An Analysis of Some Control Strategies for Environmental Lead

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SUMMARY

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This study is designed to examine the cost and effectiveness of controlling automotive lead emissions. Such emissions, accounting for 90% of all airborne lead, result from the combustion of gasoline containing lead-alkyl antiknock agents. These lead compounds are not naturally present in petroleum and so it would be possible to eliminate the major source of atmospheric lead pollution by discontinuing their use. Hence this policy study determines the cost-effective ranking of a number of suggested control strategies. This work complements the broad range of clinical and epidemiological studies carried out into the effects of environmental lead.

The phenomenon of engine knock, and its prevention, is described to illustrate the role of lead in gasoline and the impact of its reduction or elimination. This is followed by a case study documenting the history of the elimination of lead from gasoline in the U.S.A., outlining the arguments for and against the control of lead emissions from automobiles. Having thus described the problem area, a number of control strategies for the U.K. are suggested and the concept of Cost-Effective Analysis as a means for their comparison introduced, along with the use of modelling to evaluate the strategies' effects. Existing models of human lead transport are reviewed and a model relating to a standard man developed. This standard man approach is used to calculate the effectiveness of the proposed strategies and their impacts on the Refinery-Engine System are assessed. Having calculated the costs of the various strategies, this information is combined with their effects to produce a cost-effective index. In conclusion, the concept of Cost-Effective Domination is introduced and employed to ascertain the dominant options, these results being presented as an input to the policy-maker.

Key Words: Strategy, Policy, Gasoline, Lead, Model.

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List of Contents

List of Contents

List of Figures

List of Figures

List of Tables

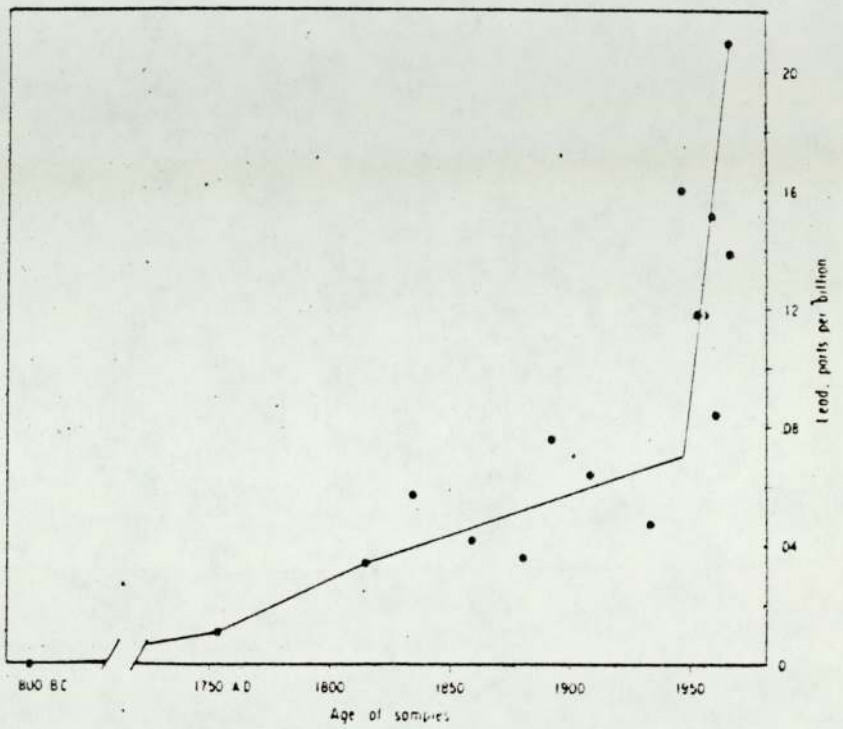
List of Tables

SECTION 1

Introduction

This thesis, "An Analysis of Some Control Strategies for Environmental Lead" may be introduced by explaining and expanding upon certain aspects of its title. The word pollution may be added to the end of the above title without changing its context in any way. The environmental lead under discussion is there as a result of pollution, the addition by man to the environment of an undesirable substance, in this instance lead. It should be noted, however, that lead occurs in the environment not only as a result of man's activities but also due to natural events such as volcanic activity and gases seeping through the earth's crust. Natural levels of lead concentration have been estimated to be of the order of 0.0005 ug/m^3[1] and result from airborne dust containing 10-15 ppm of lead[2]. However, the vast majority of current quantities of lead found in the environment are there primarily as the direct result of man's activities. Analyses of snow strata from polar areas have shown that lead concentrations grew from 0.0005 ug/kg in 800 BC to more than 0.2 ug/kg in 1965. A major rise occurred circa 1750, the start of the industrial revolution and then during the second half of the eighteenth century they tripled. From 1935 to 1965 they abruptly tripled again, the sharpest rise occuring after 1940, the Second World War. Current lead levels in Greenland snow are estimated to be about 400 times the original natural levels. Although these data document the dramatic rise of lead in some environmental components, notably the atmosphere, they are not meant to be interpreted as representative of world-wide conditions. Lead pollution in

the southern hemisphere, as measured via Antartic snow studies, is much lower, rising to 0.02 ug/kg only after 1940[3]. This variation is possibly explained by a lower degree of industrialisation in the southern hemisphere coupled with barriers to north-south tropospheric mixing via winds etc. Figure 1-1[4] shows the rise in environmental lead concentrations as measured from the artic snow strata. This increase was almost wholly due to man-made uses of lead and Table 1-1 overleaf shows the lead usage, for the United States, in 1968. The product spread shown in this table would be similar to that for Europe, although for the U.K. the quantities involved would be much smaller. As a result of man's usage of lead, quantities of the element escape into the environment where they become a pollutant. Lead is toxic to man in quantity and serves no known biological function. Therefore it would be reasonable to take whatever steps possible to reduce man's exposure to environmental lead. Lead has been detected in all times in man and animals in all geographic areas, including those remote from man-made sources of lead exposure. The primary sources for human lead input are food, water and other beverages, and atomospheric lead picked up via the gut and lungs respectively. From these two areas lead is transferred to the blood and thence to a variety of body sites, or is excreted. Lead tends to build up within the body over time and is stored primarily in the skeleton where it supplants calcium. Hence, to reduce human exposure to lead it is necessary to reduce the concentration of lead in the various sources of exposure, or to place a barrier to lead absorbtion between the source and humans. From the nature of the sources



Growth of Lead in the Environment

Figure 1-1

From NAS Study, 1972[4]

Table 1-1

Lead Consumption in the U.S.A., 1968

Product		Lead Consumption Tons
Metal Products	Subtotal	915,500
Ammunition		82,193
Bearing Metals		18,441
Brass and Bronze		21,021
Cable Covering		53,456
Caulking Lead		49,718
Casting Metals		8,693
Collapible Tubes		9,310
Foil		6,114
Pipes Etc		21,098
Sheet Lead		28,271
Solder		74,074
Storage Batteries:		
Battery grids		250,129
Battery Oxides		263,574
Terne Metal		1,427
Type Metal		27,981
Pigments	Subtotal	109,734
White Lead		5,857
Red Lead and Litharge		86,480
Pigment Colours		14,163
Other		3,234

Table 1-1 cont'd

Chemicals	Subtotal	262,526
Gasoline antiknock agents		261,897
Other		629
Miscellaneous Uses	Subtotal	23,106
Annealing		4,194
Galvanising		1,755
Lead Plating		389
Weight and Ballast		16,768
Unclassified Uses		17,924
	Total	1,328,790

mentioned above it is apparent that this latter course of action is somewhat impractical; e.g. issuing the population with breathing apparatus to prevent pick-up of airborne lead. However, the magnitude of the lead pollution question is such that it would not be within the scope of a single project to examine all the possibilities for its control. For this reason two projects were conducted within the Technology Policy Unit of Aston University on the above topic. The subject was broken up according to the two primary exposure routes, the lungs and the gut. Edwards, 1979[5] studied the control of exposure from dietary sources via the gut whilst this study is concerned with the reduction of man's exposure to lead from the atmosphere. With respect to this, it may be considered that there is a single source for airborne lead, the combustion of gasoline containg lead alkyl antiknock agents. This results in the emission of inorganic lead compounds from the exhaust systems of vehicles using leaded gasoline and these emissions account for in excess of 90% of airborne lead[6]. As a result of this fact, this study concerns itself with the control of environmental lead occuring as a result of the use of lead antiknock agents in gasoline.

Before continuing to describe the project in detail, it would be useful to outline the reasons why it is considered that environmental lead should be a cause for concern. The toxicity of lead has been known for centuries and its widespread use in industry has led to a considerable body of knowledge concerning the effects of exposure to high (industrial) concentrations of the metal. Having carried out

research on lead industry workers Kehoe suggested that exposure to the metal is harmful only when a blood lead concentration of 80 ug Pb/100 ml blood is exceeded[7]. However, environmental exposure to lead at current concentrations would never be expected to produce a blood lead of this magnitude. Kehoe's investigations were concerned with clinical lead poisoning - poisoning producing overt signs or symptoms which are evident to the doctor and sufferer. For adults such symptoms may include abdominal pain, constipation, vomiting, general debility and diarrhoea. Cases of industrial lead poisoning have been steadily decreasing in number due to on-going improvements in industrial hygiene but researchers have expressed concern regarding a different problem posed by environmental lead. Evidence is accumulating from a number of areas that exposure to low levels of lead, such as those found in the environment, may produce harm long before the 80 ug Pb/100 ml limit of Kehoe is reached. However, the evidence for this sub-clinical lead poisoning is not indisputable and it may be that conclusive proof for these effects will never be found[8,9]. A major risk group which it is said may suffer harm due to sub-clinical effects of lead exposure at environmental levels is young children. A number of studies[10,11] have suggested that children may suffer behavioural, mental and developmental disturbances as a result of lead exposure; e.g. impairment of fine motor, perceptual and visual skills, lower age-adjusted I.Q.. Lead has also been implicated as a causative factor in hyper-activity in young children[12], hyper-active children showing higher blood lead concentrations

and higher lead loss after chelation therapy than non-hyper active controls. A study[13] on young adults being treated for behaviour disturbances and learning difficulties showed a significant correlation between increased systolic blood pressure, decreased hand-eye coordination, shortened reaction times and higher blood lead concentration. However, all the measured blood lead levels were in the normal range for U.S. males and no subject was suspected of ever having suffered undue lead exposure. Lead, it has also been suggested, plays a role in motor neurone disease[14], multiple sclerosis[15,16] and, in the U.K., the correlation between cardio-vascular disease and soft water has also been intensively studied - soft water dissolves lead from piping more readily than hard[17,18]. Animal studies based on lead in water concentrations typical of those found in Glasgow showed biochemical and morphological changes to the heart muscle[19]. Further health damage allegedly related to lead exposure is the high infant mortality rate found in soft water areas[20] and renal insufficiency found in people living in houses equipped with lead piping[21].

However, it must be emphasized at this point that the evidence relating exposure to environmental lead to the various aspects of health damage mentioned above cannot be interpreted as conclusive by any means, and in most cases may barely be regarded as circumstantial. With regard to lead exposure relating to emissions from automobile exhausts, there has never been a single case of recorded childhood illness attributed to this source, a point cited by the manufacturers

of lead alkyl antiknock compounds[22].

The issue is further complicated by the narrowness of the safety margin for lead exposure - the smallest for any substance widespread in the environment. Kehoe found that members of a rural Mexican population remote from any man-made source of lead exposure exhibited blood lead concentrations of the order of 0-15 ug Pb/100ml. These figures are not far below the typical values for urban U.K. dwellers who exhibit blood leads ranging from 10-25 ug Pb/100 ml. Since the Mexican's blood leads represented natural contact with the metal, Kehoe suggested that they must be safe, and that urban lead levels posed no threat to the populations health[23]. Patterson[24] argued, from a different standpoint, that natural blood lead concentrations ought to be as low as 0.2 ug Pb/100 ml, suggesting that current environmental lead levels may be causing severe and widespread damage to the population. The ensuing debate between Kehoe and Patterson illustrated the point that the real issue to be considered is whether the safety margin between environmental exposure to lead and exposure levels which cause harmful effects is sufficient? This disregards what natural lead levels in humans might be and the paucity of irrefutable evidence for the various harmful effects described earlier. Reviewing the existing information regarding whether or not current environmental lead concentrations are, or are not, harmful leads inevitably to the conclusion that there is insufficient, unequivocal evidence to answer the question. However, if it is decided that current fears about the possible damage being caused by

lead pollution may be proven correct at some time in the future, it is obviously necessary to act now, in a state of almost complete ignorance, to reduce lead concentrations in the environment. If a decision is delayed until solid proof of lead's harmful effects is found the damage might well be irreversible, particularly in areas such as developmental and I.Q. damage to children. This raises the problem of how to make a rational decision when the state of ignorance of the decision-maker is so large. Not knowing the extent of the damage possible from environmental lead concentrations, the benefit obtained by reducing these levels cannot be calculated, although the costs of such reductions, particularly those related to airborne lead, may be estimated with a reasonable degree of accuracy. Therefore, it is impossible to balance the benefits against the costs of reducing lead levels in the environment. For this reason, a cost-effective methodology has been adopted in this study where the costs of a number of possible strategies will be related to their effectiveness in reducing the possible harm caused by lead exposure from the atmosphere, due to the fact that this project concerned itself solely with airborne lead pollution originating from automobile exhaust emissions. Thus if there should be a relatively cheap, i.e. highly cost-effective, means of reducing or eliminating the above lead exposure then, even though the cost involved cannot be compared with the benefits obtained, the method may still be adopted on an 'insurance policy' basis against the possibility of harm caused by environmental lead levels. If, however, no such

cheap methods exists, it may be decided that the cost of reducing or eliminating possible harm from lead is too great and the money involved might be better spent elsewhere, e.g. cancer research, and population accepts the possible dangers from the metal. It should be noted that it is not the objective of this project to provide an answer to this latter choice, but rather to obtain objective data upon which a policy-maker might base a solution to the problem. Although a full description of the cost-effective approach will be given in Section 4, it would be useful to examine, albeit briefly, the methodology for the measurement of the cost-effectiveness of strategies at this point. Given what is known about lead; the evidence for its possible sub-clinical effects and the narrowness of its safety margin, what should be done, if anything, to reduce the average person's exposure to the metal from the environment? The obvious difficulty is that knowledge regarding the behaviour of lead at these low exposure levels is far from complete. As previously mentioned, all the evidence cited at the beginning of this section may be reasonably contested at the present time. Hence, as stated above, a decision, if one is to be taken, will have to be made in a state of gross ignorance about the relevant facts. If it is decided to reduce lead levels in the atmosphere and such levels are in no way harmful, then a great deal of resources will have been wasted. On the other hand, if current fears are later proved correct, and no action is taken, considerable harm will be done to the population as a result of lead exposure. The classical approach to this

problem, Cost-Benefit Analysis, is ruled out because in no way is it possible to value the benefits accrued. This will be discussed further in Section 4. For this reason a Cost-Effective Approach seems required.

