis: the statistical differences observed in theta band power between HI and LI groups during the Immediate condition, were localised in source-space, see section 2.8.3.3 for details. The colour scale represents the t-values.

Alpha band activity

Cluster-based permutation tests, which contrasted the HI and LI groups in the DDT Immediate reward condition with respect to time-averaged power for the frequency range of 8-12 Hz, revealed a significant difference between groups. As shown in Figure 6.6.A, a significant difference was observed in this frequency range between 1 and 3 s after the stimulus onset ($p < .01$). Here, the HI group showed significantly higher alpha power than LI individuals in frontal, temporal and occipital sensors.

At source-level, the statistical comparison of the HI group against the LI group in alpha band (8-12 Hz), showed significant positive clusters located in the orbital part of inferior, medial and superior frontal gyrus, SMA, ACC, middle cingulate cortex, paracentral lobule, rectus, fusiform, bilateral temporal, right parietal and bilateral occipital regions; $p < .05$, see Figure 6.6.C.

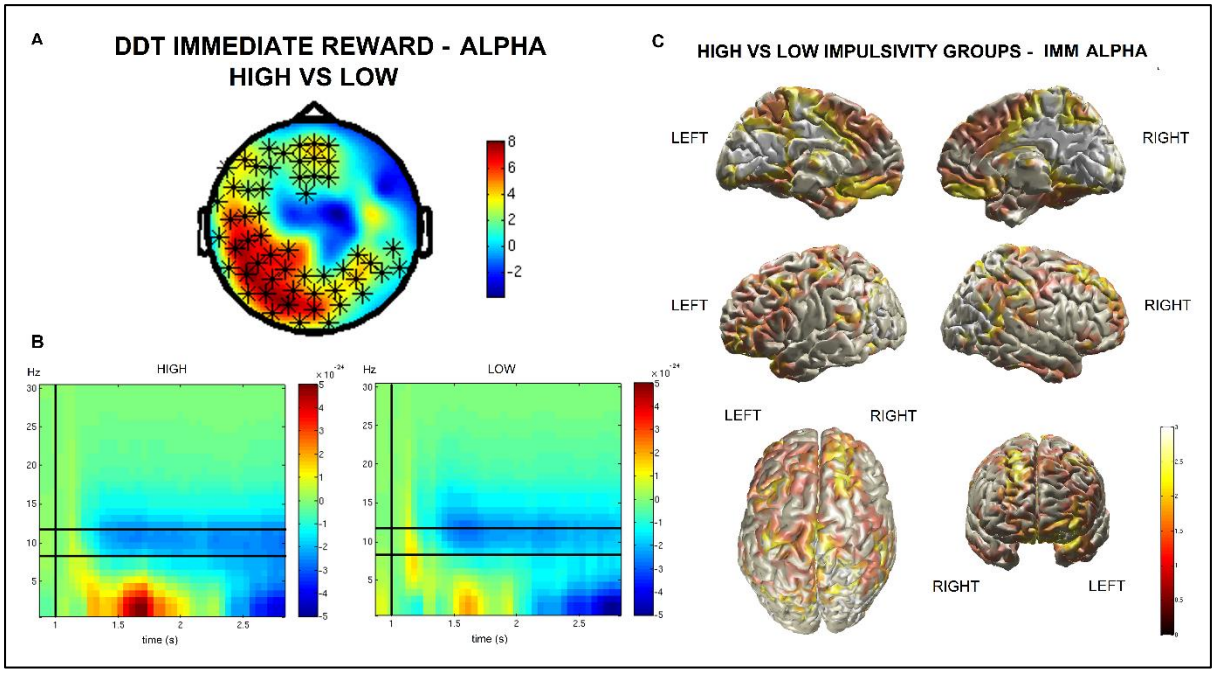

Figure 6.6. (A) Results from sensor-level analysis: topography of the statistical difference in alpha power between HI and LI groups during the Immediate condition, between 1 and 3 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$; see section 2.8.2.2 for details. As a result of contrasting the HI against the LI group, hot colours denote positive power

differences (larger power in the HI group), while cool colours denote negative power differences (larger power in the LI group). The colour scale represents the *t*-values, hot for positive and cool colours for negative values; * indicates significant clusters with $p < .01$. **(B)** Baseline corrected TFR mean power plots of all sensors in the HI and LI groups, showing delta, theta, alpha and beta bands. The y-axis denotes frequency, the x-axis denotes time in ms, t=0 and vertical black lines represent the presentation of the Immediate reward cue, horizontal black lines limit alpha band. The colour scale represents power in relation to the baseline period, power values from -5 to 5 ($x10^{-24}$). **(C)** Results from source-level analysis: the statistical differences observed in alpha band power between HI and LI groups during the Immediate condition, were localised in source-space, see section 2.8.3.3 for details. The colour scale represents the t-values.

Beta activity

Cluster-based permutation tests, which contrasted the HI and LI groups in the DDT Immediate reward condition with respect to time-averaged power for the frequency range of 13-30 Hz, revealed significant differences between groups. As shown in Figure 6.7.A, two significant differences between groups were observed in this frequency range. A significant negative difference was observed between 1 and 3 s after the stimulus onset ($p < .01$). Here, the LI group showed significantly higher beta power than HI subjects in right frontal and temporal sensors. A second significant positive difference was found between 1 and 3 s after the presentation of the immediate reward cue ($p < .01$). HI subjects showed significantly higher beta power than LI subjects in occipital and left temporal sensors.

At source-level, the statistical comparison of the HI group against the LI group in beta band (13-30 Hz), showed significant negative clusters located in the left SMA, fusiform, bilateral middle and superior temporal regions, right inferior temporal, right inferior parietal, right inferior and middle occipital regions; $p < .05$, see Figure 6.7.C. The same cluster-based permutation testing also revealed significant positive clusters in left inferior, middle and superior frontal gyrus, bilateral orbital part of middle and superior frontal gyrus, bilateral medial part of superior frontal gyrus, middle cingulate, insula, bilateral temporal, left parietal and bilateral occipital regions; $p < .05$, see Figure 6.7.C.

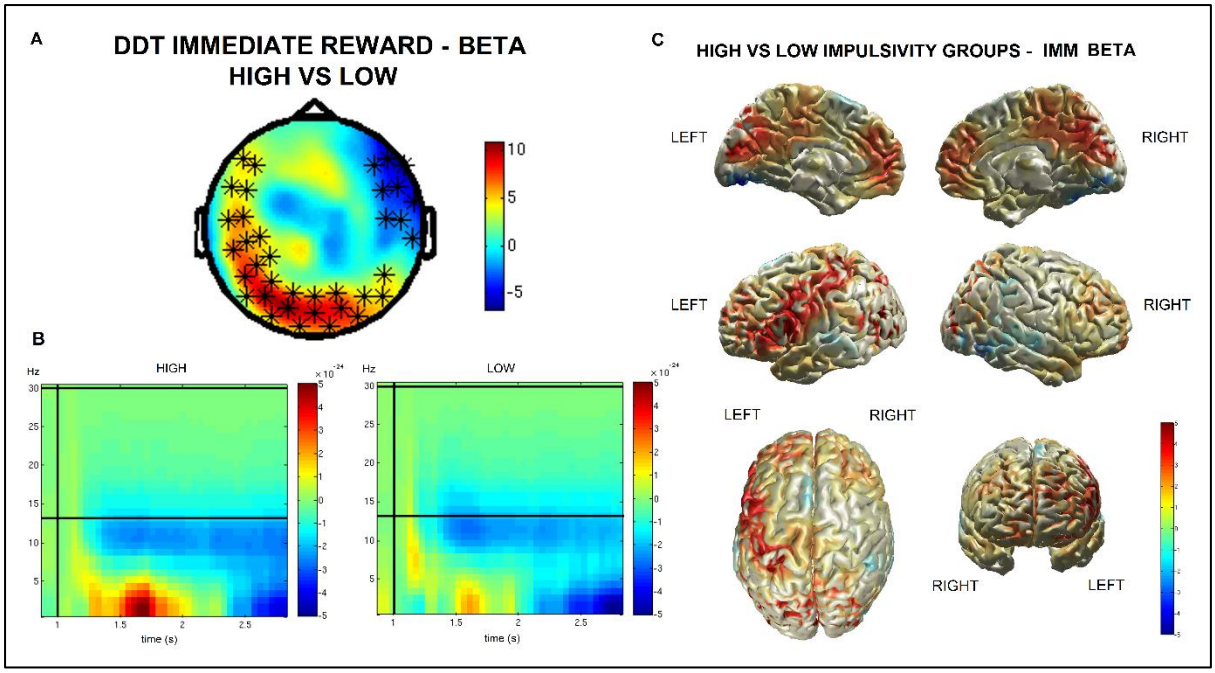

Figure 6.7. (A) Results from sensor-level analysis: topography of the statistical difference in beta power between HI and LI groups during the Immediate condition, between 1 and 3 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$; see section 2.8.2.2 for details As a result of contrasting the HI against the LI group, hot colours denote positive power differences (larger power in the HI group), while cool colours denote negative power differences (larger power in the LI group). The colour scale represents the *t*-values, hot for positive and cool colours for negative values; * indicates significant clusters with $p < .01$. **(B)** Baseline corrected TFR mean power plots of all sensors in the HI and LI groups, showing delta, theta, alpha and beta bands. The y-axis denotes frequency, the x-axis denotes time in ms, t=0 and vertical black lines represent the presentation of the Immediate reward cue, horizontal black lines limit beta band. The colour scale represents power in relation to the baseline period, power values from -5 to 5 ($x10^{-24}$). **(C)** Results from source-level analysis: the statistical differences observed in beta band power between HI and LI groups during the Immediate condition, were localised in source-space, see section 2.8.3.3 for details. The colour scale represents the t-values.

High vs Low Impulsivity groups – DDT Future reward Magnitude condition

Delta band activity

Cluster-based permutation tests, which contrasted the HI and LI groups in the DDT future reward magnitude condition with respect to time-averaged power for the frequency range of 1-4 Hz, revealed significant differences between groups. As shown in figure 6.8.A, a significant positive difference was observed in this frequency range between 3 and 5 s after the presentation of the immediate reward cue ($p < .01$). The HI group showed significantly higher delta power relative to the LI group in left anterior, central and posterior sensors.

At source-level, the statistical comparison of the HI group against the LI group in delta band (1-4 Hz), showed significant positive clusters located bilaterally in the superior frontal gyrus, left inferior frontal gyrus, middle temporal, left inferior temporal, right superior temporal, right superior parietal, left inferior parietal and bilateral occipital regions; $p < .05$, see Figure 6.8.C.

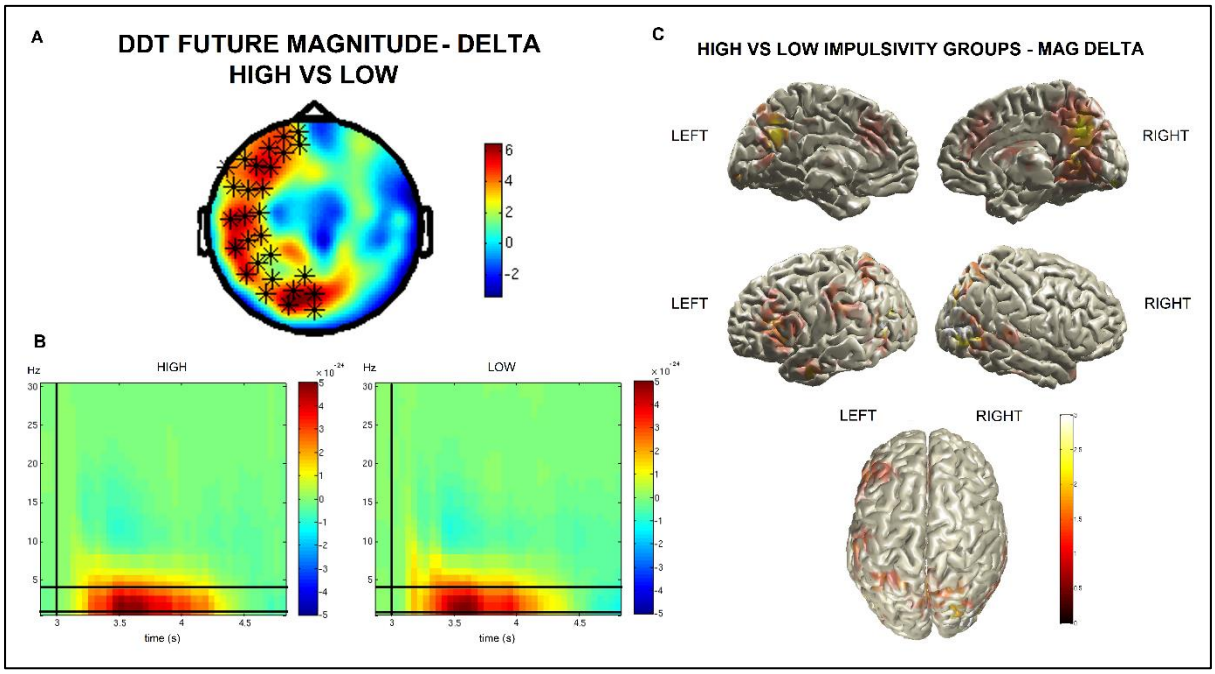

Figure 6.8. (A) Results from sensor-level analysis: topography of the statistical difference in delta power between HI and LI groups during the future reward Magnitude condition, between 3 and 5 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$; see section 2.8.2.2 for details. As a result of contrasting the HI against the LI group, hot colours denote positive power differences (larger power in the HI group), while cool colours denote negative power differences (larger power in the LI group). The colour scale represents the *t*-values, hot for positive and cool colours for negative values; * indicates significant clusters with $p < .01$. **(B)** Baseline corrected TFR mean power plots of all sensors in the HI and LI groups, showing delta, theta, alpha and beta bands. The y-axis denotes frequency, the x-axis denotes time in ms, t=0 and vertical black lines represent the presentation of the future reward Magnitude cue, horizontal black lines limit delta band. The colour scale represents power in relation to the baseline period, power values from -5 to 5 ($x10^{-24}$). **(C)** Results from source-level analysis: the statistical differences observed in delta band power between HI and LI groups during the Magnitude condition, were localised in source-space, see section 2.8.3.3 for details. The colour scale represents the t-values.

Theta band activity

Cluster-based permutation tests, which contrasted the HI and LI groups in the DDT future reward magnitude condition with respect to time-averaged power for the frequency range of 4-8 Hz, revealed significant differences between groups. As shown in Figure 6.9.A, a significant positive difference was observed in this frequency range between 3 and 5 s after

the stimulus onset (p < .01). The HI group showed significantly higher theta power than LI subjects in left anterior, central and posterior sensors.

At source-level, the statistical comparison of the HI group against the LI group in theta band (4-8 Hz), showed significant positive clusters located in the left medial part of the superior frontal gyrus, left inferior and middle frontal gyrus, left ACC, bilateral middle cingulate, right fusiform, precuneus, left postcentral and paracentral lobule, left thalamus, right temporal and right occipital regions; p < .05, see Figure 6.9.C.