As discussed in Sections 4, 5 and 6, blood lead reductions were chosen as the index of the various strategies' effectiveness. If the aim of each strategy is to lower blood lead levels in the general population, then for each strategy the cost per unit reduction may be calculated; i.e. cost per 1 ug Pb/100 ml blood reduction in blood lead concentration. The cost-effectiveness of those strategies under consideration may then be compared, and a choice of strategy made. It follows from what was said earlier that, even for the most cost-effective option, it cannot be determined whether its benefits in reducing or eliminating harm caused by lead exposure actually outweigh its costs. This latter point is an inevitable restriction of the cost-effective approach, but this approach would appear to be the only feasible one at present. The outcome of this methodology, should a highly cost-effective strategy be found, would be that the policy-maker accepts the costs of the strategy as the premium for insuring the population against the possible risks of environmental lead exposure. Of course it may be decided that the premium is too high in which case it is decided to accept the risks of harm from environmental lead and allocate the resources for its control elsewhere. Obviously should the potential risks from lead exposure become more definite, the acceptable premium becomes accordingly greater.

The methodology employed in this project follows five stages:

1) Identify feasible control strategies for lead. (N.B. In this instance lead refers to airborne lead resulting from the combustion of leaded gasoline).

2) Develop a model linking exposure to the various forms of lead through different channels to blood lead levels.

3) Use the model to calculate the effectiveness of those strategies under consideration.

4) Establish estimates for the cost of each strategy.

5) From 3 and 4 calculate the cost-effectiveness of those strategies under consideration.

As an aside it should be noted that this methodology may be applied to a wide range of pollutants where there is a paucity of knowledge regarding their harm at environmental concentrations. However, returning to the methodology's application to lead, there follows a brief examination of each point in turn. Firstly it is necessary to define what is meant by a strategy. A strategy is any way of lowering the population's exposure to lead. Strategies for handling the lead problem must be clearly distinguished from policies. A policy, in this context, is a political decision to deal with the

problem of environmental lead by implementing one, or possibly more, strategies for its control. Whether or not a policy decision is taken to employ a particular strategy is a normative question beyond scientific investigation. For obvious reasons this project was limited to discussion of strategies which, although it may be somewhat artificial - the very best strategy may be completely, politically unacceptable - seems an inevitable restriction. In considering possible strategies, it became apparent that two approaches were of particular importance; reducing or eliminating the use of lead in gasoline and preventing the escape of lead from vehicles' exhaust systems. Both of these will be discussed at length in Section 6.

Secondly, in order to assess the effectiveness of the various strategies, a model was developed linking lead exposure via the lungs and gut to blood lead concentration. Ideally the model would have related lead exposure to harm caused by such exposure but this was not possible as described in Sections 5 and 6. The model eventually related exposure to lead to the blood lead of a standard man, this latter factor being employed as a surrogate for potential harm to any member of the population. It is unfortunate that the model presently refers to the standard adult male as suspected risk groups at present include children and expectant females but, given current knowledge regarding the metabolism of lead by human beings, this is an unavoidable restriction. However, the use of the model permits the effectiveness of each strategy to be calculated in terms of the reduction in

blood lead concentration it brings about. Furthermore, the use of the model allows strategies employing different approaches and technologies to be directly compared one against another, as described in Sections 5, 6 and 8.

Thirdly, the calculation of the costs of strategies to control airborne lead is made more difficult by the many options available for this latter task. However, the various choices fall into two main categories; those which reduce or eliminate the lead antiknock compounds from gasoline, and hence require alteration of petroleum refineries, and those which reduce or eliminate lead emissions from vehicles, and hence require modification of the vehicles' exhaust systems. Various options from both of these categories may be combined to give further control strategies as discussed in Section 6. With regard to the estimation of the costs of these strategies one major criticism which may be levelled is that most of the available data comes from the two most interested parties, the lead antiknock manufacturers and the petroleum refiners, or their representative bodies, such as CONCAWE which represents the major European refiners. Nevertheless if a monetary valuation of the costs of the various strategies is deemed necessary, then data from the above sources has to be utilised. The strategies considered in this project, six in all, are not an exhaustive list of the options available for the control of lead emissions from motor vehicles. In fact these strategies represent the most viable variations of the two main approaches to such control; firstly elimination of the lead additives from gasoline and secondly the prevention

of the escape of lead compounds to the atmosphere. These were chosen because reviewing the literature suggested that they were the most likely options to be considered seriously in the near future. Alternative approaches such as the use of manganese compounds as antiknock additives or adding alchohol to gasoline were examined briefly but discounted as impractical solutions to the environmental lead problem presented by automotive lead emissions. Manganese can replace at most two of the six research octane numbers lost with the elimination of lead additives, saving at most 1% of the additional crude oil required for lead free fuel[25], its economic attractiveness being greatest at low concentrations. With regard to the use of alchohol as a fuel additive a U.K. Working Party stated, "There was no proven technical case for introducing a blend of petrol containing methanol and higher homologues as a motor spirit"[26].

The costs for each strategy were calculated for the U.K. although some of the cost data applied to the EEC as a whole. Translation to U.K. values is possible, however, because refinery technology is fairly constant across Europe. Indeed many major refiners operate on a Europe-wide basis balancing crude oil supply and product output throughout the EEC. Costs are calculated as conservative values and the simplest implementation of each strategy is considered; e.g. all existing automobiles are fitted with lead particulate traps within twelve months, as opposed to some form of phased introduction. This will be described fully in Sections 6 and 7.

Having calculated the costs of each strategy and their effectiveness, obtaining the cost-effectiveness is elementary. In the present state of uncertainty about lead, the ranking of possible control strategies by their cost-effectiveness inevitably involves many assumptions. However, these assumptions may be tested in the light of further research as to whether they are critical to the cost-effective ranking. This aspect of the methodology is best illustrated by Figure 1-2. The feedback loops, indicated by the dotted lines, show how the cost-effective ranking may undergo iterative improvement. The cost-effective ranking obtained by this method obviously may not be conclusive by itself. A variety of other factors, including radically different approaches to the problem's solution will have to be considered by the policy-maker; e.g. the possibility of altering vehicle and/or engine design. These factors will be outlined more fully in Section 8 when possible alterations to the cost-effective ranking will be considered, particularly those which might be brought about by factors external to this study.

As an introduction to the project and to illustrate the arguments involved with the elimination, or reduction, of airborne lead pollution, a case study will be described in Section 3. This will chart the history of lead control policy in the United States concerning lead emissions from automobiles, and it should be noted that this represents the only fully documented study of its type. Due to the nature of the U.S. legislative procedure an open and full discussion

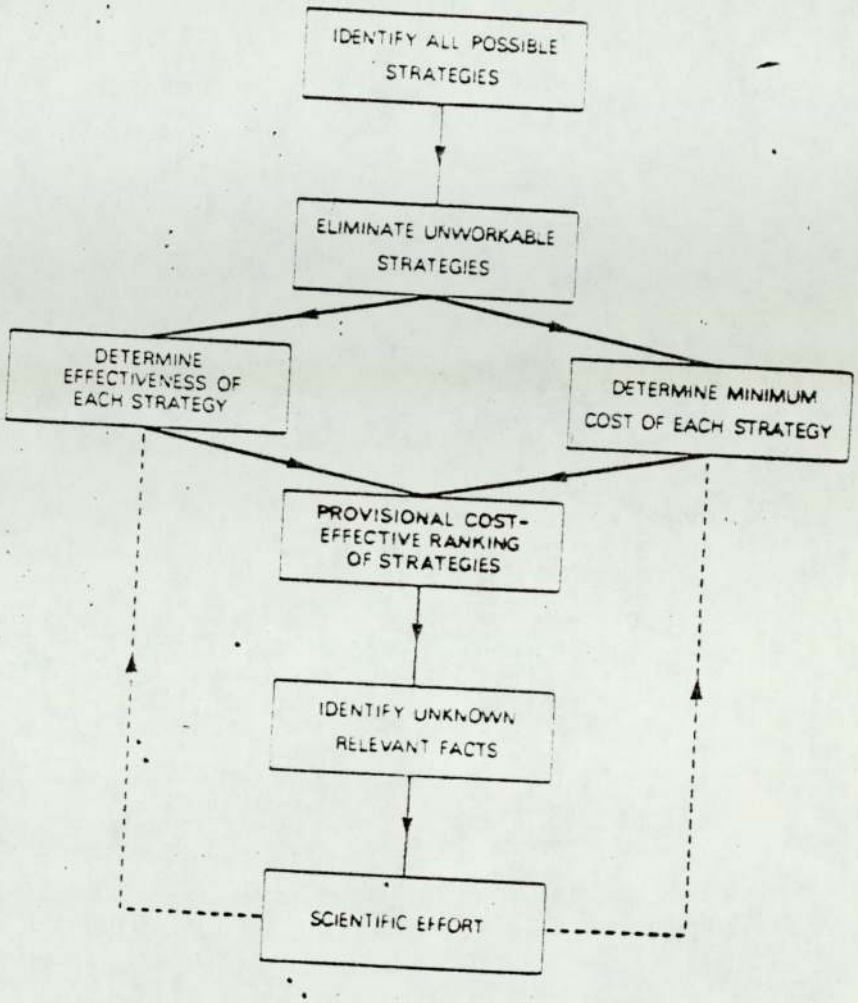

IDENTIFY ALL POSSIBLE STRATEGIES
ELIMINATE UNWORKABLE STRATEGIES
DETERMINE EFFECTIVENESS OF EACH STRATEGY
DETERMINE MINIMUM COST OF EACH STRATEGY
PROVISIONAL COST-EFFECTIVE RANKING OF STRATEGIES
IDENTIFY UNKNOWN RELEVANT FACTS
SCIENTIFIC EFFORT

Figure 1 - 2

of any legislation has to take place before the adoption of such legislation. In this case, the opponents of lead additives in gasoline, the U.S. Environmental Protection Agency, proposed rules to eliminate lead antiknocks from gasoline which were fiercely contested by the lead additive manufactures, the Ethyl Corporation of Detroit. Although there are major differences between the Refinery-Engine System which exists in the U.S. and that which exists in the U.K. and Europe, the U.S. case study provides a useful insight into the problems that would arise should a similar lead elimination policy be adopted in the U.K. Furthermore, the history of the elimination of lead from gasoline in the U.S. also serves to illustrate a major change in awareness regarding lead pollution; it was deemed necessary to consider the entire population to be at risk from environmental levels of lead exposure and studies to determine the accuracy of this supposition were implemented, whereas earlier work, such as that of Kehoe[27], had centered on lead industry workers. This latter group represents a specialised population, male, deliberately screened of those found to be sensitive to lead. Only recently has it been thought to question the application of health criteria developed for this group to the general population and the arguments documented in the EPA/Ethyl Corporation case in the U.S. illustrate this latter point in detail.

By adopting the role of a policy study it is hoped to complement the large volume of clinical research that has been carried out on the airborne aspect of environmental lead

pollution. Although these studies provide a useful insight into the clinical, and more recently sub-clinical, effects of exposure to lead, they do not provide the decision/policy-maker with information directed at solving the problem. Pressure groups, as exemplified by the Campaign Against Lead In Petrol (C.A.L.I.P.) demand action but again, for the most part, fail to provide the necessary information to facilitate the removal of lead additives from gasoline. It is readily apparent that simply "turning of the lead tap at the refinery" could well have catastrophic consequences for one of the country's major modes of transportation, and hence a policy study of this nature would appear to be a prerequesite of any action to control airborne environmental lead. Given a finite budget, it is necessary to attempt to assess the impact of any policy to reduce pollution before its implementation. It may be that other demands on the same budget may take priority for financial, technical or political reasons; e.g. inner-city renovation. As mentioned previously, this latter point is a normative question quite beyond scientific investigation. The conclusions of this study can only provide the policy-maker with the basic information upon which to base a rational decision.

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SECTION 2

The Purpose of Lead in Gasoline

Originally lead alkyl compounds were added to gasoline to improve the octane quality of the fuel and hence eliminate the phenomenon of 'knock'. As the price of crude oil has risen, particularly after the action of the OPEC nations in 1973, lead additives have also played an economic role as they represent the cheapest route to the high octane (99 Research Octane Number, RON) gasoline required by the majority of European cars.

Before continuing, it would be helpful to outline what is meant by 'knock'. When the gasoline - air mixture enters the engine cylinder, it is compressed by the piston and ignited by the sparking plug. During the normal combustion process, the fuel charge is consumed smoothly by the advancing flame front. However, under certain conditions the charge furthest away from the sparking plug, referred to as the 'end gas' becomes overheated by radiation from the advancing flame front, and by compression by the expanding gas behind the flame front, and prematurely detonates. The shock waves from these detonations produce an audible, metallic ringing sound commonly known as 'pinking'. These detonations produce mechanical and heat stresses within the engine which reduce efficiency, and in extreme cases may result in premature engine failure. This phenomenan is also known as low-speed knock and is usually experienced whilst the engine is running slightly faster than 'tick-over' , e.g. when the vehicle pulls away from rest. Knock is also experienced, however, at higher engine speeds, for a different reason and is known in this case as 'high-speed knock'. This may result from deposits within the engine cylinder reducing the thermal conductivity

of the engine block and producing a hot-spot on the cylinder or piston. At low running speeds this has no effect but under high speed-load conditions, e.g. climbing a motorway gradient at speed, the temperature rises sufficiently to permit the hot-spot to cause premature detonation as described previously. However, the characteristic ringing noise cannot be heard in this instance due to the increased noise levels associated with high speed running. Thus the driver is unaware that knocking is taking place until damage actually occurs, such as the hot-spot burning a hole through the piston. Since the 'pinking' cannot be heard, the short-term solution as used for low-speed knock, i.e. closing the throttle, cannot be applied. In both cases the only effective solution is to have the engine correctly tuned and employ a suitable grade of gasoline, taking into account the engine's compression ratio, to prevent knock. Combustion of the fuel without detonation is necessary to obtain the engine's maximum efficiency and this can be achieved by adding small quantities of lead anti-knock agents to the fuel.