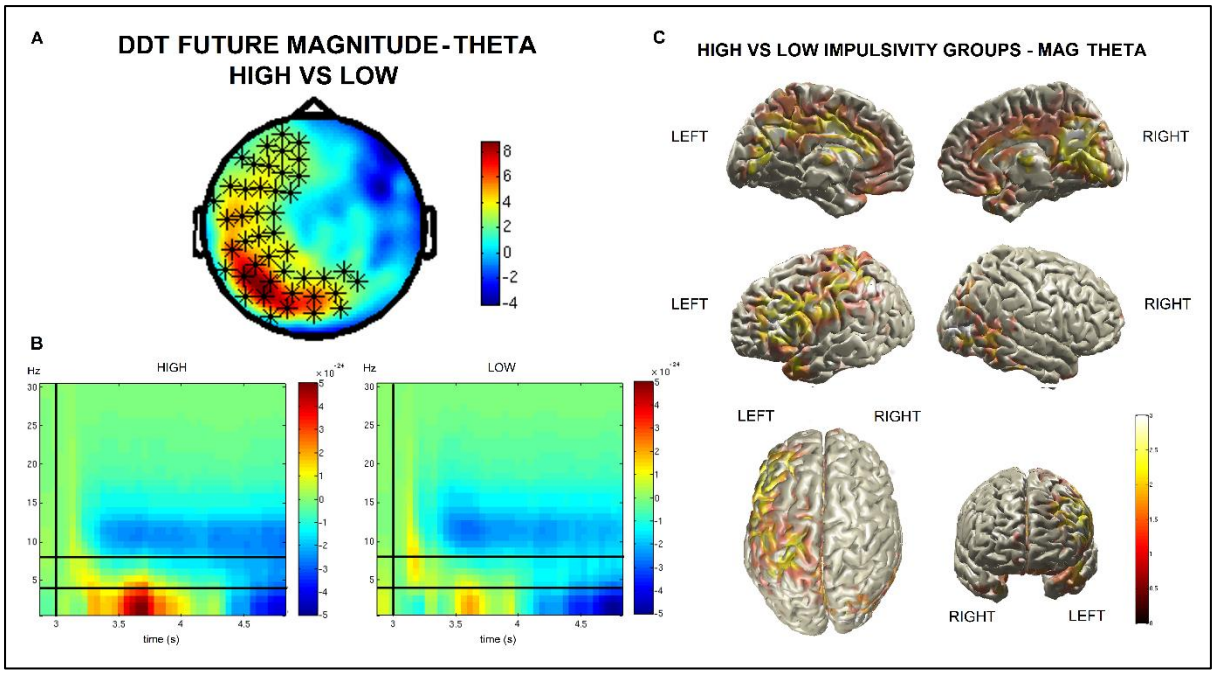

Figure 6.9. (A) Results from sensor-level analysis: topography of the statistical difference in theta power between HI and LI groups during the future reward Magnitude condition, between 3 and 5 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and p < .05; see section 2.8.2.2 for details. As a result of contrasting the HI against the LI group, hot colours denote positive power differences (larger power in the HI group), while cool colours denote negative power differences (larger power in the LI group). The colour scale represents the *t*-values, hot for positive and cool colours for negative values; * indicates significant clusters with p < .01. **(B)** Baseline corrected TFR mean power plots of all sensors in the HI and LI groups, showing delta, theta, alpha and beta bands. The y-axis denotes frequency, the x-axis denotes time in ms, t=0 and vertical black lines represent the presentation of the future reward Magnitude cue, horizontal black lines limit theta band. The colour scale represents power in relation to the baseline period, power values from -5 to 5 ($x10^{-24}$). **(C)** Results from source-level analysis: the statistical differences observed in theta band power between HI and LI groups during the Magnitude condition, were localised in source-space, see section 2.8.3.3 for details. The colour scale represents the t-values.

Alpha band activity

Cluster-based permutation tests, which contrasted the HI and LI groups in the DDT future reward magnitude condition with respect to time-averaged power for the frequency range of 8-12 Hz, revealed significant differences between groups. As shown in Figure 6.10.A, a significant difference was observed in this frequency range between 3 and 5 s after the stimulus onset ($p < .01$). The HI group showed significantly more alpha power than LI individuals in anterior, central and posterior sensors.

At source-level, the statistical comparison of the HI group against the LI group in the alpha band (8-12 Hz), showed significant positive clusters located in the left inferior and middle frontal gyrus, medial part of superior frontal gyrus, right superior frontal gyrus, anterior and middle cingulate, right thalamus, fusiform, left hippocampus, left amygdala, postcentral and precentral, bilateral temporal and occipital regions; $p < .05$, see Figure 6.10.C. The same cluster-based permutation testing also revealed significant negative clusters in the right orbital part of inferior and middle frontal gyrus, right middle frontal gyrus, right inferior parietal, left middle and superior occipital regions; $p < .05$, see Figure 6.10.C.

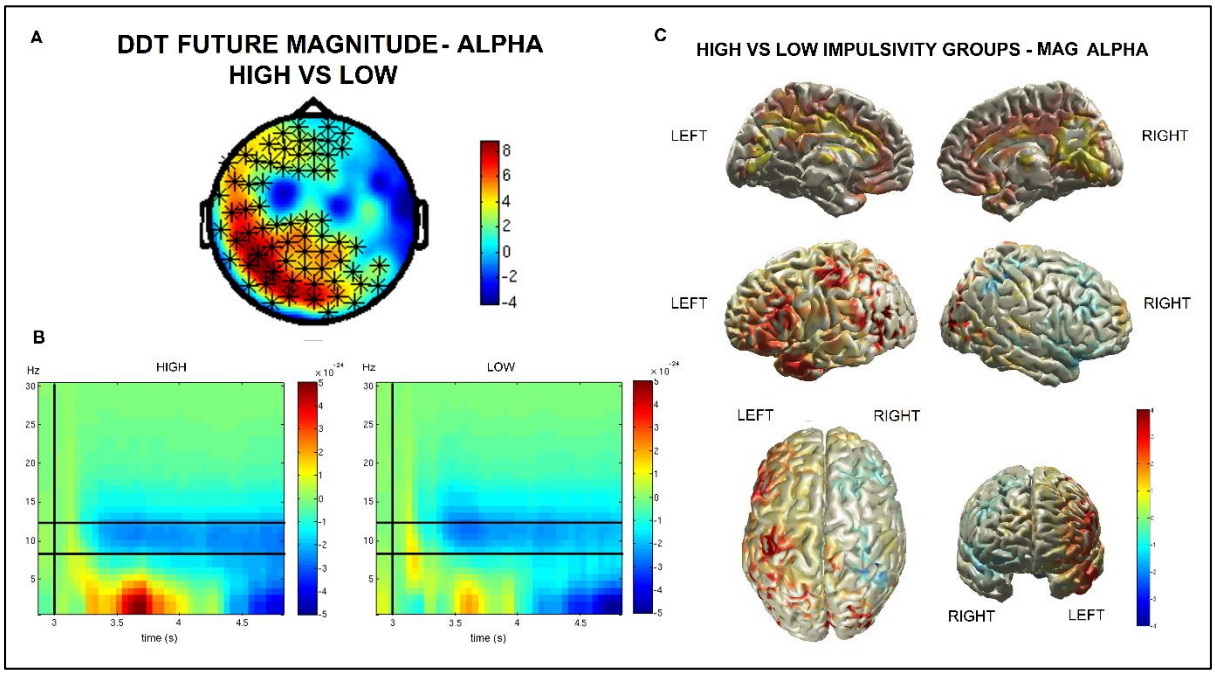

Figure 6.10. (A) Results from sensor-level analysis: topography of the statistical difference in alpha power between HI and LI groups during the future reward Magnitude condition, between 3 and 5 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$; see section 2.8.2.2 for details. As a result of contrasting the HI against the LI group, hot colours denote positive power differences (larger power in the HI group), while cool colours denote negative power differences (larger power in the LI group). The colour scale represents the *t*-values, hot for positive and cool colours for negative values; * indicates significant clusters with $p < .01$. **(B)** Baseline corrected TFR mean power plots of all sensors in the HI and LI groups, showing delta, theta, alpha and beta bands. The y-axis denotes frequency, the x-axis denotes time in

ms, t=0 and vertical black lines represent the presentation of the future reward Magnitude cue, horizontal black lines limit alpha band. The colour scale represents power in relation to the baseline period, power values from -5 to 5 ($x10^{-24}$). **(C)** Results from source-level analysis: the statistical differences observed in alpha band power between HI and LI groups during the Magnitude condition, were localised in source-space, see section 2.8.3.3 for details. The colour scale represents the t-values.

Beta activity

Cluster-based permutation tests, which contrasted the HI and LI groups in the DDT future reward magnitude condition with respect to time-averaged power for the frequency range of 13-30 Hz, revealed significant differences between groups. As shown in Figure 6.11.A, two significant differences were observed in this frequency range. A significant difference was observed between 3 and 5 s after the stimulus onset ($p < .01$). Here, the HI group showed significantly higher beta power than LI subjects in right frontal and temporal sensors**.** A second significant difference was found between 3 and 5 s after the presentation of the immediate reward cue ($p < .01$). HI subjects showed significantly higher beta power than LI subjects in posterior and left fronto-temporal sensors, and lower beta power in right fronto-temporal and central sensors.

At source-level, the statistical comparison of the HI group against the LI group in beta band (13-30 Hz), showed significant positive clusters located in the right opercular part of inferior frontal gyrus, medial part of superior frontal gyrus, left orbital part of inferior and middle frontal gyrus, left middle frontal, left postcentral, right temporal, left inferior and superior parietal, bilateral occipital regions; $p < .05$, see Figure 6.11.C. The same cluster-based permutation testing also revealed significant negative clusters in right middle frontal, right SMA, right middle cingulate, right thalamus, right parietal, left temporal and bilateral occipital regions; $p < .05$, see Figure 6.11.C.

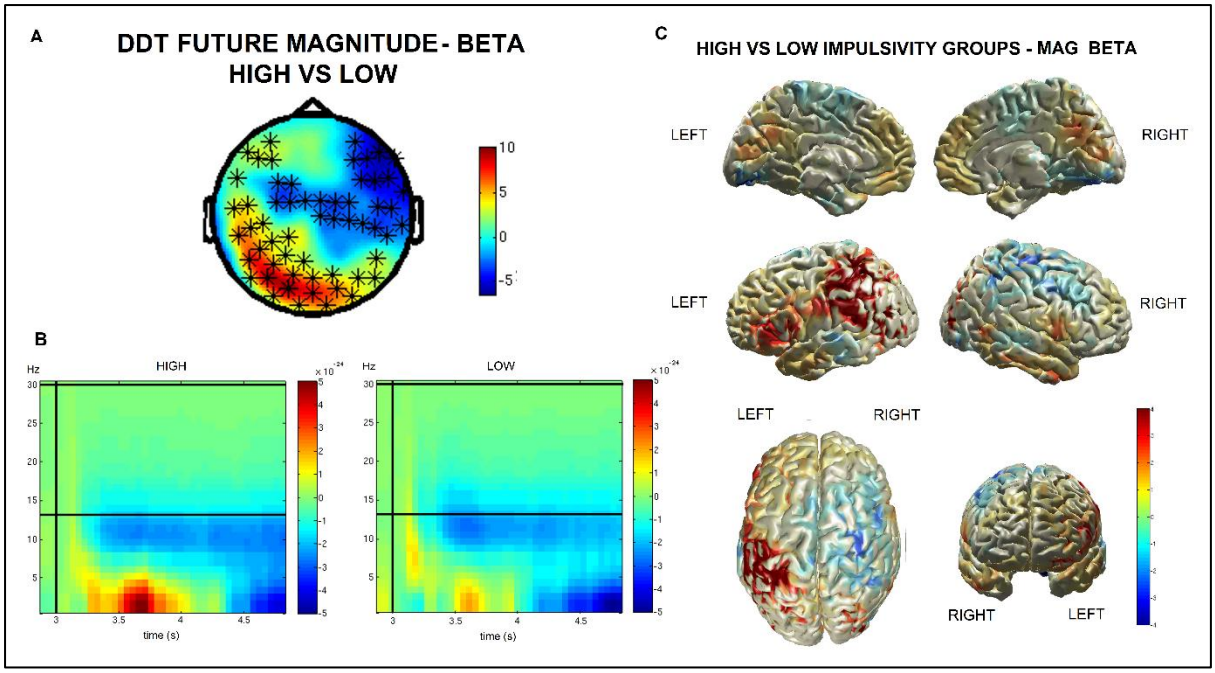

Figure 6.11. (A) Results from sensor-level analysis: topography of the statistical difference in beta power between HI and LI groups during the future reward Magnitude condition, between 3 and 5 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$; see section 2.8.2.2 for details. As a result of contrasting the HI against the LI group, hot colours denote positive power differences (larger power in the HI group), while cool colours denote negative power differences (larger power in the LI group). The colour scale represents the *t*-values, hot for positive and cool colours for negative values; * indicates significant clusters with $p < .01$. **(B)** Baseline corrected TFR mean power plots of all sensors in the HI and LI groups, showing delta, theta, alpha and beta bands. The y-axis denotes frequency, the x-axis denotes time in ms, t=0 and vertical black lines represent the presentation of the future reward Magnitude cue, horizontal black lines limit beta band. The colour scale represents power in relation to the baseline period, power values from -5 to 5 ($x10^{-24}$). **(C)** Results from source-level analysis: the statistical differences observed in beta band power between HI and LI groups during the Magnitude condition, were localised in source-space, see section 2.8.3.3 for details. The colour scale represents the t-values.

High vs Low Impulsivity groups – DDT Future reward Delay condition

Delta band activity

Cluster-based permutation tests, which contrasted the HI and LI groups in the DDT future reward delay condition with respect to time-averaged power for the frequency range of 1-4 Hz, revealed significant differences between groups. As shown in Figure 6.12.A, one significant difference was found between 5 and 7 s after the presentation of the future reward delay cue ($p < .01$). The HI group showed significantly more delta power compared to the LI group in left anterior, central and posterior sensors.

At source-level, the statistical comparison of the HI group against the LI group in delta band (1-4 Hz), showed significant positive clusters in left inferior and middle frontal gyrus, right posterior and middle cingulate, right hippocampus, right precuneus, right temporal, left parietal and right occipital regions; $p < .05$, see Figure 6.12.C.

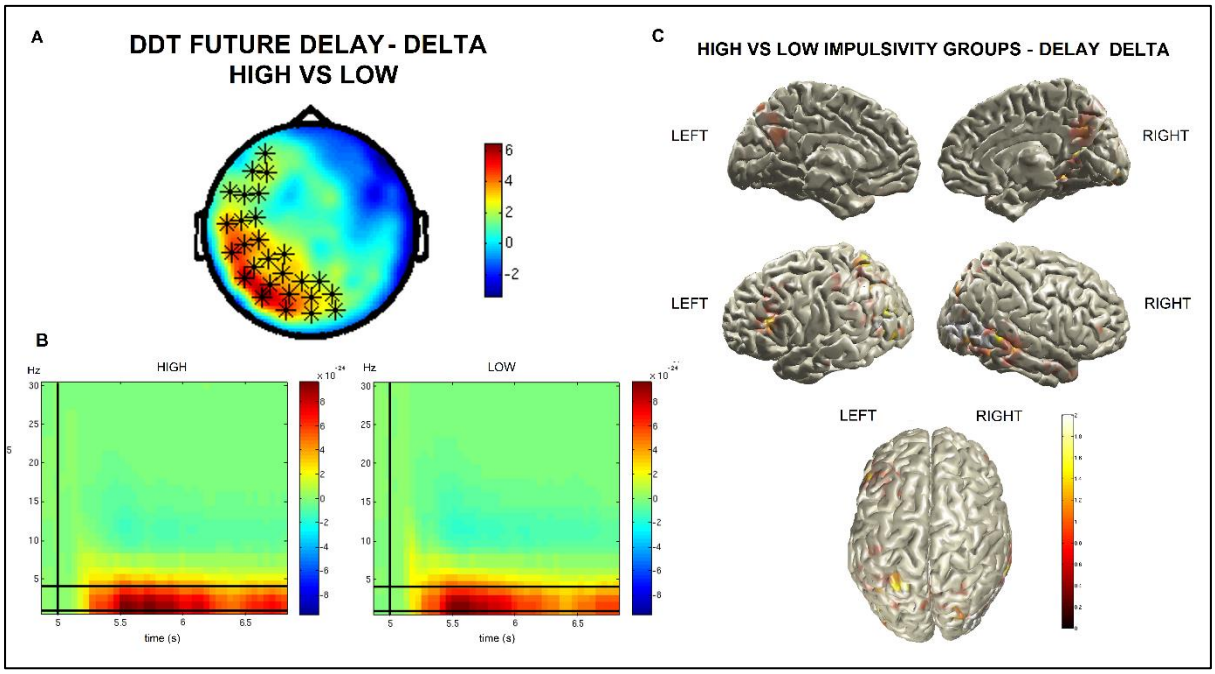

Figure 6.12. (A) Results from sensor-level analysis: topography of the statistical difference in delta power between HI and LI groups during the future reward Delay condition, between 5 and 7 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$; see section 2.8.2.2 for details. As a result of contrasting the HI against the LI group, hot colours denote positive power differences (larger power in the HI group), while cool colours denote negative power differences (larger power in the LI group). The colour scale represents the *t*-values, hot for positive and cool colours for negative values; * indicates significant clusters with $p < .01$. **(B)** Baseline corrected TFR mean power plots of all sensors in the HI and LI groups, showing delta, theta, alpha and beta bands. The y-axis denotes frequency, the x-axis denotes time in ms, t=0 and vertical black lines represent the presentation of the future reward Delay cue, horizontal black lines limit delta band. The colour scale represents power in relation to the baseline period, power values from -8 to 8 ($x10^{-24}$). **(C)** Results from source-level analysis: the statistical differences observed in delta band power between HI and LI groups during the Delay condition, were localised in source-space, see section 2.8.3.3 for details. The colour scale represents the t-values.

Theta band activity

Cluster-based permutation tests, which contrasted the HI and LI groups in the DDT future reward magnitude condition with respect to time-averaged power for the frequency range of 4-8 Hz, revealed significant differences between groups. As shown in Figure 6.13.A, two significant differences between groups were observed in this frequency range. A significant difference was observed between 5 and 7 s after the stimulus onset ($p < .01$). The HI group

showed significantly higher theta power than LI subjects in left anterior, central and posterior sensors.

At source-level, the statistical comparison of the HI group against the LI group in theta band (4-8 Hz), showed significant positive clusters located in the left inferior, middle and superior frontal gyrus, left precentral and postcentral, ACC, left thalamus, left middle cingulate, bilateral temporal, left inferior parietal and bilateral occipital regions; $p < .05$, see Figure 6.13.C.

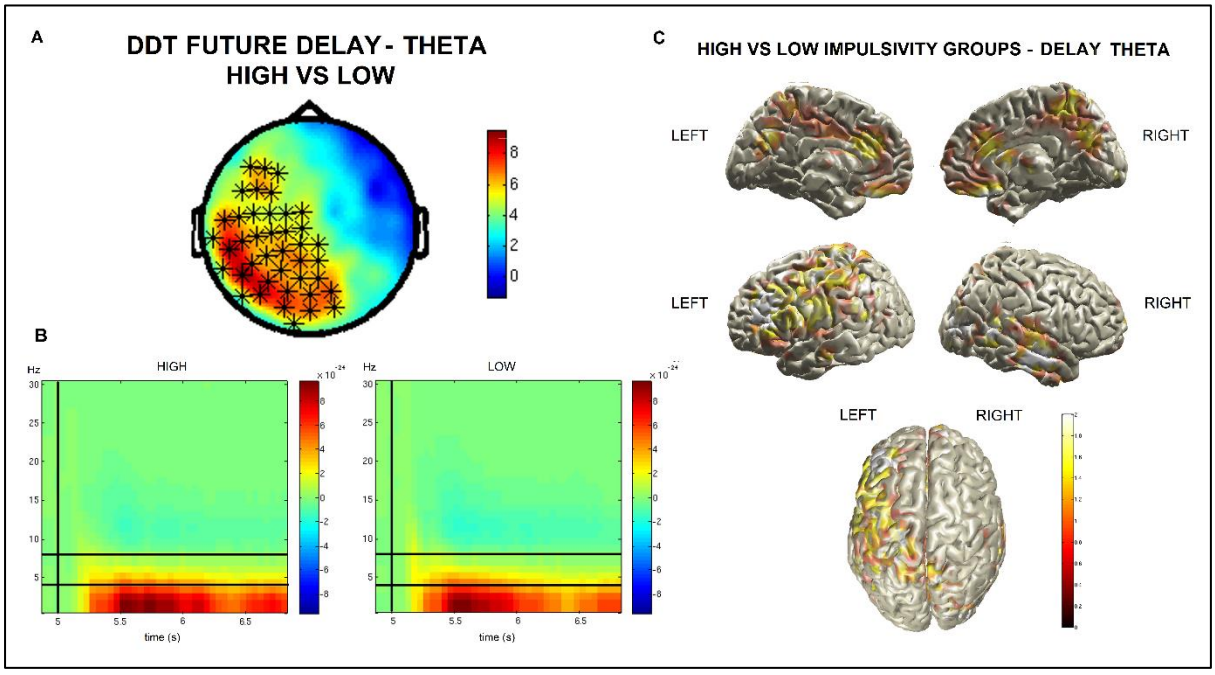

Figure 6.13. (A) Results from sensor-level analysis: topography of the statistical difference in theta power between HI and LI groups during the future reward Delay condition, between 5 and 7 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$; see section 2.8.2.2 for details. As a result of contrasting the HI against the LI group, hot colours denote positive power differences (larger power in the HI group), while cool colours denote negative power differences (larger power in the LI group). The colour scale represents the *t*-values, hot for positive and cool colours for negative values; * indicates significant clusters with $p < .01$. **(B)** Baseline corrected TFR mean power plots of all sensors in the HI and LI groups, showing delta, theta, alpha and beta bands. The y-axis denotes frequency, the x-axis denotes time in ms, t=0 and vertical black lines represent the presentation of the future reward Delay cue, horizontal black lines limit theta band. The colour scale represents power in relation to the baseline period, power values from -8 to 8 ($x10^{-24}$). **(C)** Results from source-level analysis: the statistical differences observed in theta band power between HI and LI groups during the Delay condition, were localised in source-space, see section 2.8.3.3 for details. The colour scale represents the t-values.

Alpha band activity

Cluster-based permutation tests, which contrasted the HI and LI groups in the DDT future reward delay condition with respect to time-averaged power for the frequency range of 8-12

Hz, revealed a significant difference between groups. As shown in Figure 6.14.A, a significant difference was observed between 5 and 7 s after the stimulus onset ($p < .01$). The HI group showed significantly higher alpha power than LI subjects in anterior, central and posterior sensors.

At source-level, the statistical comparison of the HI group against the LI group in alpha band (8-12 Hz), showed significant positive clusters located in right inferior, middle and superior occipital regions, left inferior and middle occipital, left middle temporal, right inferior, middle and superior temporal, medial part of superior frontal gyrus, anterior and middle cingulate, left middle and superior frontal gyrus, left caudate, hippocampus, left amygdala, fusiform, left inferior temporal, right opercular part of inferior frontal gyrus; $p < .05$, see Figure 6.14.C.

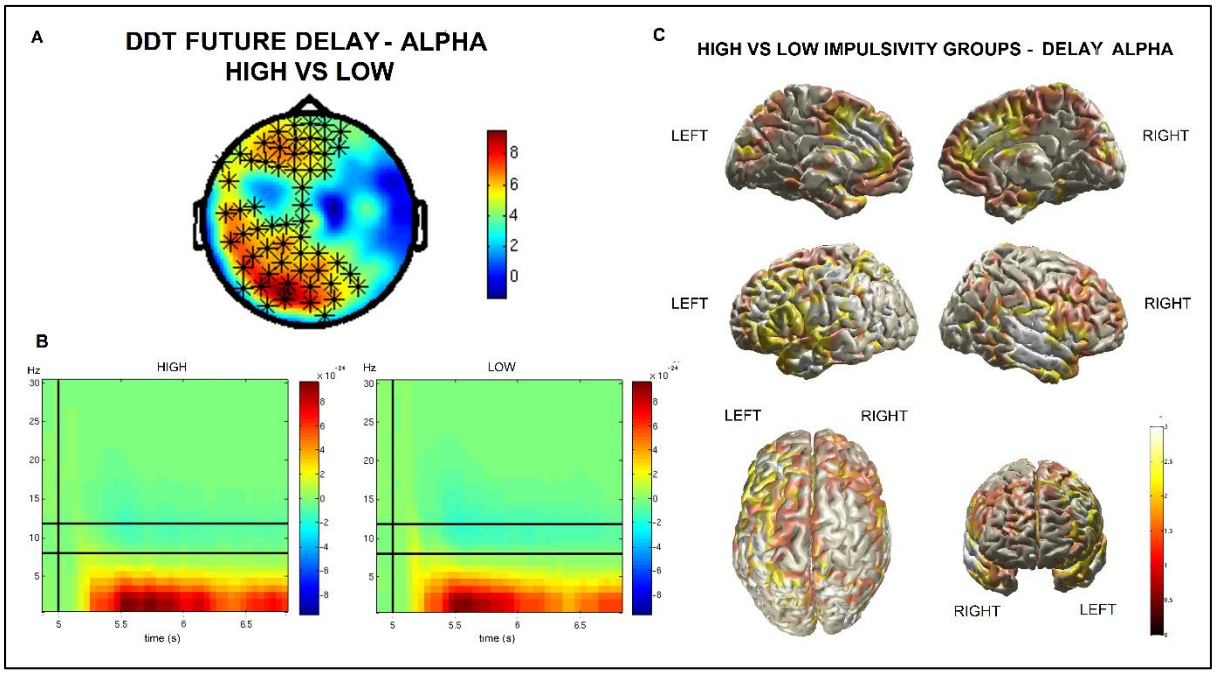

Figure 6.14. (A) Results from sensor-level analysis: topography of the statistical difference in alpha power between HI and LI groups during the future reward Delay condition, between 5 and 7 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$; see section 2.8.2.2 for details. As a result of contrasting the HI against the LI group, hot colours denote positive power differences (larger power in the HI group), while cool colours denote negative power differences (larger power in the LI group). The colour scale represents the *t*-values, hot for positive and cool colours for negative values; * indicates significant clusters with $p < .01$. **(B)** Baseline corrected TFR mean power plots of all sensors in the HI and LI groups, showing delta, theta, alpha and beta bands. The y-axis denotes frequency, the x-axis denotes time in ms, t=0 and vertical black lines represent the presentation of the future reward Delay cue, horizontal black lines limit alpha band. The colour scale represents power in relation to the baseline period, power values from -8 to 8 ($x10^{-24}$). **(C)** Results from source-level analysis: the statistical differences observed in alpha band power between HI and LI groups during the Delay condition, were localised in source-space, see section 2.8.3.3 for details. The colour scale represents the t-values.

Beta activity

Cluster-based permutation tests, which contrasted the HI and LI groups in the DDT future reward delay condition with respect to time-averaged power for the frequency range of 13-30 Hz, revealed significant differences between groups. As shown in Figure 6.15.A, two significant differences between groups were observed in this frequency range. A significant difference was observed between 5 and 7 s after the stimulus onset ($p < 0.01$). Here, the HI group showed significantly lower beta power than LI subjects in right anterior and central sensors**.** A second significant difference was found between 5 and 7 s after the presentation of the future reward delay cue ($p < .01$). The LI group showed significantly higher beta power relative to the HI group in left anterior, central and posterior sensors.

At source-level, the statistical comparison of the HI group against the LI group in beta band (13-30 Hz), showed significant positive clusters located in left inferior and superior parietal, right superior parietal, left inferior, middle and superior frontal gyrus, right middle and superior temporal pole, right inferior and middle temporal, left medial part of superior frontal gyrus, anterior cingulate; $p < .05$, see Figure 6.15.C. The same cluster-based permutation testing also revealed significant negative clusters in right middle and superior frontal gyrus, right medial part of superior frontal gyrus, supplementary motor area, right postcentral, left precuneus, left paracentral lobule, left middle and superior temporal, right inferior and middle occipital, left superior occipital regions; $p < .05$, see Figure 6.15.C.

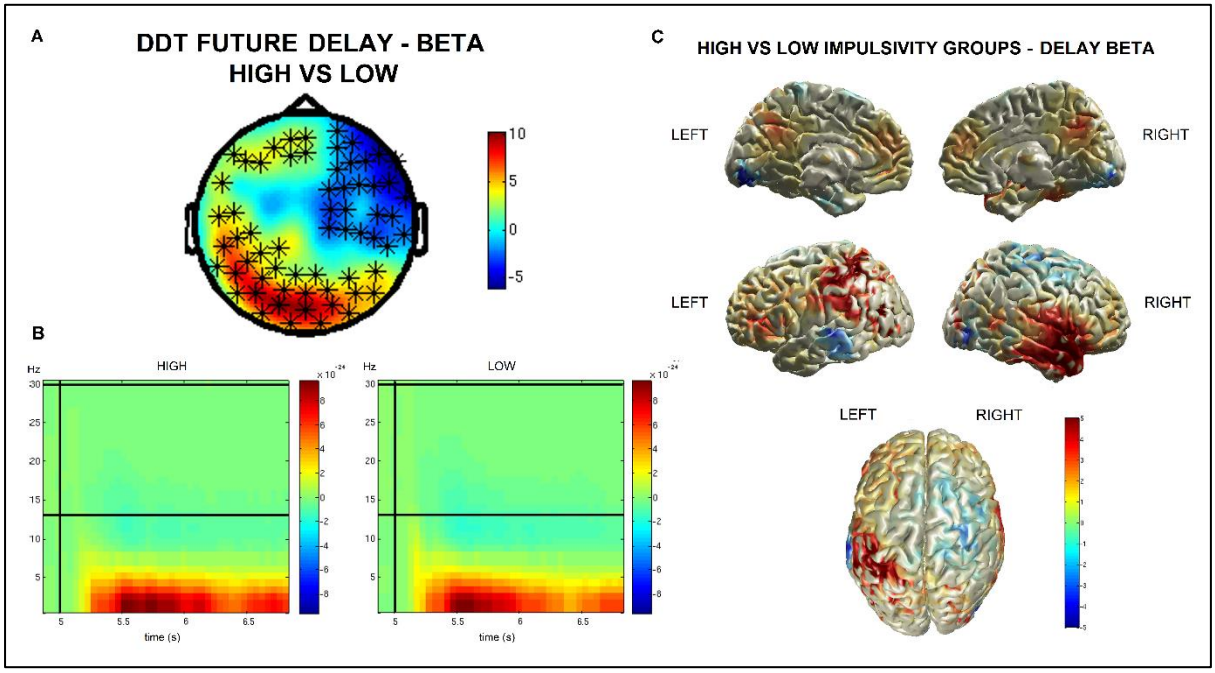

Figure 6.15. (A) Results from sensor-level analysis: topography of the statistical difference in beta power between HI and LI groups during the future reward Delay condition, between 5 and 7 seconds after stimulus onset. T-values were computed using non-parametric cluster-based permutation tests, with 1000 iterations, two-sided and $p < .05$; see section 2.8.2.2 for

details. As a result of contrasting the HI against the LI group, hot colours denote positive power differences (larger power in the HI group), while cool colours denote negative power differences (larger power in the LI group). The colour scale represents the *t*-values, hot for positive and cool colours for negative values; * indicates significant clusters with $p < .01$. **(B)** Baseline corrected TFR mean power plots of all sensors in the HI and LI groups, showing delta, theta, alpha and beta bands. The y-axis denotes frequency, the x-axis denotes time in ms, t=0 and vertical black lines represent the presentation of the future reward Delay cue, horizontal black lines limit beta band. The colour scale represents power in relation to the baseline period, power values from -8 to 8 ($x10^{-24}$). **(C)** Results from source-level analysis: th111e statistical differences observed in beta band power between HI and LI groups during the Delay condition, were localised in source-space, see section 2.8.3.3 for details. The colour scale represents the t-values.