Historically the phenomenon of knock has been studied since the introduction of the spark ignition internal combustion engine. In England the early work was carried out by Hopkinson and Ricardo, whilst in the U.S.A. similar research was being conducted by Midgley and Kettering[1]. This work led to the discovery of a relationship between knock and the molecular structure of the fuel components. Ring-shaped aromatics or branched-chain structures were found to be more resistant to knock than straight-chain paraffins. In 1916 Midgley found that knock could be suppressed in kerosene fuelled engines by

the addition of iodine to the fuel. From this Midgley concluded that iodine, being coloured, absorbed light energy and thus helped the kerosene resist knock. For three years he tried to find further dyes which would act as effective antiknock agents without success. Then, in 1919, he decided to try the addition of aromatics and found that aniline was an effective antiknock and he then abandoned the colour theory.

Continuing his research into possible antiknock materials, it was necessary to employ compounds soluble in the fuels of the day, namely gasoline and kerosene. Midgley worked in a systematic manner, following the behaviour of different elements according to their position in the Periodic Table. In this way he studied a number of organo-metallic compounds and altogether he discovered some 143 antiknock agents. The most outstanding of these was Tetra Ethyl lead (TEL), first tested in the 1920's, whose performance in terms of cost-effectiveness far exceeded that of all the other compounds that Midgley had discovered. It first became available for commercial use in 1923, and there shortly followed the discovery of Tetra Methyl lead (TML) which was found to be equally effective although more volatile. This latter compound, however, did not enter commercial use until about fifteen years ago.

As mentioned previously, high-compression engines require a high octane (RON) fuel if knocking is to be avoided. In studying the behaviour of fuels during combustion it became necessary to grade the fuels in some way. Iso-octane which has a very high resistance to knock was rated 100, while n-heptane which has a high susceptability to knock was rated

zero. Mixtures of these two hydrocarbons are used as reference fuels; the percentage of iso-octane in the fuel is termed the octane number (RON). Commercial fuels are rated by an octane number measured by comparison with these reference fuels. The higher the octane number the greater the resistance to knock.

Antiknock compounds are blended into commercial fuels in order to improve the fuels' octane quality and reduce the tendency for knocking to occur within the engine. The anti-knock agents present in these compounds are primarily Tetra Ethyl lead and Tetra Methyl lead, however, triethylmethyl lead, diethyldimethyl lead and ethyltrimethyl lead may also occur[1]. During combustion, lead oxides are formed which if allowed to build up would reduce the engine's performance. Thus antiknock compounds contain scavengers dibromoethane or a mixture of dibromoethane and dichloroethane, to convert, the lead oxides to lead halides which are volatile at engine operating temperatures and so are carried off with the engine exhaust gases.

Since the introduction of lead additives in the early twenties there has been an ever increasing demand for gasoline. This has resulted in large advances in refining technology, but despite these advances, because of the increasing scarcity and cost of crude oil, the most economic method of improving gasoline octane quality is by the addition of lead based antiknock compounds. This also permits increased flexibility in refinery operations to meet production demands.

TML was discovered shortly after TEL, but at that time, 1923, it showed no advantages over the latter compound. Furthermore, it was envisaged that its higher volatility would lead to handling problems. At this point it should be noted

that all organo-lead compounds are highly toxic and a small quantity will prove fatal whether inhaled, ingested or absorbed via the skin. Rigorous government safeguards are enforced at all stages of production and distribution[2]. However, currently TML and TEL are both used in improving the octane quality of gasoline. Some refinery processes produce gasoline components with a wide range of octane ratings which, when used in multi-cylinder engines, separate giving an uneven distribution of octane ratings in different cylinders. The higher volatility of TML compared with TEL may be an advantage under these conditions. TML is more easily distributed between the cylinderswith the result that knocking is effectively suppressed throughout the engine.

The current automotive internal combustion engine has evolved in close association with available fuel quality. At present lead additives are employed because they are the most convenient and economical method of increasing the octane quality of all grades of gasolines and in particular they are responsible for obtaining the final 6 to 8 octane numbers (RON) for premium gasoline, equivalent to 4-Star in the U.K. The availability of this high octane fuel has permitted the development of high-speed, high-compression engines which are used in most European vehicles. The thinking behind these fuel/engine developments is simplified if it is seen in thermodynamic terms. For a given engine capacity the efficiency that can be obtained from an internal combustion engine varies directly with the temperature gradient, mechanical considerations being considered as constant; the higher the engine operating temperature the more efficient it becomes. One means of

raising the operating temperature is to increase the engine's compression-ratio which increases the tendency for knocking to occur with low octane fuel. The obvious solution to this problem is to employ a high octane fuel. Current European engines have compression-ratios ranging from 9:1 to 11:1, requiring 99 RON fuel, (4-Star). The manufacturer's objective in fitting these power plants to their vehicles is to achieve satisfactory on-the-road performance from a light-weight engine with good fuel economy. Hence, improvements in performance, brought about by increases in engines' power outputs, tend to be obtained by raising the operating temperature, by raising the compression-ratio, rather than by alternate approaches such as increasing engine capacity. This route to improved performance is chosen on the grounds that it is the most cost-effective option available. Normal refinery operations produce premium gasolines with an octane quality of about 93-95 RON[3]. To obtain 99 RON fuel lead antiknocks are added effectively raising the fuels' octane quality by up to 6 octane numbers. Naturally 99 octane fuel can be refined directly but at an increased cost in terms of crude oil and energy consumption at the refinery; much greater than the cost of employing lead antiknock additives. This latter point provides the crux of the economic argument for the use of lead additives in gasoline.

It is readily apparent that the technical and economic reasons for the use of lead antiknock additives are closely interrelated. The performance of motor gasoline is governed by two factors, gasoline volatility and its antiknock characteristics. These factors also determine gasoline consumption, as well as availability and refinery configuration and hence

gasoline cost. Those aspects of engine design affecting volatility or antiknock performance, e.g. compression ratio, should therefore be considered as interrelated components of the refinery-engine system. Utilisation of this approach could, theoretically, lead to a minimisation of costs to the producers and consumers of both gasoline and motor vehicles. Recent increases in the price of crude oil and refinery plant make this integrated approach a very desirable option to the refiner and vehicle manufacturer alike.

To fully understand the purpose of lead antiknock additives, it is necessary to examine the refinery-engine, or more specifically the fuel-engine system in more detail. Refined gasoline is a mixture of hydrocarbons and thus the volatility covers a range of values. The initial boiling point of motor gasoline is about 30°C[3], determined by the pentane components, the butanes hardly affecting it. However, the butane components do affect the vapour pressure and hence the hot handling properties of the fuel. To avoid difficulties associated with this, such as vapour lock and evaporation losses, the refiners restrict the butane content of motor gasoline to about 8% by volume, although many refineries tend to have surplus butane blending stocks. The end (boiling) point of gasoline is between 205°C and 220°C[3] to avoid problems with engine fouling and crank-case oil dilution. This temperature range, 30°C - 220°C, represents the boiling range of motor gasoline and hence the quantity of this fuel which can be obtained from crude oil by means of straight distillation. Crude oil contains about 25% of components within this range, although the exact boundaries of the range

are subject to seasonal variation brought about by conflicting requirements for effective cold-starting, warm-up period, full-throttle acceleration and a minimisation of carburettor icing, crank-case oil dilution and vapour lock. Thus it can be seen that not only the octane quality of a fuel, but also the combination of hydrocarbons, governing its volatility, are important factors in the fuel-engine system. Fuel volatility influences engine performance through its effect on the distribution of the air/fuel mixture in the inlet manifold. Table 2-1 shows the octane quality of the main components of motor gasoline, and also the octane quality of the major European fuel grades. Front end octane number is the octane value of the more volatile fractions of motor gasoline which affect the starting performance, from cold, of an engine.

Current vehicle engines have a high thermal efficiency and employ fuels with a high resistance to knock. On the engine side this state of affairs has been made possible chiefly by improvements in ignition systems and increased compression ratios, and on the fuel side by improvements in refinery technology and the use of lead antiknock additives. An increase in compression ratio of 2.5, from 7 to 9.5:1, improves thermal efficiency by 10%. This requires an increase in octane quality of about 10 units (Research Octane Numbers). Thus, a RON increase of 10 units allows a 10% improvement in efficiency, and hence an improvement in thermal efficiency, and so mileage, of about 1% per RON, assuming a corresponding change in compression ratio. Data from the literature suggests this is a reasonable approx-

Table 2-1

Octane quality of main gasoline components

Group	RON
N Paraffins	
nC6	29
I Paraffins	
50/50 iC5/iC6	89
Aromatics	
50/50	108
Benzene/Toluene	
Olefins	
C5/C6/67 mixture	90

Typical European Gasoline Octane Quality

	Premium	Regular
Research Octane Number (RON)	98-100	91-93
Front-end Octane Number	91-92	82-85

imation[4,5,6]. The demand for premium as against regular grade gasoline (4-, and 2-Star respectively) is roughly 80/20[3]. It is therefore premium gasoline quality which determines gasoline process configuration in the refineries. At present, and in the light of proposed EEC legislation on the addition of lead additives to gasoline, premium grade gasoline must have an octane quality of 93-95 RON before the addition of the antiknock agent. The last 5 to 7 octane numbers are provided by the addition of lead alkyl compounds.

However, there is an additional benefit brought about by the use of lead antiknock additives. This is termed the 'Lead Road Bonus'[7]. It is a combination of three effects; lead-in-fuel, fuel sensitivity and engine deposits. The latter point is of little importance for the majority of European vehicles. The most interesting of the above three effects is the first, lead-in-fuel. This means that when a lead antiknock is added to a fuel, the road octane gain is usually greater than the gain in laboratory octane ratings. In practical terms, an engine which gives knock-free operation when fuelled with 98 RON leaded fuel will, in fact knock if a 98 RON unleaded fuel is employed. An added complication is that the lead road bonus may be positive or negative. This is dependant upon the distribution of the high octane gasoline components within the boiling range of a particular fuel and the cars and testing conditions employed. If a fuel's natural high octane numbers are in the lighter fractions and the road octane ratings are obtained from a car that is sensitive to front-end octane number, i.e. it knocks at the start of low speed accelerations, the addition of TEL will yield a negative lead road bonus. This is because TEL gives its octane boost

to the lighter fractions of the fuel, i.e. to the front end gasoline. The average lead road bonus due to the lead-in-fuel effect is shown in Figure 2-1, derived from experimental work on U.S. vehicles.

If RON specification is to be maintained without the addition of lead addtives, the refiner must add more high octane fractions to the gasoline. This increases the RON without raising the Motor Octane Number (MON). This increase in the fuel's sensitivity (RON-MON) leads to a lower road octane rating for the same RON value lead-free fuel. In practical terms the engine no longer operates at its most efficient With respect to the lead road bonus, Hornbeck et al., 1975 conclude;

1) The use of lead antiknocks in European motor gasolines usually provides road octane advantages over and above those indicated by Research and Motor ratings - a positive lead road bonus.
2) The magnitude of the lead road bonus varies with the composition of the base gasolines, the cars considered, and the operating conditions imposed.
3) In European cars and fuels tetramethyllead (TML) shows larger road octane advantages than tetraethyllead (TEL) for most road operating conditions.
4) Lead advantages are greatest when the operating conditions are severe.

Thus the use of lead antiknock additives, under certain conditions, offers a further economic advantage, in addition to the reduction in the quantity of crude oil consumed to produce a given gasoline octane quality; particularly in

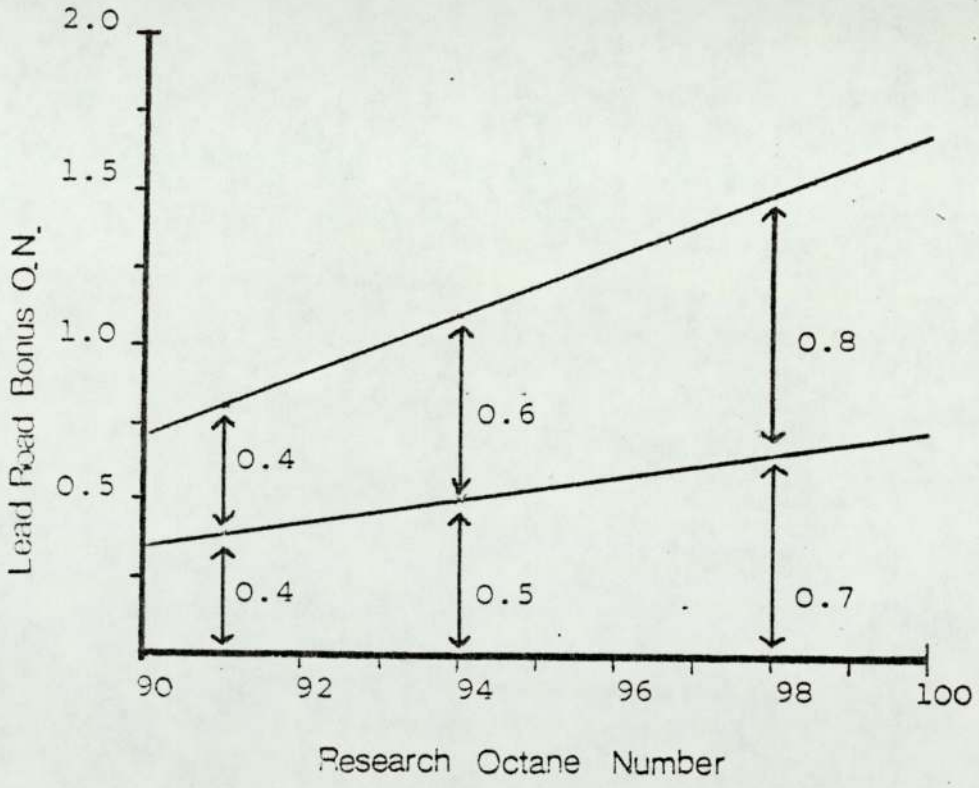

Figure 2-1

From Hornbeck et al., 1975

in the case of high-octane, 98 RON, premium gasoline. At present vehicles requiring this grade of fuel perform acceptably using 98 RON leaded fuel. The nett effect of the lead road bonus would be that these vehicles would require 100-102 RON unleaded fuel.