6.5. DISCUSSION

In Chapter 3, findings demonstrated that the DDT was more sensitive to differences between groups defined by the combination of two impulsivity factors, rapid-response and reward-delay impulsivity, than groups of each dimension alone (Jauregi et al., 2018). Considering the behavioural differences found between high and low impulsivity groups, the primary aim of the current experiment was to examine these behavioural differences in delay-discounting using MEG. This was conducted in a healthy undergraduate student population, divided by their level of impulsivity as measured by questionnaires, see section 2.4.4 for details.

Given the significant differences observed between groups in the behavioural study, Chapter 3, it was hypothesised that the HI group would prefer small but immediate over larger, delayed rewards significantly more often than the LI group, reflecting more impulsive choices. Although high impulsivity individuals preferred smaller immediate rewards more, the mean proportions of smaller immediate vs. larger delayed reward choices were not significantly different between HI and LI groups in this MEG subsample. Nevertheless, given the significant differences reported in Chapter 5, brain patterns were expected to differ between the HI and LI groups, reflecting a propensity towards more impulsive choices despite a lack of an overt behavioural choice difference in this subsample. Our hypotheses are summarised in the following, along with a brief summary of the actual findings. Subsequently, findings and expectations will be discussed in more detail and within the context of the wider literature.

ERF-related hypotheses for the DDT

(1) It was expected that high impulsivity individuals would show reduced M1 amplitudes compared to low impulsivity individuals, reflecting a possible attention deficit, as observed during the NOGO and STOP conditions. The amplitudes of the M1 component across the three conditions were found to be significantly reduced in the HI group compared to the LI group.

(2) In the Immediate condition, high impulsivity individuals were expected to show larger M2 amplitudes in frontal sensors compared to low impulsivity individuals, representing preference for immediate rewards (Gui et al., 2016). During the Immediate reward condition, the HI group indeed showed significantly larger M2 amplitudes bilaterally in frontal regions, compared to the LI group.

(3) In the future Magnitude and Delay conditions, high impulsivity individuals were expected to show larger M2 amplitudes compared to low impulsivity individuals, as the M2 component has previously been reported to reflect the first valuation of reward magnitude and delay (Gui et al., 2016). The HI group was found to show significantly reduced M2 amplitudes during the two conditions in the right parietal cortex, compared to the LI group.

(4) High impulsivity individuals were expected to show reduced M3 amplitudes compared to low impulsivity individuals, reflecting less efficient reward processing (Kamarajan et al., 2015). Reduced M3 amplitudes in the HI compared to the LI group were indeed observed in parietal regions in the Immediate condition. Also, during the future Magnitude condition, reduced M3 amplitudes were found in right parietal and temporal regions in high impulsivity individuals. However, significantly increased M3 amplitudes were also found during the Immediate reward and future reward Delay conditions.

Hypotheses related to oscillatory activity during the DDT:

(1) High impulsivity individuals were expected to show more delta band power than low impulsivity individuals, representing an increased sensitivity towards reward. Increased delta band power was indeed observed across all three DDT conditions in the HI group compared to the LI group in left frontal, temporal and parietal regions, also bilaterally in the occipital cortex.

(2) High impulsivity individuals were expected to show significantly increased theta band power compared to low impulsivity individuals, as a result of the association between increased theta band activity and impulsivity (Gui et al., 2018). The HI group was found to show more theta band power across the three conditions in left frontal, temporal, parietal and occipital regions, compared to the LI group.

(3) High impulsivity individuals were expected to show more alpha power (less alpha suppression) than low impulsivity individuals, indicating less attentional processing of the cue. The HI group indeed showed significantly more alpha band power than the LI group in frontal, temporal, parietal and occipital regions across the three DDT conditions. However, in the future reward Magnitude condition, the pattern was more mixed, with the HI group showing more alpha suppression than the LI group in right frontal, temporal and parietal regions, while

less alpha suppression was also observed in the left hemisphere and bilaterally in the occipital cortex.

(4) High impulsivity individuals were hypothesised to show less beta band power compared to low impulsivity individuals as a reflection of reduced behavioural suppression. Current findings were mixed, showing significantly more beta band power in left frontal, temporal, parietal and occipital regions and less beta band power in right frontal, temporal and parietal regions for the HI group compared to the LI group.

6.5.1. Differences in delay discounting: event-related fields

Considering previous findings, differences between high and low impulsivity individuals in temporal discounting were expected. Here, results from sensor- and source-level analyses of the ERFs during the three DDT conditions are discussed.

M1 component

Regarding the ERFs during the DDT and considering our previous MEG findings in RI, see Chapter 5 for details, I expected to find reduced M1 amplitude in the HI compared to the LI group, reflecting a possible attention deficit. Significantly reduced M1 amplitudes were found in the HI group during the Immediate, Magnitude and Delay conditions, compared to the LI group. Across the three conditions, a negative cluster was consistently found in the right parietal cortex, see Figures 6.1, 6.2 and 6.3. Only during the Magnitude condition, the significantly reduced amplitude of the M1 component in the HI group was also observed in the SMA, right temporal and left parietal regions, see Figure 6.2.

Electrophysiological studies have reported larger early evoked responses over the occipital cortex during the presentation of reward cues compared to the presentation of non-reward cues between 100ms and 155ms after stimulus onset (e.g., peaking at 100ms in Apitz & Bunzeck, 2012; peaking at 155ms in Thomas et al., 2013). Here, the significant differences were observed to last longer, between 140 and 190 ms after stimulus onset in the Immediate reward condition, between 90 and 210 ms in the future reward Magnitude condition and between 130 and 210 ms in the future reward Delay condition. Although significant negative clusters were also found in occipital regions, as previously reported (e.g., Apitz & Bunzeck, 2012; Doñamayor et al., 2012; Thomas et al., 2013), a significant negative cluster was consistently found across the three conditions in the right inferior parietal cortex, see Figures 6.1, 6.2 and 6.3.

A later effect in electrophysiological studies on reward cues compared to non-reward cues, at approximately 170ms after stimulus onset has also been reported in the fusiform gyrus (Apitz & Bunzeck, 2012) and at 200 ms over the dorsal PCC (Doñamayor et al., 2012). Interestingly, the HI group showed significantly reduced M1 amplitudes in the left fusiform gyrus in the Delay condition and in the middle cingulate cortex during the three DDT conditions compared to the LI group.

It could be argued that the group differences in M1 amplitude observed here include both the earlier and later effects previously reported (Apitz & Bunzeck, 2012; Doñamayor et al., 2012; Thomas et al., 2013). Current findings are also consistent with results from Chapter 5, in which the M1 amplitudes were significantly reduced during both NOGO and STOP conditions in the HI compared to the LI group, reflecting a potential attentional deficit.

M2 component

Larger amplitudes in the M2 component were expected in the HI group compared to the LI group. Specifically, during the Immediate reward condition, I expected to find a stronger frontal M2 component in the HI group, representing the preference for immediate rewards (Gui et al., 2016; 2018). Current results were partly consistent with these previous studies. Contrary to our expectations, the HI group showed significantly reduced M2 amplitudes in the future Magnitude and Delay conditions in the right parietal cortex, see Figures 6.2 and 6.3. This component has been reported to reflect the first valuation of the reward magnitude and delay (Gui et al., 2016). Here, the group difference on the M2 amplitude was specifically found during the future reward magnitude and delay conditions, which could suggest that in the HI group the reward evaluation process might occur differently and be reflected by reduced M/P2 amplitudes compared to the LI group.

Consistent with our hypothesis, significantly increased M2 amplitudes in the Immediate reward condition were also observed bilaterally in frontal regions in the HI compared to the LI group, see Figure 6.1. Specifically, a frontal N2 component has previously been suggested to be a potential candidate for group differences, reflecting an increased preference for immediacy, as it has been found to correlate with immediate rewards (Gui et al., 2016). Current findings provide further evidence for this, indicating the importance of the frontal Immediate-M2 component observed here as a marker of preference for immediacy. I believe the literature on this topic would benefit from further studies examining the differences in ERFs between healthy individuals scoring high and low on impulsivity. This could clarify the role of the M2 during non-immediate conditions.

M3 component

Regarding the M3 component, reduced amplitudes were expected in the HI group compared to the LI group, as smaller amplitudes have been suggested to represent dysfunctional reward processing (Kamarajan et al., 2015b). A previous EEG study which compared young individuals at high- and low-risk for alcoholism using a monetary gambling task, showed that in addition to reduced P3 amplitudes, the high-risk group also showed significantly higher scores on the BIS-11 questionnaire compared to the low-risk group. Here, results are partly consistent with this study, as reduced M3 amplitudes were found in the HI group during the Immediate condition in the SMA, middle cingulate cortex and medial part of the superior frontal gyrus, see Figure 6.1, and during the future reward Magnitude condition in right frontal, temporal and parietal regions, see Figure 6.2. Yet, in the Immediate condition, significant larger M3 amplitudes were also observed for the HI group in the left fronto-temporal cortex and occipital regions. Furthermore, in the future reward Delay condition, only larger M3 amplitudes in the HI compared to the LI group were observed in frontal, temporal and occipital regions, see Figure 6.3.

Conclusions from ERF findings during temporal discounting

Overall, the amplitude of the M1 component was observed to be reduced in individuals scoring high on impulsivity questionnaires compared to those scoring low, which could reflect an attentional deficit towards the stimuli in the HI group. The amplitude of the M2 is significantly larger in the HI group during the Immediate reward condition in frontal regions, but not during the future reward Magnitude and Delay conditions, which could reflect an increased preference for immediacy. The amplitudes of the M3 component were significantly reduced in the HI compared to the LI group in the Immediate and future reward Magnitude conditions, representing less efficient reward processing. Yet, the M3 amplitudes in the HI group were significantly larger during the future reward Delay condition in frontal, temporal and occipital regions, compared to the LI group, which does not seem to fit expectations.

Considering that the HI group did not show a behavioural choice significant difference compared to the LI, it is possible that the HI group had to recruit more cognitive resources to show a similar discounting rate compared to the LI group, which might be reflected by the stronger Delay-M3 component found in the HI.

6.5.2. Differences in delay discounting: oscillatory activity

The a priori hypotheses made here were based on electrophysiological studies investigating reward processing, reward anticipation and delivery (e.g., Doñamayor et al., 2012; Kamarajan et al., 2015), which have provided useful information regarding the oscillatory pattern of neural activity involved in reward processing.

Delta band

Since larger amplitudes in delta band have previously been associated with larger reward magnitudes and suggested to represent a reward-sensitive component of feedback processing (Bernat et al., 2011; 2015). It could be argued that activity in this band might reflect sensitivity towards reward and, specifically, towards larger reward magnitudes. Based on these results, increased delta power was predicted in the HI group compared to the LI group. As expected, significantly higher delta power in posterior and anterior sensors were found in the HI group in the three conditions, see Figures 6.4, 6.8 and 6.12. The location of the significant positive clusters observed in the HI group compared to the LI group were similar across all three conditions: left frontal, temporal, parietal and bilateral occipital regions showed significantly more delta band power. Only in the Immediate reward condition was a significant positive cluster in the right temporal cortex observed in addition to the clusters already mentioned, see Figure 6.4.

The significant increase in delta band power observed here in the HI group compared to the LI group, provides further support for the initial indications reported in previous studies (Bernat et al., 2011; 2015). This finding also suggests that HI individuals might show an increased sensitivity towards reward, compared to LI individuals, as reflected by higher delta power.

Theta band

In line with previous studies examining differences in oscillatory activity during temporal discounting in individuals with ADHD (e.g., Gui et al., 2018; Loo et al., 2013; Monastra et al., 2001), increased theta band power was found in the HI group in left frontal, temporal, parietal and occipital regions, see Figures 6.5, 6.9 and 6.13. Oscillatory activity in the theta band has been associated with cognitive processes such as alertness (Basar, 1999), focused attention and signal detection (Basar-Eroglu et al., 1992), short-term memory (Klimesch, 1999) and reward processing (Cohen et al., 2007; Kamarajan et al., 2008; 2015a; Marco-Pallares et al., 2008).

Contradictory results have been reported to date regarding theta in relation to impulsivity. In a study by Kamarajan and colleagues (2015a), adolescents and young adults at high risk for alcoholism were compared with participants at low risk, during a monetary gambling task.

Individuals at high risk showed lower theta power than individuals at low risk during reward processing and interestingly, individuals at high risk also showed increased impulsivity, as measured by the BIS-11, compared to individuals at low risk (Kamarajan et al., 2015a). Other studies have examined temporal discounting by comparing individuals diagnosed with ADHD with age-matched controls and reported individuals with ADHD to exhibit both impaired response inhibition (e.g., longer stop-signal traction time, Lijffijt et al., 2005) and higher delay discounting (e.g., Bitsakou et al., 2009; Paloyelis et al., 2010; Solanto et al., 2001),. In contrast to Kamarajan et al. (2015a) however, other research indicated that ADHD participants showed significantly increased theta power compared to controls during intertemporal choice (Gui et al., 2018; Loo et al., 2013; Monastra et al., 2001). Furthermore, Gui et al., (2018) suggested that increased impulsivity and increased theta band activity might be closely related.

It could be argued that because in the study by Kamarajan et al. (2015), the high-risk group showed lower theta power and also scored significantly higher on the BIS-11 questionnaire compared to the low-risk group, the HI group here would also show reduced theta band power compared to the LI group. Yet, other studies have reported the opposite, a significant increase in theta power in ADHD participants compared to controls during intertemporal choice (Gui et al., 2018; Loo et al., 2013; Monastra et al., 2001). The deficits observed in individuals diagnosed with ADHD, such as response inhibition and impulsivity temporal discounting, are in line with the characteristics of the HI group investigated here. In Chapter 3, results demonstrated that the delay-discounting task was more sensitive to differences between groups defined by the combination of two impulsivity factors, rapid-response and reward-delay impulsivity, than groups based on each dimension alone (Jauregi et al., 2018). Therefore, it is not surprising that current results are consistent with the increase in theta found in ADHD compared to controls (e.g., Gui et al., 2018; Loo et al., 2013; Monastra et al., 2001), in contrast to the findings from individuals at high risk for alcoholism who also scored high on the BIS-11 (Kamarajan et al., 2015a).

Significantly more theta band power was observed in the HI compared to the LI group across the three DDT conditions, sharing common locations of significant positive clusters in left frontal, temporal, parietal and occipital regions. Interestingly, during the Immediate condition a main difference was observed relative to the other two conditions, specifically, significantly more theta band power was observed bilaterally in frontal regions in source-space. This group difference was found in the SMA, middle frontal, right inferior and superior frontal cortex, while in the future reward Magnitude and Delay conditions, the differences were located predominantly in the left hemisphere, see Figure 6.9 and 6.14.