The quantity of lead antiknock agents added to fuel grades varies from one refinery run to another. A British Standard, BS 4040[8], lays down a maximum allowable level but this is not required in all cases. The actual quantity added will depend on the type of crude oil being refined, the type of refinery plant available, the availability of surplus high-octane blending stocks, if any, the seasonal octane quality and hydrocarbon component variation and market demand for the various gasoline grades. Thus the purpose of lead additives and the quantities employed are not controlled by the same parameter but rather by a range of techno-economic variables.

A major point in favour of the lead alkyls, TEL and TML, as fuel octane quality enhancers is that they appear to have no detrimental side effects[9]. In this they are alone amongst the possible antiknock additives, and between them they have the capability of boosting octane quality across the boiling range of motor gasoline. Thus they give the refiner a tool with which to bring gasoline up to fuel specifications without altering any other refinery or fuel parameters. Lead additives therefore play a double role in gasoline manufacture and use. They permit refiners an unparallelled degree of flexibility in blending which could only be approached at great cost by installation of stand-by

manufacturing and re-blending plant and make available, at a reasonable cost, balanced gasolines of high overall octane quality with a good distribution of octane quality throughout the boiling range and with volatility characteristics suited to modern, high compression ratio engines.

Returning to the effects of lead additives within the engine itself, there is the question of valve seat wear[9]. Since the advent of high octane fuels, made possible by the lead antiknocks, engine compression ratios have increased and consequently conditions within the engine have become more severe. Valve gear and valve seats have been subjected to heavier loading and recent experimental work has shown that the lead antiknock agents provide some degree of protection to the above mechanisms. The results of work on engines using unleaded fuel showed that valves would rapidly wear through cast iron valve seats when no lead was present. This problem may be solved by metallurgical treatment of the valve seats or using valve seat inserts manufactured from a tough alloy, although these options would add somewhat to the cost of the engine. A greater problem would be the reaction of vehicles already on the road; conversion of existing valve seats is possible but requires major servicing of the engine, and if this is not carried out, the use of lead-free fuel could lead to serious engine wear. However, the quantity of lead required to prevent this wear is much less than that used to boost octane quality of gasoline, roughly 33% of the current maximum allowable concentration.

Lead additives have also been shown to reduce the build

up of deposits within the engine[9]. If the quantity of lead additives employed is reduced, deposits build up which increase the octane requirement of the engine. Initial results from the U.S.A., employing lead-free gasoline indicate an increased octane requirement of two to three octane numbers. However, further research on European cars indicates the problem to be less severe for these models.

To recap, lead alkyl compounds were first added to petrol over fifty years ago to combat the phenomenon of knock, or 'pinking'. In its more usual form knock occurs at low speed during full throttle acceleration, or during hill climb, and is readily audible to the driver. The mechanical consequences of this form of knock are not very severe and damage is only likely to occur if the vehicle is operated continuously under these conditions. More recently, as a result of the introduction of high speed inter-urban driving, a more severe manifestation of knock has been discovered. Continuous full throttle driving causes engine temperature to rise and, as heat dissipation may be less than heat generation, knock may occur. This high speed knock will go unnoticed by the driver because of the overall high noise level associated with high speed driving. Although little is understood regarding this phenomenon at present, early research indicates that Motor Octane Number, MON, is more significant than a fuel's RON with respect to reducing high speed knock.

Furthermore the addition of lead additives reduces the build up of engine deposits and valve seat wear, although this latter effect may be brought about by the addition of much less lead than is used at present.

It is apparent that the relationship between the components of the engine-fuel system has been governed by a positive feedback load, brought about by the availability of a cheap, effective octane booster in the form of lead alkyls. The higher the octane quality of available fuel the greater the compression ratio available to the engine designer, and as the engine designer raised compression ratios, the higher the demand for a higher octane quality fuel. This loop raised the octane requirement above that which could be provided by the straight run gasoline pool; currently 91-92 RON as opposed to the 99-100 RON required by engines with a compression ratio of 9:1, or above. To combat this, refiners add lead alkyls to their gasolines to raise the octane quality of the fuels as required and prevent the effects outlined above. However, there has been an important change in the philosophy behind the use of lead antiknock additives since they were first introduced in 1923. At that time engines knocked because the fuels produced by the refineries were too low in octane quality, and lead alkyls were introduced as a solution to this problem. Present day refinery technology, however, could produce gasoline with an octane quality sufficient for all current engines. This would be at an economic penalty of increased energy and crude oil consumption at the refinery, and not all refineries are presently equipped to produce high octane gasolines directly from crude oil. There would be an associated reduction in the flexibility of refinery operations proportional to the reduction in lead additives envisaged.

In conclusion, there has been a reversal of the technical and economic roles of lead additives in recent years. Whereas

originally lead additives were employed to combat the phenomenon of knock, they are currently employed as the most economic means of raising the octane quality of the gasoline pool, in order to satisfy the market demand for premium grade (4-Star) gasoline. This will be discussed further in Section 7.

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SECTION 3

The Elimination of Lead Additives from Gasoline in the United States - A Case Study

Having examined the purpose and operation of lead anti-knock additives in the previous section, it would be useful to present any information currently available to the decision-maker on the pros and cons of lead alkyl reduction or removal from gasoline. The experience of the United States in this matter provides a useful insight into this question and it is proposed to review this in as uncritical and objective a manner as possible in order to prevent any possible confusion regarding the nature and purpose of the U.S. action. Table 3-1 provides a chronology of events up to Ethyl going to court which might be considered useful in giving an 'order of magnitude' time scale for action concerning an environmental pollutant.

From a constitutional viewpoint, the Environmental Protection Agency (EPA) obtained the authority to act from Sections 108 and 109 of the U.S. Clean Air Act, and proceeded to take, in their opinion, appropriate steps to protect public health and welfare from a variety of airborne pollutants, including lead as emitted from motor vehicles.

To date this has been the only well documented debate over the question of lead additives in gasoline. This was in the United States after the EPA promulgated regulations concerning the quantities of lead additives employed in gasoline, in accordance with the 1970 amendments to the Clean Air Act. Specifically, under the Act, the 'issuance of air quality criteria (by the EPA) is a vital step in a programme of responsible technological, social and political action to protect the public from the adverse effects of air pollution'. These health and welfare criteria fulfil the regulatory purpose of serving as the basis upon which the

TABLE 3-1

1972	1 January	EPA publishes "Health Hazards of Lead".
	23 February	EPA's proposed lead regulations published in U.S. Federal Register.
	11 April	EPA revises "Health Hazards of Lead".
	15 May	Ethyl Corporation replies with "Comments of Ethyl Corporation on EPA's Proposed Lead Regulations".
	14 June	EPA Questions in U.S. Federal Register.
	13 July	Ethyl Corporation replies to EPA's Questions of the previous month.
	29 November	EPA publishes new health document, "EPA's Position on the Health Effects of Airborne Lead".
1973	10 January	EPA's Notice of Proposed Rule Making published in U.S. Federal Register.
	9 March	Ethyl Corporation reply to above with "Critique of EPA's Position on the Health Effects of Airborne Lead".
	28 June	Further EPA Rules published in U.S. Federal Register.
	31 October	EPA Rules Published in U.S. Federal Register.
	28 November	Reviewed Support document for lead regulations entitled "EPA's Position on the Health Implications of Airborne Lead".

6 December	Final lead Regulations published.
6 December	Ethyl Corporation petition U.S. Court of Appeals to have EPA regulations set aside.

Administrator of the EPA must promulgate national primary and secondary ambient air quality standards for lead under Section 109 of the Clean Air Act. Thus the EPA was given the authority to control or prohibit the sale of any fuel which might endanger the health or welfare of the public, or which would impair the efficiency of exhaust emission control apparatus, then under development. These exhaust emission control devices were variations of what was termed the 'catalytic convertor'. This used a catalyst bed to reduce NO_x and CO emissions from automobile tailpipes in accordance with the new limits set out under the CAA.

Thus on 23 February 1972 the EPA promulgated a set of regulations[1] which required a phased reduction in the quantity of lead antiknocks added to gasoline to 1.25 g (US) $gallon^{-1}$ in 1977 (0.33 g l^{-1}), and the general availability of a 91 RON (Research Octane Number) lead free of gasoline by 1 July 1974. In fact, at this time, there was no effective exhaust emission control device in existence, but the most promising area of research lay with the perfection of the 'catalytic convertor'. These devices suffered from poisoning of the catalyst bed if lead was present in the vehicle's fuel, and if these devices were to be used to meet the strict 1974 emission regulations there would have to be a lead-free grade of fuel for them. This fact accounts for the necessity for the regulations concerning the 91 RON lead-free grade of gasoline. However, the EPA used their authority, relating to the health and welfare of the general public, to propose those regulations dealing with the reduction, as opposed to the elimination, of lead antiknocks added to gasoline on the grounds that lead

emissions from automobile tailpipes posed a health hazard in their own right. The EPA supported this latter claim with a document entitled "Health Hazards of Lead[2]." It stated that, "This paper contains a summary and analysis of all relevant medical and scientific evidence available to the EPA as of 1 January 1972, on the health hazards of airborne lead". The paper concluded that airborne lead concentrations exceeding 2 ug m^{-3}, averaged over three months, constituted a public health hazard and that to achieve airborne lead concentrations below this level would require a 60-65% reduction in the lead emissions from automobiles. This paper underwent some modification in April, 1972, but the conclusions remained unaltered.

After a brief public discussion, as required by U.S. Federal Law, the EPA revised their regulations and produced two different sets. The first set[3], as before, provided for a 91 RON lead-free grade of gasoline to be generally available as from 1 July 1974. On this occasion, however, it was made clear that the lead-free requirement was based solely on the need to protect catalyst-based emission control devices. The second set of regulations called for the same phased reduction in the quantity of lead antiknocks added to petrol, as in the 1972 regulations, but stating that this was necessary because current airborne lead concentrations constituted a health hazard. This second set of regulations was supported by a revised document entitled "EPA's Position on the Health Effects of Airborne Lead[5]." Final regulations[6] were promulgated on 6 December 1973, the only change being that even more severe lead reductions were imposed, with the permissable quantity of lead antiknock being limited to 1.7 g $gallon^{-1}$ (0.45 g l^{-1}) of

the total gasoline pool in 1975 and to 0.5 g gallon^{-1} (0.13 g l^{-1}) in 1979. These final regulations were supported by a third document entitled "EPA's Position on the Health Implications of Airborne Lead[7]."

Whilst the regulations were being produced Federal Law required the EPA to call for public comment. It is not surprising to learn that the most vociferous of its critics was the Ethyl Corporation who, along with Du Pont, are the major U.S.manufacturers of lead antiknock compounds. The extended debate between the EPA and the Ethyl Corporation represents, as mentioned at the beginning of this section, one of the best documented examples of the arguments concerning the removal of lead additives from gasoline. It should be remembered, however, that not all of the U.S. arguments can be applied to the U.K. situation due to various factors such as differences in the motor vehicle populations and refinery techniques in the two countries. Nevertheless, the fundamental problems caused by lead in the environment which originated in motor vehicles are the same in both countries and the options open to the decision-maker are basically the same.

The debate is essentially about values, in particular environmental values, and in many ways it is unique. It is well documented, unlike many such arguments, and it was carried on between parties who had ample specialist knowledge of the topic in question. At this point it is important to note that the case was, of course, governed by U.S. Law and hence any legal points, or technicalities may well not apply in the U.K.

The EPA opened the debate with their proposed regulations concerning lead-free fuel and the phased reduction of lead

additives in the remaining fuel grades. There can be no argument, on technical grounds, against the first part of the rules concerning the necessity for a lead-free grade of gasoline to be available from 1974, although the Ethyl Corporation argued that there would be no practical necessity for such a grade at that time. The only device capable of satisfying the proposed 1975-76 motor vehicle emission standards was the catalytic convertor and there was no doubt that this device required the use of lead-free fuel if it was to operate effectively, but the Ethyl Corporation suggested that it would not be developed to production standards by 1974. In the subsequent case in the U.S. Court of Appeal this first point was not contested by the Ethyl Corporation. On this point therefore, both sides agreed upon the facts of the matter and the debate regarding it virtually ceased, other than the Ethyl Corporation suggesting that there might well be no viable emissions control device available to meet the 1975-76 standards. The Ethyl Corporation also referred to what they considered to be the excessive costs of meeting the proposed emission standards alleging that they would only bring about a marginal improvement in the environment.

However, the second part of the EPA's proposed regulations was subject to a much more rigorous attack by the Ethyl Corporation. The EPA stated that reducing the quantity of lead antiknocks employed in gasoline would eliminate a possible health hazard and supported this with "Health Hazards of Lead[2]." This document was based on the National Academy of Sciences report "Airborne Lead in Perspective[9]" which concluded that;

1) Since lead has not been shown to have any biologically useful function in the body, any increase in body burden of lead is accompanied by an increased risk of human health impairment.

2) In many cities air lead concentrations are slowly rising.

3) Human blood lead levels begin to rise appreciably with an exposure to airborne lead concentrations in excess of 2 ug m^{-3}.

4) Elevated lead intake for periods as short as three months produce an increase in blood lead levels.

5) Body burdens of lead increase with age, at least to 40 years, and probably thereafter.

6) Although the ingestion of leaded paint is the predominant cause of lead poisoning in children, some children may show high blood lead levels from the ingestion of dust contaminated by fall-out from airborne lead.

7) Average blood lead levels tend to be higher among urban residents than among rural residents and higher among groups occupationally exposed to vehicle exhaust. (e.g. traffic police and garage workers)".

Basing his case on the above, the Administrator of the EPA recommended that "... airborne lead levels exceeding 2 ug m^{-3}, averaged over a period of three months or longer, are associated with a sufficient risk of adverse physiological effects to constitute endangerment of public health. Since airborne lead levels in many major urban areas currently range

from 2 to somewhat over 5 ug m^{-3}, and since motor vehicles are the predominant source of airborne lead in such areas, attainment of a 2.0 ug level will require a 60-65% reduction in lead emissions from motor vehicles[2]".

The Ethyl Corporation's reply to the EPA's case, contained in the document entitled "Comments of Ethyl Corporation on EPA's Proposed Lead Regulations", was very extensive, although it concentrated on contradicting the EPA's opinion that, "reducing the amount of lead in gasoline would eliminate a health hazard". Ethyl claimed that current airborne lead concentrations did not constitute a health hazard and presented the following claims to support their case;[10]

1) Years of experience with occupationally exposed groups show blood lead levels well in excess of those found in the normally exposed population to be perfectly safe.
2) The evidence indicates that air lead concentrations in many cities are falling. U.S. population blood levels are of the same order as those for many non-industrialised populations, indicating that lead from industrial sources makes only a small contribution to blood lead levels.
3) The data used in the calculation of the 2.0 ug m^{-3} limit is seriously suspect, as is the statistical device used in the calculation, the Goldsmith-Hexter Regression Line. More reliable data (The 7-City Study) shows no correlation between air lead levels and blood lead levels. In addition, the EPA assumed that about 30% of the lead inhaled

is retained in the lung. The true figure is nearer 10%.

4) There is no evidence to indicate that the alleged "excess" blood lead levels resulting from exposure to airborne lead are a health hazard.

5) The data on lead body burdens shows that they do not increase with age. Even if they did, this would merely reflect the very long time (circa 30 years) required for the body to come into equilibrium with environmental lead.

6) There is no known correlation between lead levels in dust and earth and the blood lead levels of children exposed to the earth and dust. There is no evidence whatsoever for the EPA's hypothesis concerning dust being a significant contributor to the blood lead of children. The rate of lead fallout is so low that it can only be an insignificant source of lead.

7) The very large 7-City Study, 1972 reveals no correlation between air lead levels and blood lead levels.

As can be seen, these facts provided a point-by-point counter to the EPA's case. Also the Ethyl Corporation went to some length to point out that there has never been a case of lead poisoning attributed to lead emissions from motor vehicles. Ethyl also described the economic and environmental penalties that would be brought about if the EPA's proposed regulations were enforced as follows;[10]

1) 5% increase in crude oil consumption due to the use of less efficient engines. This could add $1.4 billion to the balance of payment deficit.

or 2) The cost to the consumer would be about C4.7 per gallon due to higher gasoline costs and lower engine efficiency.

or 3) Extra refining investment to meet the requirement for low lead gasoline would amount to $4 billion. Small refineries would be unable to make such a large investment and would have to cease trading.

If lead compounds are not added to gasoline, the only way the required octane quality can be achieved is by altering the chemical composition of the product. This is done by altering the refining process to produce higher volumes of aromatic hydrocarbons of high octane value, thus increasing refinery costs and energy consumption. Ethyl suggest that this action may pose an environmental hazard since;[10]

1) Emissions of carcinogenic polynuclear aromatic hydrocarbons (PNA's) would increase.

2) Emissions of those compounds responsible for photo-chemical smog and eye irritation would also increase.

3) Although the EPA dismissed the above two points on the grounds that possible increased emissions of these compounds would be dealt with by the catalytic convertors, to be fitted to motor vehicles, the Ethyl Corporation pointed out that no such production convertor existed at that time, and those prototypes that did found difficulty in

coping with emissions from fuels containing a high percentage of aromatic hydrocarbons.

If the EPA accepts the facts and claims produced by the Ethyl Corporation, then they must revise their regulations. Ethyl offered an alternative[10] to the EPA's proposed regulations, suggesting that a lead-free grade of gasoline be made available from when the catalytic convertor was known to work, other grades of fuel remaining unchanged. All new vehicles would be fitted with this device, leading to a gradual elimination of leaded fuel. If, on the other hand, a lead-tolerant emissions control device was developed in the meantime, one could continue to use leaded fuel and eliminate lead from the emissions via a lead trap, then under development. This strategy offered apparently the same benefits as the EPA's plan, but at a vastly reduced cost. With this state of affairs the first phase of the debate came to a close.

In response to the objections raised by the Ethyl Corporation, and others, the EPA revised their proposed regulations. As previously described, the EPA's first proposed set of regulations based the need for both lead-free gasoline, and reduction in the quantities of lead employed in other grades, on human health considerations. In the revised regulations, the EPA now based the need for a lead free grade of gasoline solely on the assumption that, if catalytic convertors were going to be employed to control automobile emissions, they had to have lead free gasoline freely available. The reductions in the quantities of lead added to other grades of gasoline, however, were still based on health considerations. The revised regulations were promulgated in January, 1973 and the health

issues were contained in a document entitled, "EPA's Position on the Health Effects of Airborne Lead[5]". Shortly afterwards, the Ethyl Corporation published their reply entitled, "Critique of EPA's Position on the Health Effects of Airborne Lead[12]".

At this time the EPA dropped the claim that blood lead levels rise when exposed to airborne lead concentration in excess of 2 ug m^{-3}. The point had been severely criticised by the Ethyl Corporation in the first phase of the debate, and is ignored by the EPA from this point on. However, the EPA reiterated that lead has no known biological function so that any increase in body burden increases the risk of health impairment; that fallout from airborne lead in dust may be a significant exposure route for children; and that blood lead levels tend to be higher for urban residents than non-urban residents, this claim now being supported by evidence from the 7-City Survey[13]. Additionally the EPA argued that:

1) Many city dwellers have abnormally high blood lead levels.
2) The susceptibility of children may be greater than adults so that children may be suffering subtle but unrecognised neurological impairment due to lead.
3) Newborn babies in cities have higher blood lead levels than newborn babies in rural areas.
4) Chromosomal damage due to lead is possible.
5) Presently recognised blood lead limits are too high to protect the public. Upper acceptable limits for blood lead for the following groups were revised to:

Expectant mothers	30 ug 100 ml^{-1}
Newborn babies	30 ug 100 ml^{-1}
Children	40 ug 100 ml^{-1}
Adults	40 ug 100 ml^{-1}

This last point is dependent upon both sides accepting the background value that the upper acceptable limit for a toxin in the body is the lowest level at which the health of someone in the population is impaired. It forms the most important part of the EPA's argument and in turn is severely criticised by the Ethyl Corporation. The Ethyl Corporation denies that there was any medical evidence for the EPA's claim that the lowest blood lead level, at which the health of some expectant mothers (newborn child, child, adult) is impaired, is 30 (30,40,40) ug 100 ml^{-1}. The evidence cited by the EPA concerned the study of umbilical cord blood lead but they found no urban-rural gradient, and concluded that there was no evidence to implicate airborne lead as a contributor to high umbilical cord blood lead levels. The Ethyl Corporation accused the EPA of making an ad hoc move in redefining upper acceptable blood lead levels without supporting evidence. They also pointed out that the U.S. Surgeon General considered children with blood lead levels below 50 ug 100 ml^{-1} to be in no danger, provided that there was no evidence of on-going high exposure. Again the Ethyl Corporation provided a point-by-point counter to the EPA's argument;

1) The upper blood lead level for city residents is around 40 ug 100 ml^{-1} which cannot be said to be abnormal. Many higher values are found to be the result of faulty analysis. For children, elevated

blood leads are due solely to the ingestion of leaded paint.

2) The claim that urban newborn children have higher blood lead levels than non-urban infants is in direct contradiction to the paper cited by the EPA.

3) The evidence for chromosomal damage originating from lead exposure is extremely speculative.

The Ethyl Corporation had expanded upon some of the points which had arisen during the first phase of the debate. An extensive survey of childhood lead poisoning had failed to identify a single case where dust could be implicated as the lead source. Well in excess of 90% of the cases reported were the result of ingesting chips of lead paint. Hence, the EPA's claim that dust, contaminated by fallout from airborne lead, might be a significant source of lead exposure for children was thrown into serious doubt. The Ethyl Corporation also cited the results of animal studies which suggested that lead, after all, might have some biological function, again contradicting the EPA's claim. In conclusion, the Ethyl Corporation argued that, on the EPA's own admission, the removal of lead paint from ageing housing would be considerably cheaper than the proposed reduction in the quantity of lead antiknocks added to gasoline. Such a programme of renovation would prevent the vast majority of existing cases of overt lead poisoning in children. It is apparent that it is not the values of the parties that are in dispute; both sides are agreed that, if a step would reduce the incidence of childhood lead poisoning, then that step ought to be taken. However, both sides dispute

the facts they each offer to support their case; e.g. the costs and the effectiveness of the various options put forward by both parties. Thus it is the factual element, rather than the value element, which is again to the fore at the close of the second phase of the debate.

As a result of the discussion during the second phase of the debate, the EPA again revised their position on the health aspects of lead emissions from automobiles in a third document entitled "EPA's Position on the Health Implications of Airborne Lead[7]". This document was used to support the final revision of the proposed regulations issued in December, 1973[6]. Unlike their earlier documents, this one was not open to public comment and the Ethyl Corporation was not allowed to reply. Undoubtedly the EPA considered that the argument had continued for a long enough period. Nevertheless, the final document contained several major changes, apparently due to the Ethyl Corporation's criticisms:

1) It was admitted that there was no solid evidence suggesting that children are more susceptible to lead than adults. The special position of children was now based on their higher exposure from paint, dust and dirt.
2) The earlier recommendations for upper acceptable blood lead levels for expectant mothers and newborn children were dropped. Instead, the limit for the four groups mentioned was set at 40 ug 100 ml^{-1}.
3) The EPA argued against the Ethyl Corporation's claim that there is no correlation to be found

between the air lead concentration and the blood lead level.

The EPA also expanded considerably two of their earlier claims; that low levels of lead exposure may cause subtle, "subclinical", neurological changes in children and that lead fallout contaminated dust is a major source of childhood exposure to the metal. The EPA concluded that in each case the evidence for the above claims was not conclusive, but that taken together, they pointed to the fact that in all probability they were correct. Quite new to the debate was a calculation of the probable increase in blood lead of a "standard man" exposed to various air lead concentrations. This calculation purported to show, that to keep below the recommended blood lead level of 40 ug 100 ml^{-1}, the ambient air lead concentration should be below 11.8 ug m^{-3} on optimistic assumptions and below 4.0 ug m^{-3} on pessimistic assumptions.

It is apparent from the debate that the values of both parties, the EPA and the Ethyl Corporation, were virtually identical in that both sides agreed that everything feasible ought to be done to benefit the environment and public health. Certainly neither party would ever openly contradict the above statement. However, throughout the argument both sides hotly contested the factual evidence produced by the other. In certain instances evidence was presented in a deliberately confusing manner. For example, in order to decry the Goldsmith-Hexter relationship between air lead concentrations and blood lead levels, the Ethyl Corporation showed it superimposed on the 7-City Study data. However, they failed to indicate that Goldsmith and Hexter's data was from male subjects whereas the

7-City Study's data was from female subjects, and it is known that female blood leads are consistently lower than male blood leads, and this is ignoring the fact that the studies were conducted at different times, that analyses were performed in different laboratories and that analytical techniques altered during the intervening period between the studies. It is interesting to note that this misrepresentation went hand-in-hand with a perfectly valid criticism of the statistical technique employed by Goldsmith and Hexter to derive their regression function and the fact that they had 'estimated' some of their data. The Ethyl Corporation were not alone in their use of this technique. The EPA had produced their maximum allowable air lead concentration of 2 ug m^{-3} originally from Goldsmith and Hexter's work. As a result of criticism of the Goldsmith-Hexter function the EPA later used the function cited in the NAS study[9] to provide the relationship between air lead and blood lead level. However, the EPA continued to cite 2 ug m^{-3} as the maximum allowable air lead level.

Returning to the debate, the fourth, and final, round commenced on the 6 December 1973 when the EPA promulgated their final regulations on the lead content of gasolines[6]. On that date the Ethyl Corporation petitioned the U.S. Court of appeals to have the EPA's regulations set aside on the grounds that[8a]:

1) The EPA has exceeded its statutory authority and violated its obligation to engage in a principled, reasoned decision-making process in issuing the regulations:

   A. The EPA has issued the regulations without the required statutory findings or basis in

the record that the vehicle emission products of lead antiknocks will endanger the public health or welfare.

B. The EPA's evaluation of the medical and scientific evidence as to the alleged health effects of lead antiknocks, as stated in the preamble to the regulations and the Agency's supporting health document, reveals such serious inconsistencies and errors in the methodologies and assumptions utilised as to constitute arbitrary and capricious action.

2) The EPA has violated basic requirements of administrative due process:

A. The EPA issued final lead reduction regulations and the supporting health document without giving interested parties or the public an opportunity to participate in the final rule making by submitting written data and comments.

B. The EPA refused to allow interested parties an opportunity to cross-examine witnesses and challenge submitted evidence in the only hearings held by EPA to consider the complex medical and scientific issues involved in the lead reduction regulations.

3) The EPA's failure to prepare an environmental impact statement or its functional equivalent violated the requirements of the National Environmental Policy Act.

As can be seen, from all three of the above counts, the Ethyl Corporation charged the EPA with exceeding its authority

and, if that was the case, there was no alternative but to set aside actions carried out while so doing. This is made very clear in the reply brief[8b];

1) The EPA does not have statutory authority to restrict the use of lead antiknocks in motor gasoline unless it establishes on the basis of the record that the vehicle emission products from such additives themselves will endanger the public health and welfare:
   A. The statutory condition has not been met.
   B. Legislative history refutes the EPA position.
   C. Recent oversight hearings confirm Congressional Intent.
   D. Recent decisions of this Court relied on by the EPA are not applicable.
2) The EPA has not demonstrated a rational, principled decision-making process in its review of the medical and scientific evidence in this case.

Of the above points, C. and D. require some clarification. C. refers to hearings carried out by the Panel on Environmental Science and Technology of the Senate Committee on Public Works which suggested that the EPA's case was not as strong as the EPA suggested. D. refers to the EPA's asserting the right to make "policy choices" based on "imperfect data". To do this the EPA cited two recent Court cases, decided in its favour, which support this approach. However, the Ethyl Corporation argued that these cases were being quoted out of context. A majority verdict was given in favour of the Ethyl Corporation, and the EPA's regulations were set aside. Their decision was

based mainly on Section 211 of the Clean Air Act which says that, in order to be subject to control, a pollutant must be such that it, "will endanger" the public health and welfare. The majority said that the EPA had not sufficiently proved their case and so their regulations should be set aside.