Based on previous studies on theta band activity, it could be argued that high impulsivity individuals here showed increased alertness (Basar, 1999) and reward processing (Cohen et al., 2007; Kamarajan et al., 2008; 2015; Marco-Pallares et al., 2008), as reflected by increased theta band power than low impulsivity individuals. Given that the HI group did not show a significant difference in behavioural choice compared to the LI, it is possible that the HI group had to recruit more cognitive resources to show a similar temporal discounting rate compared to the LI group, which might be reflected by the stronger theta band activity found in the HI.

Alpha band

Although there is limited knowledge of alpha band power during delay discounting, alpha power is considered to represent the inhibition of neural activity (Jensen & Mazaheri, 2010) and stronger alpha suppression has been previously reported following monetary-reward cues (Hughes et al., 2013). A previous study investigating response preparation and cue processing in adolescents diagnosed with ADHD using a cued flanker task, found significantly less alpha suppression in the visual cortex in this group compared to controls, which suggests reduced processing of the cue (Mazaheri et al., 2014).

Here, the HI group showed significantly more alpha band power than the LI group in frontal, temporal, parietal and occipital regions across the three DDT conditions, see Figures 6.6, 6.10 and 6.14. Only in the future reward Magnitude condition, the HI group showed more alpha suppression in source space than the LI group, specifically, in right frontal, temporal and parietal regions, while less alpha suppression was also observed in the left hemisphere and bilaterally in the occipital cortex, see Figure 6.10.

A study by van den Berg et al. (2014) demonstrated that after the presentation of reward-related cues in a Stroop task, individuals with increased alpha suppression over occipital regions displayed better behavioural performance on reward trials. It has been suggested that alpha power suppression reflects increased attentional processing during the expectation of reward-related cues (Pornpattananangkul & Nusslock, 2016). In their majority, the current findings suggest that the HI possibly recruit less attentional resources, as reflected by less alpha suppression observed in this group, compared to the LI group.

This result is consistent with those from ERFs, which showed significantly reduced M1 amplitudes across the NOGO, STOP and the three DDT conditions, which is considered to reflect a deficit in attentional processing of the stimuli (Vogel & Luck 2000). Nevertheless, the additional enhanced alpha suppression observed in the HI group during the future reward Magnitude condition, suggests that it was during the display of this condition only, which produced an increase in their attentional processing. It could be argued that the magnitude of a potential future reward is a fundamental factor for the decision-making process of choosing

the smaller immediate or delayed but larger future reward. Consistently, individuals scoring high on impulsivity showed enhanced attentional processing to the magnitude of a future reward.

Beta band

Current findings showed significantly more beta band power in left frontal, temporal, parietal and occipital regions and less beta band power in right frontal, temporal and parietal regions in the HI group compared to the LI group. Previous studies have shown that decreases in beta power are associated with motor control (Neuper et al., 2006) and because this decrease in beta has been found in sensorimotor areas, it has been suggested it could be related to motor preparation (Doñamayor et al., 2012). Here, significantly less beta power in the HI group was observed in the SMA across the three conditions, compared to the LI group, which is in line with these studies (e.g., Doñamayor et al., 2012; Neuper et al., 2006)., see Figures 6.7, 6.11 and 6.15.

Previous studies have also reported increased beta power for rewards compared to non-reward cues (Cohen et al., 2007; Doñamayor et al., 2012; Marco-Pallares et al., 2008). An increase in beta power in fronto-central areas specifically, has been associated with positive feedback compared to negative feedback in studies using gambling and feedback-learning tasks (Cohen et al., 2007; Doñamayor et al., 2012; Marco-Pallares et al., 2008). In the current study, no prominent fronto-central positive cluster was observed, however, significant positive clusters were found in left frontal, temporal, parietal and occipital areas in the HI group. These results could suggest an increased sensitivity towards reward-related cues, as reflected by the increased beta band power in the HI group compared to the LI group.

Conclusions on oscillatory activity during temporal discounting.

Young adults scoring high on self-report measures of impulsivity showed more delta, theta, alpha and beta band power across the three conditions, compared to those scoring low. Delta and theta band power followed a similar pattern, showing significant positive clusters in the HI group in frontal, temporal and parietal areas. This finding suggests that the HI group might show an increased sensitivity towards reward, compared to the LI group, as reflected by higher delta power. This is also in line with a previous study which suggested an association between impulsivity and increased theta band activity (Gui et al., 2018). Alpha band power was found to be predominantly increased in the HI group compared to the LI group in all conditions, suggesting reduced attentional processing (alpha suppression), but also revealed an additional reversed effect (stronger alpha suppression) in left fronto-temporo-parietal areas, but only in the future reward Magnitude condition. Only beta band power was found in the HI group to be also supressed across the three conditions in right frontal, temporal and parietal

regions, suggesting an increased sensitivity towards reward-related cues, compared to the LI group. These results suggest an increased sensitivity towards reward-related cues, as reflected by the increased beta band power, while also preparing to execute a motor response, as reflected by the decreased beta band power found in SMA for the HI group compared to the LI group.

6.6. Conclusions

This experiment investigated the neural correlates of delay discounting using MEG in a healthy undergraduate student population, divided by their level of impulsivity as assessed by self-report measures. Regarding the analyses of event-related fields, the amplitude of the M1 component was observed to be reduced in individuals scoring high on impulsivity questionnaires compared to those scoring low, which could reflect an attentional deficit towards the stimuli in the HI group. The amplitude of the M2 was significantly larger in the HI group during the Immediate reward condition in frontal regions, but not during the future reward Magnitude and Delay conditions, which could reflect an increased preference for immediacy. The amplitudes of the M3 component were significantly reduced in the HI compared to the LI group in the Immediate and future reward Magnitude conditions, which could represent less efficient reward processing. Yet, the M3 amplitudes in the HI group were significantly larger during the future reward Delay condition in frontal, temporal and occipital regions, compared to the LI group. Considering that the HI group did not show a behavioural choice significant difference compared to the LI, it is possible that the HI group had to recruit more cognitive resources to show a similar discounting rate compared to the LI group, which might be reflected by the stronger Delay-M3 component found in the HI.

Regarding the analysis of oscillatory activity, significantly increased power in delta, theta, alpha and beta frequency bands was observed in the HI compared to the LI group during the delay-discounting task. Current results suggest that the HI group might show an increased sensitivity towards reward, compared to the LI group, as reflected by higher delta and theta power during intertemporal choice. At source-level, the HI group only showed significantly more alpha suppression during the future reward Magnitude conditions, reflecting a possible attentional processing deficit towards the Immediate and future reward Delay cues, compared to the LI group. It could be argued that the future reward Magnitude cue, specifically, produced an increase in attentional processing in high impulsivity individuals. Low impulsivity individuals showed less beta power than those scoring high in impulsivity, which has been associated with motor control (Neuper et al., 2006). In the HI compared to the LI group, only beta band power was supressed across the three conditions, suggesting an increased sensitivity towards

reward-related cues. Even though this study is based in a young adult non-clinical population, these findings illustrate how trait impulsivity relates to differences in the neural correlates of delay discounting.

CHAPTER 7. General discussion

7.1. Summary of aims and background

Impulsivity, as a trait, is defined as “a predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions to the impulsive individual or to others” (Moeller et al., 2001; p. 1784). It is considered a multifaceted construct which comprises at least two different dimensions: rapid-response impulsivity and reward-delay impulsivity (Dawe et al., 2004; Dawe & Loxton, 2004; Winstanley et al., 2004; Alloy et al., 2009; Swann, 2010; MacKillop et al., 2016).

Despite the vast literature investigating impulsivity using cognitive and self-report measures, it is still unclear which aspects of trait impulsivity, as assessed by self-report measures, are related to rapid-response impulsivity and/or to reward-delay impulsivity, as different results have been reported in studies using both types of measures, see section 1.2.2.2. for details. This was addressed in Chapter 3, by conducting a behavioural experiment in a healthy undergraduate sample, in which self-report measures of trait impulsivity, the BIS-11 and BIS/BAS questionnaires, were administered to classify participants into high and low impulsivity groups. This classification was then related to cognitive measures of the two dimensions of impulsivity, the GNGT and SST for response inhibition and the DDT for delay discounting, along with other self-report measures of impulsivity, the UPPS and EPQ questionnaires. This study was aimed to clarify the robustness of self-report questionnaires as genuine predictors of Behavioural impulsivity. Group differences were examined using MANCOVA and correlations between all measures of impulsivity were also analysed.

The data collected during the behavioural study was examined differently in Chapter 4, by taking advantage of different approaches, such as exploratory factor analysis and structural equation modelling. Although the multi-dimensional construct of impulsivity is generally accepted, agreement about how many dimensions it consists of is lacking. Previous studies have used factor analysis and SEM on both self-report and behavioural measures of impulsivity and have provided well fitted models. Most studies have included cognitive measures which assess reward sensitivity but did not include specific psychometric measures of reward sensitivity (e.g., Cyders & Coskunpinar, 2012; MacKillop et al., 2016). Here, an exploratory factor analysis was conducted which, in addition to the most widely used measures of impulsivity, also included venturesomeness-related scales, such as sensation-seeking, and measures of reward-delay sensitivity, such as the BAS subscale of the BIS/BAS scale. The aim was to test the hypothesis that a three-factor model of impulsivity as reported previously (MacKillop et al., 2016), would benefit from the inclusion of a specific psychometric measure of reward-delay impulsivity, and yet, be differentiated into three latent constructs. To test this hypothesis, a principal axis factoring analysis was conducted. The resulting model and its set

of relationships were then tested using SEM, with structural path analyses examining the associations between identified factors.

Considering the results found in Chapter 3 and 4, see Tables 7.1 and 7.2, it was clear that the cognitive measures of impulsivity examined were sensitive enough to pick-up the differences between impulsivity groups, as assessed by self-reports. Findings from both analyses also provided further evidence for the necessity of including both dimensions of impulsivity in any further assessment, to capture as much as possible of the impulsivity construct. The next step involved examining these behavioural group differences further, by using neuroimaging methods. For this purpose, MEG was used, which allowed the investigation of potential group differences in the spectral and spatio-temporal dynamics of response inhibition (Chapter 5) and delay discounting (Chapter 6). The spatial resolution of MEG when coupled with individual MRI data and MEG's millisecond temporal resolution, made it ideally suited for this objective.

Although there is limited data on the spectral and spatio-temporal dynamics of response inhibition, previous studies have reported differences in the neural correlates of response inhibition in high impulsivity individuals compared to those with low impulsivity, see section 1.3.2. In Chapter 5, the neural correlates of response inhibition were investigated using MEG in a healthy undergraduate student population, divided by their level of impulsivity as assessed by self-report measures. This study also aimed to investigate the conflicting results from previous studies. Differences between groups in event-related fields and oscillatory activity were tested statistically at sensor- and source-level, allowing the direct comparison of individuals scoring high and low on impulsivity measures during a combined Go/No-Go and Stop-Signal Task. The use of a combined version of the response inhibition tasks tested in the behavioural study, provided a more comprehensive assessment of this dimension of impulsivity compared to previous studies, which have predominantly used one of the two, see section 1.3.2.1.1.

Regarding reward-delay impulsivity, most of the previous literature comes from studies using fMRI during monetary gambling tasks, while some have used delay discounting tasks (e.g., Ballard & Knutson, 2009; Sripada et al., 2011). There is limited data on the spectral and spatio-temporal dynamics of delay discounting (also referred to as temporal discounting or intertemporal choice), during which high impulsivity individuals have been found to decide more impulsively, see section 1.3.2.1.2. In Chapter 6, the experiment aimed to fill the current gaps found in the literature on reward-delay impulsivity, by investigating the neural correlates of delay discounting using MEG in a healthy undergraduate student population, which was divided into high and low impulsivity groups. Again, differences between groups in event-related fields and oscillatory activity were tested statistically at sensor- and source-level.

The comprehensive analyses conducted here on the most widely used cognitive and self-report measures of impulsivity, provided a detailed picture of the behavioural and neural correlates that distinguished impulsive individuals in various measures. In the following sections, the main findings of each chapter are discussed, then limitations and future directions are described, ending with the final conclusions.

7.2. Principal findings

7.2.1. Linking cognitive measures of response inhibition and reward sensitivity to trait impulsivity

In the experiment described in Chapter 3, different cognitive and self-report measures of impulsivity were compared within a sample of undergraduate students. The aim was to clarify which aspects of trait impulsivity are related to response inhibition and which to reward responsiveness. For this purpose, I examined how sensitive these measures are to detect differences between groups classified as being low in impulsivity and high in impulsivity, based on two factors: level of rapid-response impulsivity (Swann et al., 2009) and reward-delay impulsivity (Alloy et al., 2006). Results are summarised in Table 7.1 and partly supported our hypotheses:

1) Participants in the high rapid-response impulsivity group as measured by scores on the BIS-11 Motor subscale, showed significantly higher reward-delay impulsivity, both on the cognitive DDT task and the BIS/BAS self-report measures assessing reward sensitivity. No associations between self-report measures and cognitive tasks measuring rapid-response impulsivity were observed. Our results, therefore, suggest that the BIS-11 Motor subscale might not be assessing rapid-response impulsivity in the same way as the cognitive tasks assessing response inhibition. This finding is in line with other studies stating a lack of associations between cognitive tasks assessing impulsivity and (self-report) trait measures of impulsiveness (e.g., Kulendran et al., 2016).

The high reward-delay impulsivity group not only showed significantly higher trait impulsivity compared to the low reward-delay impulsivity group, but also performed significantly different on the reward-delay impulsivity task, the DDT. Consistent with previous findings (e.g., Alloy et al., 2006, 2009), our results indicate that participants with high reward-delay impulsivity show higher trait impulsivity and higher rapid-response impulsivity. This provides further evidence for reward-delay impulsivity being a crucial characteristic of trait impulsivity.

(2) The high-impulsivity group, as defined by high scores on rapid-response impulsivity as well as on reward-delay impulsivity self-report measures, showed significantly reduced task

performance on the GNGT and SST and, on the DDT, preferred small but immediate rewards over larger, delayed rewards significantly more often than the low-impulsivity group. The latter indicates a more pronounced reward-delay impulsivity than that for the low-impulsivity group. While differences between high- and low-impulsivity groups were less clear when individuals were categorised based on either impulsivity dimension alone, differences between high- and low-impulsivity groups were more specific, and pronounced, when combining both dimensions.

Chapter	Findings
3	• The high rapid-response impulsivity group showed significantly higher reward-delay impulsivity on both, the DDT and on self-report measures of reward-delay impulsivity, than the low-risk group. • The high reward-delay impulsivity group scored significantly higher on task-based and self-report measures assessing rapid-response impulsivity than the low reward-delay impulsivity group. • Combining both dimensions of impulsivity showed that the high-impulsivity group showed significantly reduced task performance in rapid-response paradigms and temporally discounted significantly more impulsively than the low-impulsivity group. • Lack of associations between response inhibition tasks and self-reports of impulsivity.

Table 7.1. Results summarised in relation to Chapter 3.

Our results suggest that self-report measures of rapid-response impulsivity alone may not be sensitive enough to pick up the differences between groups in response inhibition paradigms. However, when including a related, and perhaps necessary, dimension, reward-delay impulsivity, differences between groups were observed. Therefore, it was the combination of the two dimensions of impulsivity that provided a more sensitive assessment.