In turn the EPA appealed against this decision. To this the Ethyl Corporation replied[8c];

1) The majority was right in holding that the "will endanger" standard of Section 211 of the Clean Air Act requires a high standard of proof which is not satisfied by the hypotheses and speculations of the EPA:
   A. The clear language of the Statute confirms the majority's interpretation of the "will endanger" standard.
   B. A comparison of Section 211 to other Sections of the Clean Air Act confirms the majority's interpretation of the "will endanger" standard.
   C. The legislative history of Section 211 supports the majority's interpretation of the "will endanger" standard.
   D. The recent cases relied on by the EPA do not support the Agency's construction of Section 211.
2) The majority was right in holding that the EPA was arbitrary and capricious in its decision-making process:
   A. The in-depth review of the record by the majority was fully justified and proper.
   B. The EPA has not engaged in a rational, principled decision-making process in its review of the medical and scientific evidence

as to the alleged health effects of lead antiknocks.

D. above refers to the EPA's claim that "recent case law" had established the rule that "all that is needed to show endangerment is the potential for serious harm", which contradicts the spirit, if not the letter, of Section 211 of the Clean Air Act cited above. However, again the Ethyl Corporation argued strongly that these cases could not be related to the lead antiknock situation and that they were being cited out of context.

It is apparent, then, that in this record appeal the Ethyl Corporation, naturally, relied heavily on the fact that they had won the first appeal on a point of law, namely Section 211 of the Clean Air Act, and as far as they were concerned this fact had not altered. In the end, however, the nine members of the Appeals Court found in favour of the EPA, a five-four decision, and the regulations passed into law.

Looking back over the whole EPA versus The Ethyl Corporation case, one can see that it was fought in two areas, scientific and legal. Throughout the debate both parties sought to refute the factual evidence put forward by the other in support of their case, and frequently both sides drew conflicting conclusions from the same piece of research; viz. Goldsmith and Hexter's work on air-blood lead relationships. The Ethyl Corporation argued strongly that, if automobile lead emissions could be proved to be harmful to the public health and welfare, they should be controlled by the use of lead particulate traps which involved a much lower economic penalty than the reduction and elimination of lead alkyls from gasoline. The Ethyl

Corporation had carried out extensive research on traps and was confident, perhaps not surprisingly, of their effectiveness. Likewise, the Ethyl Corporation never challenged the need for lead-free gasoline for automobiles fitted with catalytic convertors, although they did say that the legislation for the lead-free fuel was premature, there being no operational, effective convertor in production at that time, 1972.

Latterly, in the legal arena, the scientific evidence took on a secondary role. The Ethyl Corporation cited the EPA's scientific and medical arguments as evidence that it had not carried out a "principled, reasoned decision-making process" as required by the law, and that the EPA had misinterpreted Section 211 of the Clean Air Act. They also faulted the EPA on the grounds that they had not acted in the approved manner when they had produced their regulations; i.e. the EPA had violated "administrative due process". Hence, it can be seen that the Ethyl Corporation's case rested not upon disproving any of the EPA's scientific and medical evidence, but rather upon proving that, in producing the proposed regulations, the EPA had acted outside the Law. If this could be shown, the regulations would have to be set aside without further argument and the EPA would have to have seriously delayed its legislation.

The case also had the role of a test case for the EPA, in which they would win or lose the power to introduce legislation on the basis of possible harm. It is often the case with environmental issues, such as pollution, that it is not possible to know the long-term effects of an on-going pollution situation, while at the same time knowing that it might well be too late to take any action if one waits until the onset of these long-term effects. Thus action has to be

taken in a state of ignorance as to the long-term effects on the grounds that they will most likely be harmful. This was the EPA's argument in the case of lead emissions from automobiles. Although no one piece of evidence suggested conclusively that the continued rise in lead emissions from automobiles would lead to a public health problem, the EPA argued that, taken together, the available evidence pointed to legislation being brought in as soon as possible to curb this potential health hazard. Thus, in winning their case, the EPA obtained a vital precedent which would enable them to act in a similar manner on future occasions without the necessity of legal action.

It is difficult to say just how far the EPA versus the Ethyl Corporation debate can be related to the U.K. scene. Certainly the sole U.K. manufacturers of lead antiknocks, the Associated Octel Company Ltd., would oppose the introduction of low lead, 0.15 g Pb/l, or lead free gasoline. In common with the Ethyl Corporation, they have carried out extensive research into lead particulate traps at their engine laboratory in Bletchley, Buckinghamshire, and argue strongly that these devices offer a much more economically viable means of controlling vehicle lead emissions than reducing, or removing, the lead antiknocks in gasoline[14].

However, it should be remembered that there are major differences between the U.S. and U.K. regarding vehicle lead emissions. Starting with the volume of gasoline produced; in Europe approximately 20% of the barrel is refined to gasoline whereas in the U.S. the figure is 50-55% due to a much greater demand. Thus the problem is much more severe in the U.S., particularly in cities. There are also major differences

between U.S. and European vehicle engine design, notably cubic capacity, but also in compression ratio - both factors which exert an influence on engine lead emissions. Not only are there differences in the physical causes of the problem, but also the approach to the problem's solution would be different in the U.K. as opposed to the U.S. For example, a private company in the U.K. cannot take the government to court in order to prevent a proposed piece of legislation from becoming law. However, there has been extensive debate on the subject of vehicle lead emissions between environmental pressure groups and both the antiknock manufacturers and the government which has a strong similarity to the debate between the Ethyl Corporation and the EPA.

In conclusion then, the Ethyl Corporation/EPA case provides a valuable discussion of the case for and against the use of lead antiknocks to improve the octane quality of gasoline. Much of the medical and scientific evidence referred to by both parties remains unchanged to the present, and hence provides valuable information for the design of environmental lead control strategies. The nature of the U.S. system of government provided the means for an open debate between all interested parties and, to date, it remains the only such case to be documented. The only European country to introduce severe lead antiknock controls, Germany, did so through direct legislation not open to opposition outside parliament. Thus, although five years old, the case remains a valuable source of information on many facets of the problem of controlling vehicle lead emissions, as a means to environmental lead reduction.

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SECTION 4

The Concept of Cost-Effective Analysis and its Application to the Control of Environmental lead. The Use of Modelling

The objective of this research is, in some way, to evaluate a variety of control strategies for environmental lead. It is therefore necessary to develop, or select, a methodology by means of which the strategies may be compared, one against another. However, it must be taken into account that the strategies involved may differ in both concept and technology required for their implementation. Before examining techniques for their comparison and assessment it is necessary to outline, briefly, the types of strategy as they will have a bearing on the technique finally selected. Whilst having referred to strategies, the term has not yet been clearly defined. A strategy is any way of lowering people's exposure to lead. Strategies for handling the lead problem must be clearly distinguished from policies. A policy, in this context, is a political decision to tackle the problem of environmental lead exposure by implementing a particular strategy, or set of strategies, for its control.[1] It is obvious that if doing something will reduce exposure to lead, the thing in question is a strategy for controlling lead. Whether or not a policy decision is taken to employ this strategy is a normative question beyond scientific investigation. This study is limited to a discussion of strategy. This may be, however, somewhat artificial - the very best strategy may be completely, politically unacceptable, but this appears to be an inevitable restriction. In considering possible strategies it soon became apparent there were three areas of particular importance; lowering lead levels in water, lowering lead levels in canned food and reducing the amount of lead additives in gasoline. As described more fully in Section 1 environmental exposure

to lead is by two main routes: via the diet and via air breathed by the individual. The control strategies considered here relate to the latter route, the lead originating from lead added to gasoline. Those strategies relating to canned food and water have been examined in the PhD thesis of Edwards, 1979[2].

In excess of 90% of airborne lead is the result of emissions produced by the combustion of gasoline containing lead antiknock additives[3]. Hence, the strategies under consideration relate to the control, by various approaches, of lead emissions from motor vehicles, or the reduction or elimination of these emissions would have a significant impact on environmental airborne lead concentrations.

When the decision - or policy-maker, has to choose between competing options some method of comparing them on equal terms is required. Two main techniques are employed for this purpose, Cost-Benefit Analysis (CBA) and Cost-Effective Analysis (CEA). Both were examined to ascertain which would be the more suitable to provide decision criteria regarding control strategies for environmental lead.

The fundamental concept of Cost-Benefit Analysis is very simple. If the decision-maker has to decide whether or not to do 'X', then the rule is "Do 'X' if the net benefits exceed those of the next best alternative course of action , and not otherwise". If this is applied to all possible decision options, the largest possible benefits ought to be identified, for given constraints. For a problem such as lead pollution in the environment, this latter outcome would appear to be a desirable goal. Continuing the above concept a little further, the net benefits of the next best alternative to 'X' are the

costs of 'X' because if 'X' is done, then the net benefits of the next best alternative to 'X' are lost. Hence the above rule becomes "Do'X' if its total benefits exceed its cost" and thus one obtains the general rule of Cost-Benefit Analysis. This is illustrated by the following example, taken from Layard, 1974[4]. It is being considered to build a bridge over a river which can only be crossed at present by means of a ferry. The ferry is a privately owned monopoly whereas the bridge would be funded by the government. At present ferry charges are £0.20 per crossing whilst its total costs per crossing are £0.15; it is used for 5,000 crossings per year. The bridge on the other hand would cost £30,000 to build but would be open free of charge, although being further upstream than the ferry, the crossing time would be the same, with 25,000 crossings a year expected. In any CBA it is usual to proceed in two stages:

a) Value the costs and benefits in each year of the project.

b) Obtain an aggregate 'present value' of the project by 'discounting' costs and benefits in future years to make them commensurable with present costs and benefits, i.e. obtain the Net Present Values (NPV's) of the costs and benefits.

At each stage the appraisal differs from commercial project appraisal because the costs and benefits to society are included in the calculations as opposed to the purely financial considerations of the latter. All parties affected by the project are noted and the effect of the project on their welfare is valued in money terms as they would value it.

In this case there are four parties; taxpayers, who pay for the government's bridge, the ferry owners, existing travellers and new travellers.

1) The taxpayers lose £30,000 now.
2) The ferry owners lose their profit of £250 p.a. in each future year for ever.
3) The existing travellers gain £1,000 p.a. in each future year for ever due to the fall in crossing cost.
4) The new travellers. Their gains are more difficult to value.

The new journey which is most highly valued was nearly made by the ferry, and so may be considered to be worth nearly £0.20. The journey which is least highly valued may be considered to be worth a little more than £0.00. If it is assumed that journey value falls linearly from £0.20 to £0.00, an intermediate journey value of £0.10 is obtained and so the gain to new travellers is £2,000 per year for ever. Table 4-1 shows the net benefits, i.e. benefits minus costs. Ignoring the possible difficulties, which will be discussed shortly, if this table is added up, the total is - £2,500 on the basis of which CBA suggests the project should be turned down.

However, the application of this apparently simple methodology, as shown by the above example, is fraught with difficulties surrounding the measurement, and valuation, of the costs and benefits of the project. The first question which must be answered is "Can everything be valued on the same scale?" If there are objections to this on practical,

Table 4-1

	Future net benefits p.a. for ever	N P V at 10% discount
Ferry owners	- £250	- £2,500
Existing travellers	+ £1,000	+ £10,000
New travellers	+ £2,000	+ £20,000
Taxpayers	-	- £30,000

or technical grounds, then the problem may be overcome by making an estimation. If, however, there are objections on theoretical grounds to measuring, or valuing, everything on one scale, then it is reasonable to ask of those carrying out the CBA what is meant by saying that 'A' is better than 'B', unless they have some means of directly comparing the differing dimensions of 'A' and 'B'.

However, before considering its application to control strategies for environmental lead, it is necessary to define the valuations which may have to be made in a CBA[4];

1) The relative valuation of different costs and benefits occurring at different points in time: the problem of time preference and the opportunity cost of capital.
2) The relative valuation of different costs and benefits at the time when they occur.
3) The valuation of risky outcomes.
4) The valuation of costs and benefits to people with different incomes.

Lead emissions from motor vehicles may be reduced in two ways, both technically quite different. Firstly, the quantities of lead alkyl antiknock compounds in the vehicles' fuel may be reduced, or eliminated and secondly lead particulate traps may be fitted into the vehicles' exhaust systems. Furthermore, these two techniques may be combined in a joint strategy involving the reduction of lead antiknocks and the trapping of the remaining lead particulates in the exhaust gases. With respect to the costs and benefits of these

strategies, considerable research has been carried out into the valuation of the costs, e.g. the RUFIT[5] study and the TRRL Report on Lead Traps[6], although comparatively little research has been centred on the valuation of the benefits.

However, perhaps the best way to evaluate the utility of CBA in determining decision criteria for control strategies for environmental lead is to follow through an example, albeit in a general manner. A frequently suggested option in the removal of lead additives from gasoline leading to the introduction of lead-free fuel, with no change in octane quality, as is the case in the United States discussed in Section 3. CBA of whether, or not, to introduce this strategy suggest the following.

Those affected by the strategy:

1) The gasoline industry - the refiners.
2) The lead additive manufacturers.
3) The car-owners.
4) All individuals in society.

The following costs would have to be valued:

1) Initial capital investment at refineries to install equipment to maintain current octane quality for lead-free gasoline pool.
2) Annual increased consumption of crude oil to provide current octane quality for lead-free gasoline pool per annum per year.
3) Annual increased consumption of crude oil within the refinery for energy purposes to maintain current octane quality for lead-free

gasoline, per annum for ever.

4) There may be increased costs to car owners for maintenance due to value seat recession brought about by the use of lead free fuel. For 10 years until the entire car population is renewed with suitably designed vehicles, assuming 10% p.a. car sales are new cars.

The benefits to be valued are:

1) Improvement in populations health brought about by the reduction in individuals' exposure to airborne lead, per annum for ever.

It is important to note the one major transfer also; the lead alkyl manufacturer will lose all U.K. revenue, per annum for ever. However, this is compensated for by the benefit accruing to other lead users brought about by a fall in the price in lead due to the reduced consumption. Furthermore, the alkyl manufacturer, there is only one in the U.K., would be unlikely to cease trading as it would still have a considerable export market, and so the strategies effects on this industry need not be considered further.