Correlations between the cognitive tasks and self-report measures were also examined, since mostly contradictory results have been reported to date (Rodrìguez-Fornells et al., 2002; Horn et al., 2003; Spinella, 2004; Keilp et al., 2005; Lijffijt et al., 2005; Enticott et al., 2006; Reynolds et al., 2006; Aichert et al., 2012; Malesza & Ostaszewski, 2016). It was hypothesised that the cognitive tasks would significantly correlate with self-reports measuring different aspects of impulsivity. This was confirmed for the DDT, as there were significant positive correlations with the BIS-11 Total scale and the BIS-11 Non-Planning subscale. However, the rapid-response

impulsivity tasks (GNGT, SST) did not directly correlate with any of the self-report measures. Researchers have suggested that this is because behavioural approaches measure task performance during a limited and exact moment in time, while questionnaires focus on self-reported trait impulsivity manifested across time and different situations (Cyders and Coskunpinar, 2011; Lane, Cherek, Rhoades, Pietras, & Tcheremissine, 2003; Reynolds et al., 2006; Swann et al., 2010; Dougherty et al., 2014). It has been suggested that the fusion, or amalgamation of the different concepts of impulsivity has resulted in such inconsistencies (Cyders & Coskunpinar, 2011).

Our findings therefore corroborate previous results (e.g., Alloy et al., 2006, 2009), which indicate that participants with high reward-delay impulsivity show higher trait impulsivity and higher rapid-response impulsivity. However, results here suggest that the BIS-11 Motor subscale might not be assessing rapid-response impulsivity in the same way as the cognitive tasks assessing response inhibition. Although some studies have reported significant associations between self-reports and response inhibition tasks (e.g., Cyders & Coskunpinar, 201; Gay et a., 2008; Marsh et al., 2002; Sharma et al., 2014; see section 1.2.2.2 for details), current findings are line with other studies stating a lack of associations between cognitive tasks assessing impulsivity and trait measures of impulsiveness (e.g., Cheung et al., 2004; Enticott et al., 2006; Keilp et al., 2005; Horn et al., 2003; Kulendran et al., 2016; Reynolds et al., 2006). It is not uncommon to find in previous research of differences between high and low impulsivity groups, studies in which either self-reports or laboratory tasks were used to measure impulsivity (e.g., Alloy et al., 2006; Chen et al., 2005; Lopez-Caneda et al., 2017; Ruchsow et al., 2008), or studies that assessed one dimension of impulsivity but not the other (e.g., Dimoska & Johnstone, 2007; Kamarajan et al., 2015a; 2015b; Ruchsow et al., 2008; Rubio et al., 2008). It could be argued that this lack of consistency across methods might bias how impulsivity is conceptualised. Thus, current findings provide further evidence that self-report and laboratory task measures should be used in conjunction.

Both reward-delay and rapid-response impulsivity have been suggested to represent shared vulnerabilities to some mental illnesses, such as alcohol and drug misuse/dependence (Nigg et al., 2006; Rubio et al., 2008), and bipolar spectrum disorders (Alloy et al., 2006), in young and adult populations. Considering that current results show that combined impulsivity factors, rapid-response and reward-delay impulsivity, provide better assessment of impulsivity than each dimension alone, and that the mentioned studies only tested one of the two dimensions, arguably, the investigation of both dimensions might be a more suitable approach.

7.2.2. Towards a unified conceptualisation of impulsivity: a factor analytical investigation of self-report and behavioural measures

Although it is widely accepted that impulsivity is a multi-dimensional construct (Alloy et al., 2009; Dawe & Loxton, 2004; Dawe et al., 2004; MacKillop et al., 2016; Swann, 2010) there is no general agreement about how many dimensions it consists of (Malesza & Ostaszewski, 2016), nor how it should be conceptualised. Instead, the concept of impulsivity adopted by different researchers seems to depend upon their personal theoretical perspective (some influenced by personality theories and others not) and preferred methods of study (behavioural tasks or self-report questionnaires). This has led to terminological confusion and an ongoing debate about what impulsivity really is (Gullo et al., 2014; Hamilton et al., 2015).

Previous studies have used factor analysis and structural equation modelling on both self-report and behavioural measures of impulsivity and have provided well fitted models. Most studies have included laboratory tasks which assess reward sensitivity but did not include specific psychometric measures of reward sensitivity (e.g., Cyders & Coskunpinar, 2012; MacKillop et al., 2016). Considering the results from the behavioural experiment, in which the combination of scores on self-reports measuring the two dimensions of impulsivity provided a better assessment, it could be argued that the inclusion of psychometric measures of reward sensitivity might benefit previous models (e.g., Cyders & Coskunpinar, 2012; MacKillop et al., 2016).

Here, an exploratory factor analysis was conducted which, in addition to the most widely used measures of impulsivity, also included venturesomeness-related scales, such as sensation-seeking, and measures of reward-delay sensitivity, such as the BAS subscale of the BIS/BAS scale. The aim of this study was to test the hypothesis that a three-factor model of impulsivity as reported previously (MacKillop et al., 2016), would benefit from the inclusion of a specific psychometric measure of reward-delay impulsivity, and yet, be differentiated into three latent constructs. To test this hypothesis, a principal axis factoring analysis was conducted. This is the most appropriate method when the main objective is to examine the structure of the data, as it identifies the least number of factors that can explain common variance. The resulting model and its set of relationships were then tested using SEM, with structural path analyses examining the associations between identified factors.

Our hypothesis was partly supported, see Table 7.2 for a summary of principal findings. Both, the exploratory factor analysis and the SEM approach supported a three-factor model. However, the structure of our model was different to a model proposed by MacKillop et al., (2016), and including a measure assessing reward sensitivity did not improve model fit. The three-factor model comprised psychometric measures of rapid-response impulsivity,

measures of reward-delay impulsivity and response inhibition behavioural tasks. Even though these SEM results can only be interpreted in a descriptive manner, the three-factor model showed a better model fit compared to one- and two-factor models. The latent constructs and their organisation within this model were different to that from MacKillop and colleagues (2016). The model presented here consisted of three latent constructs: rapid-response impulsivity, reward-delay impulsivity and behavioural impulsivity; the first two constructs included self-report measures of impulsivity, whereas the third included laboratory task measures of impulsivity. The model presented in MacKillop et al. (2016) also consisted of three latent constructs: impulsive action, impulsive choice and impulsive personality traits; in contrast to our model, the first two latent constructs included laboratory tasks, whereas the third construct included self-report measures of impulsivity. This difference between models is likely due to the inclusion of reward sensitivity and sensation seeking self-report measures.

Importantly, these results corroborated what many researchers have emphasised before and what was also observed in the behavioural study (Chapter 3): self-report and behavioural measures of impulsivity do not correlate well. In an attempt to explain this observation, it has been suggested that behavioural approaches measure task performance during a limited and at an exact moment in time, whereas questionnaires focus on self-reported trait impulsivity, which may be manifested across time and in different situations (Cyders & Coskunpinar, 2011; Reynolds et al., 2006; Swann et al., 2010; Dougherty et al., 2014). These findings also strengthened that rapid-response impulsivity and reward-delay impulsivity are two major yet different dimensions which contribute independently to the multifaceted nature of the impulsivity construct.

Chapter	Findings
4	• Best fit provided by a three-factor model comprising: psychometric measures of rapid-response impulsivity, measures of reward-delay impulsivity and response inhibition behavioural tasks. • Including a reward sensitivity psychometric measure did not improve the model fit. • Lack of associations between tasks and self-reports of impulsivity. • Rapid-response impulsivity and reward-delay impulsivity are two major yet different dimensions which contribute independently to the multifaceted impulsivity construct.

Table 7.2. Results summarised in relation to Chapter 4.

In MacKillop et al. (2016), the laboratory tasks conformed two different latent constructs, whereas self-reports measuring different aspects of trait impulsivity conformed a single latent construct. Here, although a cognitive measure of reward-delay impulsivity, the DDT, was initially included, it had to be excluded from the model. It is possible that this is due to the inclusion of different measures of impulsivity, compared to MacKillop's.

7.2.3. MEG Experiment 1: rapid-response impulsivity

Previous studies have reported differences in the spectral and spatio-temporal dynamics of response inhibition in high impulsivity individuals compared to those with low impulsivity, see section 1.3.2. Considering the behavioural differences found in the behavioural experiment (Chapter 3) between high and low impulsivity groups, the primary aim of the current experiment was to examine these behavioural differences in response inhibition using MEG. This was conducted in a healthy undergraduate student population, divided by their level of impulsivity as measured by questionnaires, see section 2.4.4 for details.

The HI group showed significantly reduced task performance than the LI group, as reflected by significantly more commission errors on NOGO and STOP trials in HI group. Although mean SSRT was higher in the HI group than in the LI group, this difference was not significant. Differences between groups in event-related fields and oscillatory activity were tested statistically at sensor- and source-level, allowing a direct comparison of individuals scoring high and low on impulsivity measures during a combined Go/No-Go and Stop-Signal Task.

Event-related fields of response inhibition

As expected, significant differences were found between HI and LI groups during this combined Go/No-Go/Stop-Signal task, while sensor- and source-level results were mostly consistent, see Table 7.3 for details. The HI group showed significantly reduced amplitudes of NOGO-M1 and STOP-M1 components in right inferior parietal regions compared to the LI group, see Figures 5.1 and 5.2. The right parietal cortex has been reported to be involved in attentional orientation processes in previous clinical and neuroimaging studies (Karnath et al. 2001; Brunetti et al. 2005). Current results suggest that the HI group shows a deficit in the attentional processing of stimuli.

Enhanced frontal M2 components have previously been reported in successful compared to unsuccessful inhibition (Schmajuk et al., 2006) and in participants with fast compared with slow SSRTs (van Boxtel et al., 2001). In clinical populations for example, children with ADHD have shown reduced M2 when compared with controls (Pliszka et al., 2000), Consistent with these studies, reduced NOGO-M2 and STOP-M2 amplitudes were found in frontal regions in the HI group compared to the LI group, see Figures 5.1, 5.3 and 5.4. However, it is unclear what the M2 represents in response inhibition paradigms (Folstein & Van Petten, 2008;

Kramer et al., 2011). During the GNGT, the frontal NOGO-M2 observed has been suggested to reflect the inhibitory process (Kok, 1986), while others have associated this component with a cognitive control process (Folstein & Van Petten, 2008). Previous studies using the SST have considered the STOP-N2 to serve as a marker of prefrontal inhibitory control (Schmajuk et al., 2006; Ramautar et al., 2004, 2006; van Boxtel et al., 2001). It could be argued that, here, individuals scoring high on impulsivity showed a deficit in the inhibitory process, reflected as a significantly reduced task performance and reduced NOGO-M2 and STOP-M2 components, compared to those scoring low on impulsivity measures.

Previous studies have shown that the N2 peaks earlier in successful trials compared to unsuccessful trials (Ramautar et al., 2004; 2006). This suggests that an early N2 component might be associated with successful response inhibition. The current findings are in line with these findings, as low impulsivity individuals showed an earlier STOP-M2 component than HI individuals. In the HI group, the network involved in the inhibition process, as reflected by the M2 component, is engaged later, resulting in more commission errors, or reduced task performance, than in LI individuals. Interestingly, this difference was not observed in NOGO trials.

Our current results add further evidence to previous reports, which have suggested that the NOGO-N2 component might serve as a measure of response inhibition efficiency (Schmiedt-Fehr & Basar-Eroglu, 2011). When considering that the HI group showed reduced task performance and a reduced NOGO-M1 component, our results suggest that high impulsivity individuals are less efficient in the (pre-) motor inhibition process than the low impulsivity individuals.

Differences between groups in the NOGO-M3 component were not observed in our study. This lack of significant differences suggests the HI and LI groups were monitoring successful motor inhibition to similar degrees, see Figures 5.1 and 5.5. The STOP condition showed significant differences between groups, specifically, increased STOP-M3 amplitude in the HI group compared to the LI group was observed in bilateral frontal, parietal and temporal regions. During the STOP-M3, the HI group engaged frontal networks significantly more than the LI group, possibly as a compensatory strategy for their response inhibition impairment. It has been suggested the larger amplitude in STOP-P3 in HI individuals reflects the demand for increased inhibitory effort in those scoring high as a compensatory strategy (Dimoska & Johnstone, 2007; Lansbergen et al., 2007). Current results favour the compensatory strategy proposed in these studies. These findings might also dispute whether the STOP-P3 reflects the evaluation of the inhibitory process (as suggested by Kok et al., 2004) or not.

Furthermore, the later STOP-M2H component and the significant group difference found during the STOP-M3 compared to their equivalents in NOGO trials, provide further evidence for task-related differences described in Chapter 1, see section 1.3.2 for details.

Chapter	Findings	
5	ERFs	• Reduced amplitudes of NOGO-M1 and STOP-M1 components in the right inferior parietal cortex among other regions in the HI group compared to the LI group. • Reduced NOGO-M1 and STOP-M1 in the HI group reflects a possible deficit in the attentional processing of stimuli. • Reduced NOGO-M2 and STOP-M2 amplitudes bilaterally in frontal and parietal regions in the HI group compared to the LI group. • The peak amplitude of the STOP-M2 component happened earlier in the LI (STOP-M2L) than in the HI group (STOP-M2H). This difference was not observed in NOGO trials. • In the HI group, the network involved in the inhibition process, as reflected by the M2 component, is engaged later, resulting in more commission errors than in LI individuals. • Differences between groups in the NOGO-M3 component were not found. • Increased STOP-M3 amplitude in the HI group compared to the LI group in bilateral frontal, parietal and temporal regions. • During the STOP-M3, the HI group engaged frontal networks significantly more than the LI group, possibly as a compensatory strategy.

Table 7.3. Results summarised in relation to Chapter 5, ERFs.

Results from source-level analysis are consistent with previous fMRI studies, which reported activation during the NOGO condition in parietal (Dambacher et al., 2014; Rubia et al., 2001; Sebastian et al., 2012; Swick et al., 2011) and pre-SMA regions (Dambacher et al., 2014; Sebastian et al., 2013; Swick et al., 2011). Findings regarding the STOP condition are also consistent with prior studies, showing significant activations in the pre-SMA (Dambacher et al., 204; Sebastian et al., 2013; Swick et al., 2011) and anterior cingulate cortex (Dambacher

et al., 2014). As previously mentioned, activity in the pre-SMA might be directly related to successful stopping, as the STOP-M2L and STOP-M3 showed group differences in this area.

Oscillatory activity in response inhibition

Sensor- and source-level analyses were mostly consistent with each other, except for beta, which will be discussed in the current section, see Table 7.4 for a summary of findings. Significant power decrease in delta and theta band power was observed in high impulsivity individuals compared to low impulsivity individuals in frontal regions during both NOGO and STOP conditions, see Figures 5.6, 5.7, 5.10 and 5.11. This finding is consistent with previous studies (e.g., Colrain et al., 2011; Kamarajan et al., 2004; Lopez-Caneda et al., 2017) and provides further support for a possible deficit in frontal pathways involved in the suppression of a motor response in individuals scoring high on impulsivity.

Results presented here suggest that in individuals scoring high on impulsivity, impairment in response inhibition might be a consequence of a deficit in the inhibitory process itself. This is reflected by decreased delta and theta power, along with significantly lower alpha power, which has been suggested to reflect an early attentional deficit that might affect the inhibition process (Pandey et al., 2016). Although comparisons between unsuccessful and successful stopping trials were not possible in the current experiment, see section 2.8.1 for details, results are in line with previous literature, supporting a possible attentional processing deficit. Current findings indicate that decreased anterior and increased posterior alpha activity during STOP and NOGO trials might characterise high impulsivity individuals. Source-level localisation of the power difference between the HI and LI groups showed, in both conditions, a significant decrease in delta, theta and alpha frequencies, predominantly in left frontal regions in the high impulsivity group. This is in line with our findings from sensor-space analyses, see Figures 5.8 and 5.12.