Examination of the above costs and benefits shows that the costs are all economic factors which naturally lend themselves to valuation in money terms. The benefit, however, poses a more difficult problem, in terms of valuation, of a three-fold nature. Firstly, what is the benefit? Reduction of the population's exposure to lead, it is suggested, would lead to an improvement in the quality of life because reducing exposure to a known toxic substance should lead to a reduction in the harm caused by that substance. However, it is impossible

to measure that improvement by any scientific method at present, and so an indicator of harm has to be used, normally blood lead, i.e. a reduction in blood lead is taken to represent a reduction in harm caused by lead. Secondly, whilst this reduction may be measured for an individual, it is impossible to predict the distribution of blood lead reductions which would occur throughout the population, because of the high individual variation in those factors governing human response to lead exposure, coupled with quantitative variation in exposure from the various sources across the population. Furthermore, the distribution of the U.K. population's blood lead has not yet been determined with any degree of accuracy[7]. Due to the fact that the effectiveness of a strategy cannot be measured for the population as a whole, this measurement was made in terms of the effect of the strategies on a standard man, as fully described in Section 5. Thirdly, the change in blood lead for a standard man cannot be directly related to a monetary valuation. This latter point is the classical problem of CBA - valuation. A CBA requires the reduction of all costs and benefits to the same units, commonly money, otherwise it cannot proceed. It is evident that the benefit in the above example, which it should be noted will be common to all control strategies for environmental lead, cannot be realistically valued in money terms at all. For this reason it is therefore apparent that CBA cannot provide the decision-maker with any useful information regarding lead control strategies.

Cost-Effective Analysis (CEA) adopts a different approach, the main advantage of which is that it avoids the necessity of

valuing the benefits of a strategy. The basic principle, less complicated than that of CBA, is that the cost-effectiveness of various projects are compared, and the project with the highest cost-effectiveness is recommended. There is one proviso, however, and that is that the projects ought to be alternatives means to the same end. Returning to the bridge-ferry example this means that no means of crossing the river currently exists and a decision has to be made whether to build a bridge or install a ferry service.

Let the capital cost of the ferry and docking points be £10,000. The total costs per car per crossing is £0.15 and it is expected that there would be 5,000 crossings per annum. There is no reason to expect that a ferry installed from scratch would be used any more than that described in the original example. The bridge on the other hand costs £30,000, and would be operated on a toll-free basis. Table 4-2 shows the costs and effectiveness of the two projects. From this, the bridge project would be accepted as it gives 0.83 crossings/£1, as opposed to the ferry's 0.29 crossings/£1, and is obviously the more cost-effective project. This further assumes that finance is available for either project without restraint, i.e. the taxpayer can actually afford the more expensive bridge project.

The above example of a CEA was simple in that the costs and effectiveness could be obtained directly from the data measured; i.e. the amounts of money to be spent and the number of crossings to be made. The chief obstacle to CEA is finding a satisfactory measure of the effectiveness, and this is true for environmental lead control strategies. It has

Table 4-2

	Capital Cost	Running Cost p.a.	N.P.V.
Bridge	£30,000	-	£30,000
Ferry	£10,000	£750	£17,500

The N.P.V. assumes a 10% discount rate.

	N.P.V.	Crossings p.a.	Cost-Effectiveness (crossings/£1)
Bridge	£30,000	25,000	0.83
Ferry	£17,500	5,000	0.29

already been mentioned that all the strategies under consideration will relate to the reduction of lead concentrations in the atmosphere by means of reducing or eliminating lead emissions from vehicles. It could be argued then that the strategies' effectiveness could be measured in terms of the reductions that they achieve. However, if this was to be the case, these strategies could not be compared with strategies to control lead exposure from the diet as described by Edwards, 1979[2]. A comparison could be made if the effectiveness of all strategies were measured in terms of daily exposure to lead but this is not an absolutely complete picture. Inorganic lead exposure from the atmosphere and the diet is not all absorbed to the blood, some being excreted, whereas all organic lead exposure is absorbed directly to the blood via inhil ation, ingestion or skin absorbtion. At present environmental exposure to organic lead is small, but if it is to be possible to compare strategies for the control of all forms of lead, in terms of cost-effectivess then the effectivenes has to be measured in terms of the changes particular strategies bring about in blood lead concentration. This is because blood lead is the only measure of effectiveness which is common to all strategies which it is possible to envisage at present. Having decided upon blood lead concentration as the measure of a strategy's effectiveness, it is then necessary in this study, to relate the primary effect of the strategies, reductions in airborne lead concentrations, to the secondary effect, the subsequent reductions in blood lead concentration. In this instance this has been achieved by developing a mathematical model relating lead exposure to blood lead level,

for a standard man. The necessity for a standard man approach, as explained earlier, arises because the distribution of blood lead levels through the population is not known[7]. As will be explained further in Section 5, a standard man has to be used due to a lack of experimental data regarding female blood lead concentrations[7,8]. Thus the effect that the control strategies have on the airborne component of the standard man's lead exposure may be related to his blood lead concentration and hence the effectiveness of the strategy may be calculated. Returning to the example strategy previously outlined, the elimination of lead additives from gasoline, CEA illustrates this latter point. (This strategy will be analysed in full detail in Sections 6,7 and 8).

1) Refinery capital costs for the installation of new plant to maintain gasoline pool octane quality.
2) Refinery production costs to maintain gasoline pool octane quality, per annum for ever.
3) Increased refinery energy consumption costs, p.a. for ever.
4) Increased maintenance costs to vehicle owners over 10 years until vehicle fleet totally renewed.

For those of the above costs which are found to have a significant value, the NPV would be calculated which represents the present-time cost of the strategy. With respect to the effect of the strategy, the total elimination of lead additives from gasoline would lead to a 90%+ reduction in airborne lead concentrations, since 90%+ of lead in the atmosphere originates from the combustion of leaded fuels. This effect is applied to

the airborne lead input component of the model relating lead exposure to blood lead concentration and the effect of the strategy on blood lead is calculated. The development of this model will be discussed fully in Section 5. From these two values, the strategy costs and effectiveness, the cost-effectiveness of the strategy may be found, i.e. the reduction in blood lead per £1.

CEA would therefore appear to be an acceptable technique to obtain useful information for the policy-maker regarding control strategies for environmental lead. It has one further advantage over CBA, relating to the use of modelling to calculate the strategy's effectiveness. The cost-effective ranking produced by this approach is a relative, as opposed to an absolute value, as mentioned in Sections 3 and 5. Hence, although the use of standard man parameters may be argued as leading to a degree of 'unreality' regarding the effects of the strategies, it is assumed that the cost-effective ranking will be similar to that produced if the exact model parameters for a given population were known. Hence, it was therefore decided to employ Cost-Effective Analysis as the technique for obtaining information regarding the strategies under consideration in this study.

Having selected a technique for their comparison, the following strategies were selected for examination in the context of this study as described earlier:

1) The elimination of lead antiknock additives from gasoline.

2) The reduction of the lead antiknock concentration

from 0.45 gPb/l to 0.40 gPb/l.

3) The reduction of the lead antiknock concentration from 0.45 gPb/l to 0.15 gPb/l.

4) The fitting of lead particulate traps to new and used, motor cars' exhaust systems.

5) The reduction of the lead antiknock concentration from 0.45 gPb/l to 0.40 gPb/l combined with the fitting of lead particulate traps to new, and used, motor cars' exhaust systems.

6) The reduction of the lead antiknock concentration from 0.45 gPb/l to 0.15 gPb/l combined with the fitting of lead particulate traps to the exhaust systems of new and used motor cars.

Having defined the strategies which are to be considered in this study, it appears valuable to give a brief outline of their background and their approach to the problem of lead control in the environment. Full details of the technology and costs will be given in Sections 6 and 7 respectively. Figure 4-1 shows the Refinery - Vehicle system within the Environment, and within whose bounds the technical aspects of all the above strategies lie. The following brief discussion of the strategies outlined above refer to this system as it includes all those factors considered to be of importance in the operation of the control strategies.

The first strategy, the total elimination of lead additives from gasoline, is the one favoured by environmentalist groups such as the Campaign Against Lead in Petrol

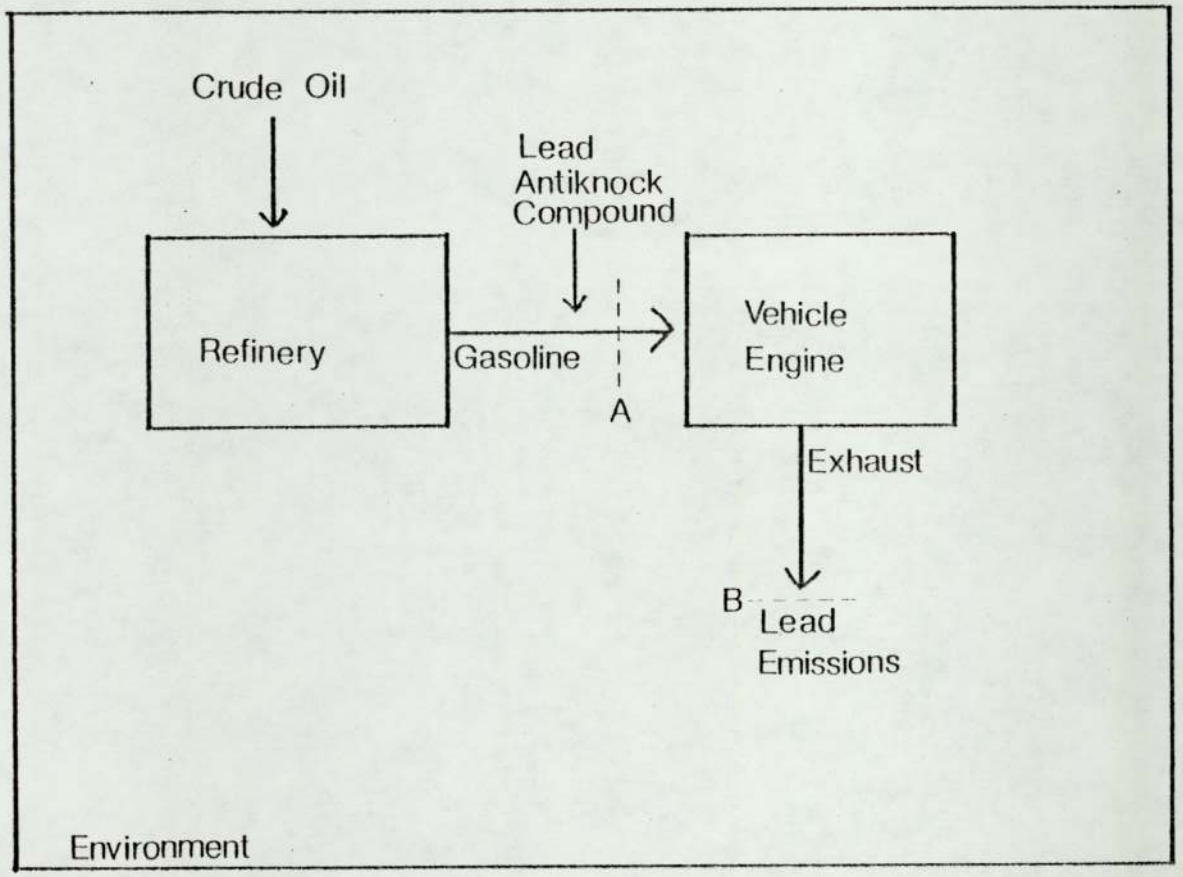

Figure 4-1

Strategies 1,2&3 – A
" 4 – B
" 5&6 – A+B

(C.A.L.I.P.)[9], and to a certain extent by the refining industry. The former group's support is understandable; they argue that elimination of lead additives from petrol would lead to a 90%+ reduction in airborne lead concentrations. The oil industry's support, albeit muted, is for somewhat different reasons. All petrol is refined lead free and at that point has an octane quality equivalent overall to 2-STAR, regular grade. Then lead antiknock additives are employed to raise the octane quality to that of 4-STAR. In fact, as refinery operations are optimised to demand constraints, lead additives are added to all grades, the concentration being dependent on the original batch of crude oil. Hence, if the total gasoline demand could be satisfied as lead free 2-STAR there would be a reduction in production costs to the refiners as they would not have to purchase lead antiknocks, or alter their plant. Thus refiners find a lead free strategy more acceptable than a low lead strategy to be discussed shortly. As yet there is no need for the U.K. to produce lead free gasoline in order to satisfy the demands of emission control devices as was the case in the U.S., discussed in Section 3. Naturally, if current octane quality is to be maintained, the refiners would not wish to follow a lead free option, as this would involve increases in capital investment and production costs.

The second strategy is due to be brought about by EEC legislation in 1980[10]. It is evidently a relatively small change and should not require any large changes in refinery plant. It will, however, necessitate an increase in crude oil consumption. This latter cost may well be recovered by

by improvements in engine design leading to greater fuel economy.

This option, the reduction of the lead concentration in gasoline to 0.15 gPb/l has already been adopted by W. Germany[11] and will be adopted by Sweden in the years 1980-81[12]. This level was chosen because it is the lowest possible which avoids the value seat recession problems associated with operation on lead free fuel. If the current distribution of grade/octane quality is to be maintained, this is an expensive option requiring large capital investment in refinery equipment and increased production costs.

An alternative approach to reducing the quantities of lead in gasoline is to prevent the lead escaping from the vehicles exhaust system to the environment. Naturally this approach is favoured by the manufacturers of lead antiknock compounds and considerable research has been carried out by two major producers, the Associated Octel Company Limited[13] and the Ethyl Corporation[14], the former in the U.K. and the latter in the U.S., into the effectiveness of lead traps. Perhaps not surprisingly both companies find that traps can produce similar reductions in lead emissions as lead reduction strategies, at much lower costs. A variety of trap designs have been studied all of which involve modification of the vehicles exhaust system. The traps themselves consist of a chamber containing steel wool which has been coated to improve its trapping properties. A number of coatings have been tested, along with various devices to agglomerate the lead aerosol before it reaches the trap, the traps being most effective for larger particle sizes. This strategy, then,

forms the fourth option under consideration.

The fifth and sixth strategies are compound approaches involving both reductions in lead antiknock concentrations in gasoline and the fitting of lead traps. These may be attractive in the light of the fact that they may achieve the effect of lead free gasoline without incurring the severe economic penalty of this option.

Having briefly reviewed the strategies under consideration, they will be assessed in detail in Sections 6 and 7, it should be noted that all lend themselves to analysis by CEA. In every case the costs may be obtained in economic terms alone, i.e. they may be expressed as monetary units. Having thus assessed the costs, the effectiveness will then be obtained via the model developed to represent the relationship between lead exposure and blood lead concentration. This will be a two stage operation. Firstly the effect the strategy would have on airborne lead concentrations is obtained and then this is related to an overall index of exposure, in this case blood lead, via the model. Thus, a cost-effective ranking may be obtained for all strategies, although this itself may have to be examined separately in the light of the effects of possible externalities, e.g. government policy, in Section 8.