Previous studies suggested that an increase in beta power in frontal areas during NOGO trials reflects motor inhibition (Alegre et al., 2004). Sensor-level analysis of beta band power showed significant positive clusters in the HI group compared to the LI group, in central and posterior sensors during NOGO trials and in posterior sensors during STOP trials. These analyses also showed a significant decrease in beta band in frontal sensors in the HI group compared to the LI group during NOGO trials, and in frontal and central sensors in STOP trials. However, results at source-level showed that the difference in beta power during NOGO and STOP conditions was predominantly positive for the HI group, showing significant clusters in frontal, temporal and parietal regions, see Figures 5.9 and 5.13. Although this finding was not expected, similar results were observed during both conditions in all four frequency bands, and beta band is no exception. The increase in beta power in high compared to low impulsivity

individuals found during the NOGO and STOP conditions suggests that beta might be involved in response inhibition in a different way to delta, theta and alpha bands.

Chapter	Findings	
5	TFRs	• Power decrease in delta and theta bands was observed in the HI group compared to the LI group in frontal regions during both NOGO and STOP conditions. • Possible deficit in frontal pathways involved in the suppression of a motor response in the HI group. • Power decrease in alpha band in frontal regions in the HI group compared to the LI group during NOGO and STOP trials. • Possible deficit in attentional processing in the HI group, as indicated by lower alpha activity in frontal regions. • Power decrease in beta band in frontal sensors in the HI group compared to the LI group during NOGO trials and in frontal and central sensors in STOP trials. • Power increase in beta band in central and posterior sensors in the HI group compared to the LI group during NOGO trials and in posterior sensors during STOP trials. • Source-level analyses showed predominant increased beta band power during NOGO and STOP conditions in the HI group compared to the LI group in frontal, temporal, parietal and occipital regions.

Table 7.4. Findings related to Chapter 5, TFRs.

Altogether, current findings showed decreased low frequency (delta and theta) power in frontal regions, reflecting a deficit in executive control, i.e. inhibitory processing, in high impulsivity individuals. Also, results showed decreased alpha power, reflecting decreased visual suppression or increased visual attention, which results in reduced motor suppression in individuals scoring high on impulsivity compared to those scoring low. These results support previous findings (e.g., Colrain et al., 2011; Kamarajan et al., 2004; Lopez-Caneda et al., 2017) and those from the behavioural experiment conducted here (Chapter 3), which showed a deficit in response inhibition in high impulsivity individuals. This deficiency was reflected in

significantly reduced task performance and significant differences in the neural correlates of the inhibition process, previously found to be related to this inhibitory deficit, in the HI group compared to the LI group.

7.2.4. MEG Experiment 2: Reward-delay impulsivity

Although there is limited data on the spectral and spatio-temporal dynamics of delay discounting, previous studies have reported differences in the neural correlates between high impulsivity individuals and those with low impulsivity, see section 1.3.2. Considering the behavioural differences found in the behavioural experiment (Chapter 3) between high and low impulsivity groups, the primary aim of the current experiment was to examine these behavioural differences in temporal discounting using MEG. This was conducted in a healthy undergraduate student population, divided by their level of impulsivity as measured by questionnaires, see section 2.4.4 for details.

Behaviourally, the HI group temporally discounted in a more impulsive manner than the LI group, but not significantly so. Differences between groups in event-related fields and oscillatory activity were then tested statistically at sensor- and source-level, allowing the direct comparison of individuals scoring high and low on impulsivity measures during the DDT.

Event-related fields of temporal discounting

As expected, significant differences were found between HI and LI groups during the delay-discounting task, see Table 7.5 for a summary of findings. First, significantly reduced M1 amplitude in the HI group compared to the LI group was observed across the three DDT conditions, see Figures 6.1, 6.2 and 6.3. Although significant negative clusters were also found in occipital regions, as previously reported (e.g., Apitz & Bunzeck, 2012; Doñamayor et al., 2012; Thomas et al., 2013), a significant negative cluster was consistently reported across the three conditions in the right inferior parietal cortex. Only during the Magnitude condition, a reduced amplitude of the M1 component in the HI group was also observed in the SMA, and in right temporal and left parietal regions, see Figure 6.2.

The duration of the difference in the M1 amplitude observed between groups suggests that this component includes both the earlier and later components previously reported (Apitz & Bunzeck, 2012; Doñamayor et al., 2012; Thomas et al., 2013). Current findings are also consistent with results from Chapter 5, in which the M1 amplitudes were significantly reduced during both NOGO and STOP conditions in the HI compared to the LI group, reflecting a potential attentional deficit.

During the Immediate reward condition, a stronger frontal M2 component in the HI group was expected, representing a preference for immediate rewards (Gui et al., 2016). Current results were consistent with our prediction and a study by Gui et al., (2016), showing significantly increased M2 amplitude in the HI group during the Immediate reward condition in frontal regions, see Figure 6.1. However, the HI group also showed significantly reduced M2 amplitudes in the future Magnitude and Delay conditions in the right parietal cortex, see Figures 6.2 and 6.3 for details. The M2 component has previously been reported to reflect the first valuation of reward magnitude and delay (Gui et al., 2016). Here, the group difference on the M2 amplitude was specifically found during the future reward Magnitude and Delay conditions, which could suggest that in the HI group the reward evaluation process might occur differently and be reflected by larger M2 amplitudes compared to the LI group. Importantly, these results provide further support for the M2 component as a potential marker of preference for immediacy, as the mean N2 peak amplitude has previously been observed to correlate with the percentages of immediate choices (Gui et al., 2016).

Reduced M3 amplitudes were expected in the HI group compared to the LI group, as smaller amplitudes have been found in young individuals at high risk for alcoholism, that also scored significantly higher on the BIS-11 questionnaire (Kamarajan et al., 2015a), and have been suggested to represent dysfunctional reward processing (Kamarajan et al., 2015b). Current results were partly consistent with these studies, as significantly reduced M3 amplitudes were found in the HI group during the Immediate and future reward Magnitude conditions in frontal, temporal and parietal regions, see Figures 6.1 and 6.2. Yet, in the Immediate condition, significant larger M3 amplitudes were also observed in the left fronto-temporal cortex and occipital regions. Furthermore, in the future reward Delay condition, only larger M3 amplitudes in the HI compared to the LI group were observed in frontal, temporal and occipital regions, see Figure 6.3.

It could be argued that, based on these results, high impulsivity individuals show less efficient reward processing of the Immediate and future reward Magnitude cues, and increased reward processing of the future reward Delay cue relative to the low impulsivity individuals. Considering that the HI group did not display a significant difference in behavioural choice compared to the LI, it is possible that the HI group had to recruit more cognitive resources to show a similar temporal discounting rate compared to the LI group, which might be reflected by the stronger M3 in the HI. Larger amplitudes were also observed in STOP-P3 in HI than in LI individuals, which has been suggested to represent the demand for increased inhibitory effort in those scoring high as a compensatory strategy (Dimoska & Johnstone, 2007; Lansbergen et al., 2007). Given the larger M3 amplitudes observed during the future Delay condition in the HI group, results suggest that the potential compensatory strategy might have

been effective during the DDT, as no significant differences were found between groups in their discounting rate. However, the HI group did show significantly reduced task performance compared to the LI group during the response inhibition task, which suggests the compensatory strategy might not have been effective enough to overcome their impairment.

Chapter	Findings	
6	ERFs	• Reduced M1 amplitude in the HI group compared to the LI group in the right parietal cortex, across the three conditions. Only during the Magnitude condition, the reduced amplitude of the M1 component in the HI group was also observed in the SMA, right temporal and left parietal regions. • Consistent reduced M1 amplitude observed in HI indicates a deficit in attentional processing of the stimuli. • Increased M2 amplitude in the HI group during the Immediate reward condition in frontal regions, but not during the future reward Magnitude and Delay conditions. • Immediate-M2 component suggested to serve as a marker of preference for immediacy. • Reduced M3 amplitude in the HI compared to the LI group in the Immediate and future reward Magnitude conditions in frontal, temporal and parietal regions. • Smaller M3 amplitudes suggested to represent less efficient reward processing. • Increased M3 amplitudes in the HI group during the future reward Delay condition in frontal, temporal and occipital regions, compared to the LI group.

Table 7.5. Principal findings related to Chapter 6, ERFs.

These findings might also dispute whether the STOP-P3 reflects the evaluation of the inhibitory process (as suggested by Kok et al., 2004) or not. Future research on the M3 component during the DDT, specifically during the future reward Delay condition in high impulsivity individuals, would reveal if this finding is seen in other impulsive populations. The potential attentional deficit and preference for immediacy, coupled with the deficient reward processing of the Immediate and future reward Magnitude cues observed in the HI group,

support previous findings (e.g., Gui et al., 2016; Kamarajan et al., 2015a; b), while also providing novel results.

Oscillatory activity in temporal discounting

As expected, significantly higher delta and theta power were found in the HI group on the three DDT conditions when compared to the LI group in frontal, temporal and parietal areas, see Table 7.6 for a summary of findings. Larger amplitudes in delta band have previously been associated with larger reward magnitudes. These have been suggested to represent a reward-sensitive component of feedback processing (Bernat et al., 2011; 2015), which was observed in the HI group, see Figures 6.4, 6.8 and 6.12. This finding suggests that HI individuals might show an increased sensitivity towards reward, compared to LI individuals, as reflected by significantly higher delta power.

In line with previous studies examining differences in oscillatory activity controls during intertemporal choice in individuals with ADHD (e.g., Gui et al., 2018; Loo et al., 2013; Monastra et al., 2001), significantly increased theta band power was found in the HI group in left frontal, temporal, parietal and occipital regions, see Figures 6.5, 6.9, 6.13 and Table 7.6. Interestingly, during the Immediate condition, a main difference was observed relative to the other two conditions. Specifically, significantly more theta band power was observed bilaterally in frontal regions in source-space in the HI group, see Figure 6.9. The increased theta power found in high impulsivity individuals provides further evidence for an association between increased theta band activity and impulsivity (Gui et al., 2018).

Although there is limited knowledge of alpha band power during delay discounting, alpha power is considered to represent the inhibition of neural activity (Jensen & Mazaheri, 2010) and stronger alpha suppression has been previously reported following monetary-reward cues (Hughes et al., 2013). A previous study investigating response preparation and cue processing in adolescents diagnosed with ADHD using a cued flanker task, found significantly less alpha suppression in the visual cortex in this group compared to controls, which suggests reduced processing of the cue (Mazaheri et al., 2014). Here, the HI group showed significantly more alpha band power than the LI group in frontal, temporal, parietal and occipital regions across the three DDT conditions, see Figures 6.6, 6.10 and 6.14 and Table 7.6. Only in the future reward Magnitude condition did the HI group show more alpha suppression than the LI group, in right frontal, temporal and parietal regions, while less alpha suppression was also observed in the left hemisphere and bilaterally in the occipital cortex, see Figure 6.10.

A study by van den Berg et al. (2014) demonstrated that after the presentation of reward-related cues in a Stroop task, individuals with increased alpha suppression over occipital regions displayed better behavioural performance on reward trials. Thus, it has been

suggested that alpha power suppression reflects increased attentional processing during the expectation of reward-related cues (Pornpattananangkul & Nusslock, 2016). Altogether, current findings suggest that the HI group possibly recruited less attentional resources, as reflected by less alpha suppression observed in this group, compared to the LI group.

This result is also consistent with those from ERFs, which showed significantly reduced M1 amplitudes across the NOGO, STOP and the three DDT conditions, which is considered to reflect a deficit in attentional processing of the cue (Vogel & Luck 2000). Nonetheless, the additional enhanced alpha suppression observed in the HI group during the future reward Magnitude condition, suggests that it was the display of this condition only that produced an increase in their attentional processing.

On the one hand, regarding beta, low impulsivity individuals showed less beta power than those scoring high on impulsivity measures, and this beta power decrease has been associated with motor control (Neuper et al., 2006). Interestingly, because the power decrease in the beta band has been found in sensorimotor areas, it has been suggested that it could be related to motor preparation (Doñamayor et al., 2012). Current results provide further evidence for this, as significantly less beta power in the HI group was observed in the SMA across the three conditions, compared to the LI group, see Figures 6.7, 6.11, 6.15 and Table 7.6. On the other hand, significant positive clusters were found in left frontal, temporal, parietal and occipital areas in the HI compared to the LI group. These results suggest an increased sensitivity towards reward-related cues, as reflected by the increased beta band power, while also preparing to execute a motor response, as reflected by the decreased beta band power found in SMA for the HI group compared to the LI group. Considering that the HI group showed a similar behavioural choice on the DDT compared to the LI group, it is possible that a potential compensatory strategy in high impulsivity individuals, might have recruited more the executive networks to show a less impulsive discounting rate, as reflected by stronger Delay-M3 amplitudes too.

Overall, findings suggest that the HI group might show an increased sensitivity towards reward, compared to the LI group, as reflected by higher delta and theta power. At source-level, the HI group only showed significantly more alpha suppression during the future reward Magnitude conditions, reflecting a possible attentional processing deficit towards the Immediate and future reward Delay cues, compared to the LI group. It could be argued that the future reward Magnitude cue, specifically, produced an increase in attentional processing in high impulsivity individuals. Low impulsivity individuals showed less beta power than those scoring high in impulsivity, which has been associated with motor control (Neuper et al., 2006).

In the HI compared to the LI group, only beta band power was supressed across the three conditions, suggesting an increased sensitivity towards reward-related cues.

Chapter	Findings	
6	TFRs	• Power increase in delta and theta bands across the three conditions in the HI compared to the LI group. • Source-level analysis of delta and theta band showed significant positive clusters in the HI group in frontal, temporal and parietal areas, compared to the LI group. • In the immediate condition only, increased theta band power was observed bilaterally in frontal regions in source-space in the HI group. In the future reward Magnitude and Delay conditions, the differences were located predominantly in the left hemisphere. • More delta power, increased sensitivity towards reward, and more theta power, increased reward processing, as observed in the HI group. • Power increase in alpha and beta bands across the three conditions in the HI compared to the LI group. • Additional enhanced alpha suppression in the HI group during the future reward Magnitude condition in right frontal, temporal and parietal regions, only this condition increased their attentional processing, compared to the LI group. • Power decrease in beta band in the HI group across the three conditions in right frontal, temporal and parietal regions, compared to the LI group. • Increased sensitivity towards reward-related cues, as reflected by the increased beta band power, while also preparing to execute a motor response, as reflected by the decreased beta band power found in the HI group, compared to the LI group.

Table 7.6. Principal findings related to Chapter 6, TFRs.

7.3. Limitations and future directions

For the behavioural experiment, certain limitations were observed. Firstly, while the high reward-delay impulsivity group made significantly more commission errors on the GNGT, I expected the SST would show similar results, as both tasks are thought to measure rapid-response impulsivity. The lack of significant results on the SST in this specific comparison might be explained by the absence of a staircase procedure. Such design would have adapted the time between the "go" and "stop" stimuli based on whether the previous trial was successfully inhibited or not, increasing the difficulty of the task. This methodological issue was considered for the MEG response inhibition experiment (Chapter 5) and was improved by implementing a staircase procedure on the SST.

Secondly, it should be noted that findings from the behavioural experiment should be taken with caution, as the number of individuals in the group which combined the high rapid-response impulsivity individuals and the high reward-sensitivity individuals was quite low. Only 13.8% of participants scored high on both dimensions of impulsivity, which limits the generalisability of our data. However, Wilbertz et al. (2014) who used the BIS-11 to identify high or low impulsivity individuals, reported selecting 52 from 452 participants which is a similar percentage (~11.5%).

Thirdly, participants here were recruited entirely from a student population and findings may thus not be generalisable to other populations (Hanel & Vione, 2016). Further limitations that should be considered are limited screening for psychopathology, as no screening for neurological conditions was taken at the time of testing. Any of these factors could affect the presented results.

Regarding the limitations found in the factor analysis, Chapter 4, as in similar studies that include both behavioural tasks and self-reports, the different assessments needed for each of these measures can be problematic. Even though all necessary and recommended transformations were made, behavioural tasks assessing prepotent response inhibition, behavioural choices on the delay discounting measure and psychometric scores on the self-report measures, were completely different to each other. Another limitation of the analysis presented in Chapter 4, was the fact that I could not statistically compare the non-nested models used in this study. Accordingly, the models were compared in a descriptive manner, taking into consideration the theoretical consistency of each model. Standard fit indices were used to compare which model might fit best. However, using such approach requires that results be interpreted with caution.

Regarding the MEG response inhibition experiment, although implementing a staircase procedure improved the stop-signal task design compared to Chapter 3, not all participants

achieved a 50% ratio of successful and unsuccessful number of trials as expected. Furthermore, considering that the combined Go/No-Go/Stop-Signal Task used here had the same conditions and parameters to that from Boehler et al. (2009), who did find this ratio in a healthy adult sample, it is a surprising result. This suggests there is still a necessity for improvement of this combined response inhibition task design.

Another relevant methodological limitation observed was the high number of females tested in the MEG study, which included the response inhibition and delay discounting experiments. Among the thirty-four participants, twenty-eight were females and six were males, which is not ideal. This was due to Psychology being the most common course undertaken by participants, in which a higher number of female students are enrolled, compared to students from other courses also examined here, see section 2.3 for details. As with the behavioural experiment, participants here were recruited entirely from a student population and findings may thus not be generalisable to other populations (Hanel & Vione, 2016). Also, the limited screening for psychopathology and neurological conditions taken at the time of testing, could affect the presented results.

Future directions

Results from the experiments presented here showed that the combination of two dimensions of impulsivity was able to differentiate individuals who showed high and low levels of trait impulsivity, as measured by self-report measures. This differentiation of impulsivity groups was observed in both the behavioural and neuroimaging experiments using the same task conditions. Considering that impulsivity has been reported to characterise several mental disorders, it is feasible to investigate impulsivity in the context of such disorders, and to consider its potential to predict risk for psychopathology. It could be argued that reward-delay impulsivity and response inhibition represent shared vulnerabilities for mental health conditions characterized by deficits in impulse control, e.g., bipolar disorder, substance use disorders and addictions (e.g., Bari & Robbins, 2013). Furthermore, both, Diagnostic and Statistical Manual of Mental Disorders (DSM) and International Classification of Diseases (ICD), recognise impulsivity as a diagnostic criterion for these disorders. Specifically, those aspects of impulsivity which can be considered unplanned, on-the-spur-of-the moment reaction with no regard for consequences, sense of urgency and self-harming behaviours, especially during times of emotional instability (Moeller et al., 2001). Therefore, it is important to investigate these two dimensions in conjunction.

Even though the MEG experiments presented here addressed some of the caveats found in the literature and provided novel results, see sections 5.5 and 6.5, there are certain questions that need further examination. For example, given the importance of the monitoring of

successful inhibition, as reflected by the NOGO-P3 component, high impulsivity individuals were expected to show differences in amplitudes, which were not observed. Interestingly, I did find differences between groups in the M3 amplitudes during the STOP condition, which indicates further examination is required on what the M3 component represents during response inhibition tasks.

Another unresolved question is the one concerning the potential compensatory strategy in HI individuals, a demand for increased inhibitory effort, as reflected by their stronger STOP-M3 component, this strategy has been proposed before (Dimoska & Johnstone, 2007; Lansbergen et al., 2007) and seems to be consistent with current results. However, it has been suggested that this compensatory strategy might only work in the auditory modality of the Stop-Signal Task (as in Dimoska & Johnstone, 2007; Lansbergen et al., 2007), but not in the visual modality (as in Shen et al., 2014). Here, individuals scoring high on impulsivity showed significantly reduced task performance in STOP and NOGO trials than those scoring low, suggesting the potential compensatory mechanism might not have worked well enough to overcome their rapid-response impulsivity, in line with Shen et al. (2014). it is intriguing that although task performance was also significantly reduced during NOGO trials, no differences were observed around the NOGO-M3 component between groups.

Nonetheless, the similarities across the NOGO and STOP conditions, both in the analysis of event-related fields and of oscillatory activity conducted here, indicate that the two conditions might be assessing a common feature of response inhibition. Yet, the differences observed between them need to be clarified. During the STOP condition specifically, high impulsivity individuals showed that the network involved in the inhibition process, as reflected by the M2 component, was engaged later, resulting in reduced performance than in low impulsivity individuals. This observation was not found during the NOGO condition, suggesting a difference in the assessment of response inhibition between the two tasks. Also, the significantly larger STOP-M3 amplitude observed in the HI group compared to the LI group, which was not found in the NOGO condition, could also suggest that the NOGO condition might not be sensitive enough to pick up differences on the M3 amplitudes between impulsivity groups. Altogether, the STOP condition proved to assess response inhibition differently compared to the NOGO condition, by showing additional differences between groups. Future studies should also consider the advantages provided by the Stop-Signal task when investigating response inhibition.

Although high impulsivity participants engaged significantly more frontal networks during the STOP-M3 component than low impulsivity participants, they still displayed reduced task performance. Impaired response inhibition, as measured by reduced task performance on

response inhibition tasks, has been related to aggravating conditions in bipolar spectrum disorders (Bari & Robbins, 2013), such as suicide attempts (Swann et al., 2005), criminal offences (Swann et al., 2011) and substance addiction (Swann et al., 2004). Rubio et al. (2008) reported that in adults, poor performance in response inhibition tasks was an indicator of consumption maintenance and of risk for alcoholic dependence. These results suggest an association between rapid-response impulsivity and course of illness (Lijffijt et al., 2014). Altogether, it would suggest that rapid-response impulsivity may serve as a marker of risk for developing a mental disorder (e.g., Aron & Poldrack, 2005; Hajek et al., 2013; Hidiroglu et al., 2015; Nigg et al., 2006; Rubio et al., 2008; Swann et al., 2004; 2005; 2009; 2011). Further studies are required to test this hypothesis by conducting follow-up studies of healthy individuals scoring high on self-report measures of impulsivity and showing impaired response inhibition.

During both response inhibition conditions, significant decreases of delta, theta and alpha bands in high impulsivity individuals were expected and found in left frontal regions, suggesting a possible deficit in frontal pathways involved in the suppression of a motor response and an attentional processing deficit in individuals scoring high. Although sensor-level analysis showed beta power to be also decreased frontally in the HI group, source-level analysis showed predominantly significant positive clusters in high impulsivity individuals, suggesting beta might be involved in response inhibition in a different way to delta, theta and alpha bands. Further research is required to investigate the differences between impulsivity groups in beta band activity during response inhibition tasks.

Regarding the reward-delay impulsivity MEG experiment, current results also raised certain questions that need further examination. For example, the amplitudes of the M3 component were significantly reduced in the HI compared to the LI group in the Immediate and future reward Magnitude conditions, suggesting less efficient reward processing. However, the M3 amplitudes in the HI group were significantly larger during the future reward Delay condition in frontal, temporal and occipital regions, compared to the LI group, which did not meet our expectations. Considering that the HI group did not show a significant behavioural choice difference on the DDT compared to the LI group, it is possible that this group had to recruit more cognitive resources to show a similar temporal discounting rate, reflected by the stronger M3 component. Future research should investigate this unexpected result, by examining further how the preference for immediacy, as reflected by the stronger frontal Immediate-M2, seen in the HI group is related to the larger M3 component seen in the future reward Delay condition only.

Regarding the oscillatory activity generated by this task, individuals scoring high on self-report measures of impulsivity showed significantly more delta, theta, alpha and beta band power across the three conditions, compared to those scoring low. However, power in alpha in the Magnitude condition and beta band across the three conditions, was also found to be significantly decreased in the HI group compared to the LI group. This was not expected, as previous studies have shown that the suppression of alpha band power is considered to index attentional processing (Mazaheri et al., 2014), whereas the suppression of beta reflects motor control (Neuper et al., 2006). Only the future reward Magnitude cue increased their attentional processing, as reflected by significantly more alpha suppression in high impulsivity individuals in right frontal, temporal and parietal regions than in low impulsivity individuals.

It is unclear whether the magnitude of a future reward, as reflected by increased beta power, or the increased sensitivity towards reward in the HI group, as reflected by more delta and theta power in frontal regions, is the crucial factor that impacts their decision the most. It is also plausible that the combination of both increased sensitivity towards reward-related cues and the increased attentional processing of the future reward Magnitude condition, characterises high impulsivity individuals. Nevertheless, current findings showed that the Immediate and future reward Magnitude cues were processed differently in high impulsivity compared to low impulsivity participants. Future research is required to investigate further what occurs specifically in impulsive individuals during daily-life choices, in which they show an impulsive preference for immediacy even if they are aware of the negative consequences such decision might cause, see section 1.2.3 for details.

It could be argued that future neuroimaging studies investigating impulsivity need to consider both dimensions in order to capture the most of this construct. This includes clinical and non-clinical studies which have investigated response inhibition tasks in populations characterised by trait impulsivity (e.g., (Benvenuti et al., 2015; Kamarajan et al., 2015a; Russo et al., 2008), and have generally used measures of rapid-response impulsivity only, such as the BIS-11, to assess impulsivity.

Finally, given the Behavioural and psychometrical base of measures of rapid response and reward-delay impulsivity, they might be used in future to aid in designing a screening tool to assess risk for psychopathology based on these two impulsivity dimensions. Such a screening tool could also benefit from the clarification of personality characteristics provided here, on each dimension of impulsivity. The acknowledgment of the specific aspects of impulsivity could also be useful when providing individuals high in impulsivity with strategies to recognize impulsive Behaviours or thoughts, or with tools to reduce their impulsivity in certain situations,

as in mindfulness-based strategies to reduce impulsivity (e.g., Stratton, 2006; Lattimore et al., 2011).

7.4. Conclusions

The aim of this thesis was to conduct a comprehensive investigation of the multi-faceted construct of impulsivity, which included the rapid-response and reward-delay impulsivity dimensions. This was first accomplished by conducting a behavioural study that compared different measures of impulsivity, the sensitivity of these measures to differences between low and high impulsivity groups was examined, based on the two impulsivity dimensions. Results showed that the proposed measures were sensitive to differences between groups. Participants with higher impulsivity, as measured by high rapid-response impulsivity scores on the BIS-11 Motor subscale, showed significantly increased trait impulsivity as well as Behavioural and self-reported reward-delay impulsivity. Conversely, the high reward-delay impulsivity group had significantly higher trait impulsivity and Behavioural and self-reported rapid-response impulsivity. When both dimensions of impulsivity were combined, the high-impulsivity group showed significantly reduced task performance on both response inhibition paradigms (GNG and SST) and temporally discounted in a significantly more impulsive manner in the reward-delay task than the low-risk group. These findings provide evidence that combining impulsivity dimensions provided a better predictor of impulsivity level than each dimension alone.

Considering the relationship between the two dimensions of impulsivity observed in the behavioural experiment, the next analyses conducted tested the hypothesis that a three-factor model of impulsivity, consisting of impulsive action, impulsive choice and impulsive personality traits, would benefit from the inclusion of a psychometric measure of reward-delay impulsivity. This hypothesis was partly supported. Results favoured a three-factor model but including a reward sensitivity psychometric measure did not improve the model fit. Furthermore, the model structure reported here was different to that proposed in previous studies (e.g., MacKillop et al., 2016). These findings strengthened what researchers have emphasised before, namely that rapid-response impulsivity and reward-delay impulsivity are two major yet different dimensions which contribute independently to the multifaceted nature of the impulsivity construct.

Altogether, the two analyses showed that both dimensions of impulsivity need to be considered and that the cognitive measures tested here were sensitive enough to pick up behavioural differences between individuals scoring high and low on impulsivity. The next step involved investigating the behavioural differences found in Chapter 3 further, by examining

potential differences in the neural correlates of both response inhibition and delay discounting between the two groups using MEG. The aim of the two MEG experiments was to examine these potential differences while also trying to clarify some of the inconsistencies found in EEG and MEG studies.

Most hypotheses described in section 1.4 were supported by current findings. Overall, these indicate that impulsive individuals, as measured by high scores on rapid-response and reward-delay impulsivity self-reports, showed significant differences in the neural correlates of response inhibition and temporal discounting compared to those scoring low.

Firstly, results suggest high impulsivity individuals showed an attentional processing deficit, as indicated by significantly smaller M1 components across all conditions in the right parietal cortex and less alpha suppression in the two tasks in posterior regions. However, it should be noted that decreased alpha power was also observed in the high impulsivity group, which indicates that decreased anterior and increased posterior alpha activity during response inhibition and delay discounting tasks might characterise high impulsivity individuals.

Regarding response inhibition specifically, and consistent with previous studies, current results suggest that the NOGO-M2 and STOP-M2 components might serve as a measure of response inhibition efficiency, while reduced efficiency was observed in individuals scoring high on impulsivity. The analyses of oscillatory activity during both NOGO and STOP conditions also showed a possible deficit in frontal pathways involved in the suppression of a motor response in individuals scoring high on impulsivity, as indicated by decreased delta and theta band power. Furthermore, analyses of the late positive component commonly found in response inhibition tasks, suggest the high impulsivity group engaged frontal networks significantly more than the low impulsivity group during the STOP condition only, possibly as a compensatory strategy. However, this strategy might not be efficient enough to prevent the reduced task performance observed in these individuals during the Stop-Signal task. These results indicate that the Stop-Signal task might be more precise when assessing response inhibition, as additional differences were found during the STOP condition.

Regarding delay discounting, along with the mentioned attentional deficit, increased preference for immediacy was observed in high impulsivity individuals, as reflected by larger Immediate-M2 amplitudes compared to low impulsivity groups. Impulsive participants also showed less efficient reward processing of the Immediate and future reward Magnitude cues. Considering that the HI group did not show a significant difference in behavioural choice compared to the LI, it is possible that the HI group had to recruit more cognitive resources to show a similar temporal discounting rate, which might be reflected by the stronger Delay-M3 component found in the HI. Analyses of oscillatory activity during temporal discounting

revealed that that high impulsivity individuals might show an increased sensitivity towards reward, compared to low impulsivity individuals, as reflected by higher delta and theta band power in frontal regions. Increased alpha power in the HI group compared to the LI group in all conditions, suggested reduced attentional processing (alpha suppression), but also revealed an additional reversed effect (stronger alpha suppression) in left fronto-temporo-parietal areas, but only in the Magnitude condition. Only beta band power was found in the HI group to be also supressed across the three conditions, suggesting an increased sensitivity towards reward-related cues, compared to the LI group.

Altogether, the experiments described in this thesis illustrated how personality traits such as impulsivity relate to differences in the behavioural and neural correlates of cognitive processes. Given that as explained before, rapid-response impulsivity has been suggested to serve as a marker for developing a mental disorder, current findings highlight the necessity for further investigation, as these show healthy young adults scoring high on impulsivity to show impaired response inhibition at behavioural and neural level. Furthermore, the differences in the neural correlates of temporal discounting, added to the finding that the combination of impulsivity dimensions provided a better predictor of impulsivity level than each dimension alone, indicate that both need to be considered in future research.

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