Returning to Figure 4-1, the points at which the various strategies take effect are shown at A and B. Those strategies which take effect at point A, 1,2 and 3, reduce environmental lead levels by eliminating, or reducing, lead from the source, gasoline, whilst those which take effect at B, 4, operate by preventing the escape of lead from the engine's exhaust system. Further strategies, 5 and 6, offer combinations of

the above techniques. As can be seen from the figure, the desired objective of reducing environmental lead concentrations may be achieved by either of the above approaches; either eliminating lead from the combustion process or by preventing its escape from the vehicles exhaust system.

However, the cost-effective approach does not provide information on areas which some parties, e.g. C.A.L.I.P., consider of marked importance. For example the costs are not related in any way to the reasons for the elimination of lead from gasoline. Evidence has been put forward to suggest that exposure, at environmental levels, to lead may be more harmful than previously thought, particularly to alleged risk groups within society. A frequently cited example of a risk group is pre-school children[7]. Their exposure to lead, it is suggested, comes not only from the atmosphere and the diet but also as a result of behavioural factors such as finger licking and outdoor play activity. Here, lead in dust, from fallout airborne lead emitted from motor vehicles, increases the child's overall lead exposure[15]. Furthermore, once at school, the child may be exposed to increased levels of airborne lead in playgrounds situated near busy traffic routes. When this increased exposure is coupled with the possible higher sensity of children to lead, it may be argued that action should be taken to eliminate this exposure whatever the cost, because of the possible effects of lead on children; e.g. reduced I.Q., reduced learning ability, hyper-activity and antisocial behaviour. However, there is no unequivocal medical or scientific evidence for the above effects and thus the policy-maker is left with the task of making a decision based on circumstantial evidence.

Scientific testing of this evidence is extremely difficult, there being only marginal correlation between alleged cause and effect[16], and thus the decision is taken in a state of ignorance with respect to its long-term possible benefits. Until the results of further research become available this state of affairs will continue and the policy-maker will have to decide on whether, or not, to act on an intuitive basis, if he considers the risk of harm being proven in the future to be too great. This "insurance policy" approach is the only one viable at present, and if adopted a technique will be required for the comparison of various options. Cost-Effective Analysis, would appear to be the only choice, because it provides the decision-maker with a cost-effective ranking of those options under consideration to which he may apply further, external criteria if necessary.

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SECTION 5

A Review of Existing Models of Human Lead Metabolism and the Development of the TPU Model

As described in Section 4 the use of a model may be of considerable benefit in the area of policy making. In the light of these advantages it was decided to develop a model of the relationship between blood lead concentration and exposure to environmental lead for a standard man . A standard man was chosen despite the fact that the group at the highest risk from environmental lead is believed to be pre-school children, because there is a large amount of experimental data available for male subjects, as well as a number of standard man data bases for physiological parameters; e.g. the ICRP reports of 1959[1] and 1975[2]. The amount of epidemiological data available for men is greater than that available for women and children, and there is also the possibility that women[3] and children both metabolise lead in a different manner from men.

A number of models are reported in the literature all of which fall into one or other of two groups, which may be designated statistical and simulation. Although both groups attempt to relate lead exposure to some index of that exposure in man, usually blood lead concentration, there are fundamental differences in the approach of the two groups to the problem. These differences are brought about by the methodology used to develop each type of model, and are reflected in the manner in which each type may be tested, as outlined in the following paragraphs.

Statistical models are defined as those developed by regression analysis of epidemiological or experimental data on lead exposure and the exposure index, usually blood lead as mentioned above. The regression analysis produces a function, e.g. linear, logarithmic, semi-logarithmic etc. as chosen by the modeller, and finds the slope and intercept of this function.

Provided that there are no statistical arguments against the model, e.g. too small a population size to provide a reasonable epidemiological study etc., one is then faced with the problem of how well the model reflects the relationship between lead exposure and blood lead. The correlation coefficient of the regression line will indicate how well the form of the line, e.g. linear, fits the data from which the slope and intercept were developed, but it will not give any indication of the accuracy of the data. This latter point arises from the technical difficulties associated with blood and air lead assays. Both of these values have been found to be subject to a high degree of experimental error on a number of occasions. Furthermore, if a large study has been carried out and a number of laboratories commissioned to carry out the analyses, inter-laboratory variation will further aggravate the problem of experimental accuracy. Thus it is possible to conceive of a statistical model, e.g. semi-logarithmic, with a high correlation coefficient, e.g. 0.75+, which is totally meaningless because the data from which it was developed had a high degree of experimental error. Assuming difficulties with the experimental analyses have been overcome, the statistical model has still to be tested for its general applicability. In a perfect situation, not only the form of the model but the slope and intercept as well should apply to any and every data set. However, the slope and intercept contain several parameters merged together which vary from one individual to another, and, more importantly from the statistical model's standpoint, from one population group to another. These parameters are mainly physiological, e.g. lung retention and absorption of lead, pulmonary ventilation, blood lead elimination, gastro-intestinal absorption of lead

etc., but may include others such as dietary lead exposure, for those models which simply relate air and blood lead. Hence it is unlikely that the actual function developed from the first set of data will give a good fit for subsequent data due to parameter variation between the two populations from which the data was obtained. However, the form of the model, if the model is a good representation of reality, should fit any data set, with certain reservations. Thus a linear relationship may be tested against lead exposure and blood lead for a number of populations to examine whether a linear model is a valid relationship. The reservations mentioned above include; populations used should be similar with respect to age, sex, type of geographic location, dietary habits and social class. The populations should also be of a similar size. The greater the variation, with respect to the above factors, from the population which originally suggested the form of the model, the greater the probability that the model will not provide a good fit for the latter data. Finally, as with all regression analysis, interpolation within the spread of the original data is reasonable, but extrapolation beyond that data must be carried out with extreme caution. Nevertheless a proven statistical model would provide useful information regarding the effects of lead exposure.

Many of the problems associated with statistical models are overcome by the second type, simulation models. In this case the model represents the metabolism of lead in a human being; i.e. the flow of lead from one body location, or compartment, to another. Most simulation models contain compartments for blood, bone and soft tissue, although others suggest further compartments, e.g. for blood plasma and the lymphatic

system. Experimental work on human subjects is used to identify values for parameters such as lung lead retention, gastro-intestinal lead absorption and excretion, lead elimination rates from the various compartments, pulmonary ventilation, blood volume etc. Values for these parameters for a standard man have been developed by the International Commission for Radiological Protection (ICRP). Thus, for a given input from airborne and dietary lead, the metabolism of lead in the various body compartments, under both dynamic and equilibrium conditions, may be charted. The models can be tested against epidemiological data, the model having been calibrated for the population involved with the epidemiological study, the standard man model may be tested against such data to examine how well it fits the real situation, and the model may undergo iterative improvement in the light of new information regarding any of the above parameters. Furthermore, assuming the accuracy of the model's parameters, a simulation model provides some indication of the accuracy of the epidemiological results by how well the model fits the data. Unlike its statistical counterpart, the simulation model may be calibrated for small population groups once the appropriate parameter values have been determined, this being particularly important where risk groups are concerned, e.g. occupationally exposed lead workers. With regard to the iterative improvement of the model, key parameters may be identified, i.e. those to which the model is most sensitive, and further research carried out to obtain their values. Similar steps may be taken as a result of testing the model against available experimental data concerning lead exposure and blood lead. These latter points cannot be applied to statistical models as all the

various parameters involved are combined in the slope and intercept of the function.

In conclusion, although the simulation model is more difficult to develop, it is more useful than the statistical model because it inherently provides more information, and is therefore more testable, regarding lead metabolism, and failure of the model generally indicates where it might be improved. Finally it may be more 'finely tuned' to any given lead exposure situation.

Goldsmith and Hexter, 1967[4], developed a logarithmic dose-response relationship from the results of the Three-City Study[5] in order to predict the higher blood lead levels of populations exposed to long-term increases in atmospheric lead. Table 5-1 shows the data they employed. They suggested the relationship might be represented by:

$$\log_{10} (\text{Blood lead}) = m \log_{10} (\text{Air lead}) + c$$

The data in table 5-1 provides values of m and c of 0.24 and 1.27 respectively with a correlation coefficient of 0.85. The output of this model is shown in Table 5-2, and Figure 5-1.

However, it is immediately obvious that the function is not defined for a zero air lead value whereas it is known that, even if the air lead is zero, there will still be a blood lead produced from dietary lead exposure. A number of other criticisms may be levelled at the Goldsmith-Hexter model. They state, "For populations with known occupational exposures, the average is a weighted average of presumed occupational and ambient exposure. Ambient exposures are estimated only and were not necessarily measured at the same times and places at which the populations were exposed". The net outcome of

Table 5-1

Type of Population	Estimated Exposure ug/m^3			Mean Blood lead ug/100 ml	
	Occupational	Ambient	Average	Male	Female
Populations without known occupational exposure					
Remote California Mountain Residents		0.12		12	9
Composite rural U.S.		0.5		16	10
Suburban Philadelphia		1.0		13	13
Composite urban U.S.		1.0		21	16
L.A. aircraft workers		1.9		19	17
Pasadena city employees		2.2		19	12
Downtown Philadelphia		2.4		24	18
Populations with known occupational exposures					
Cincinnati policemen (all)	4.7	1.4	2.1	25	
Cincinnati traffic policemen	12.8	1.4	3.8	30	
Cincinnati auto test lane inspectors	14.8	1.4	4.2	31	
L.A. traffic policemen	16.5	2.2	5.2	21	
Cincinnati garage workers	21.1	1.4	5.5	31	
Boston Summer Tunnel workers	44.5	1.1	6.3	30	

Table 5-2

Airborne lead Concentration ug/m^3	Blood lead Concentration ug/100 ml
0.00	Not Defined
0.25	13.35
0.50	15.77
0.75	17.38
1.00	18.62
1.25	19.65
1.50	20.52
1.75	21.30
2.00	22.00
2.25	22.62
2.50	23.20
2.75	23.74
3.00	24.24

Goldsmith – Hexter Model

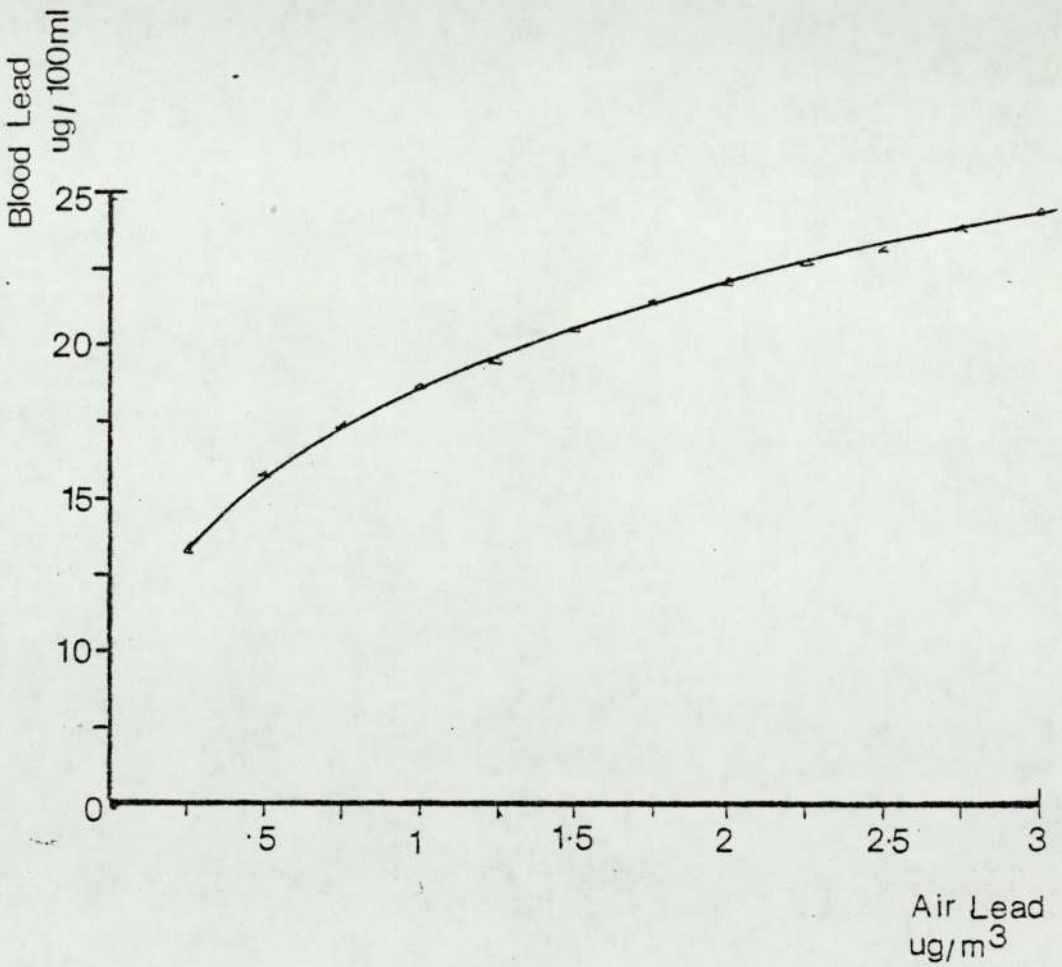

Figure 5-1

this statement is that 46% of their sample population are effectively estimations open to considerable doubt. This in turn throws doubt on the logarithmic regression line and this point formed the basis of the Ethyl Corporation's[6] criticism of the Goldsmith-Hexter model, referred to in Section 3. This latter body also cited a second measurement of male blood lead levels for remote Californian Mountain residents of 20 ug Pb/100 ml blood. The U.S. Environmental Protection Agency (EPA) obtained their average, minimum, acceptable air lead concentration of 2 ug/m^3[7] from this model by finding the lowest air lead value which produced a change in blood lead level, statistically, significantly different from zero. However, if the Ethyl Corporation's value of 20 ug/100 ml is employed, the regression so produced was shown not to be significantly different from zero up to the highest air lead level found, namely 6.3 ug/m^3. Thus, Ethyl argued, the 2.0 ug/m^3 level was completely dependant on the first data point, further casting doubt on the Goldsmith-Hexter model. Goldsmith and Hexter also state, "Such variations as are introduced by dietary fluctuations apparently are averaged out by taking adequately large populations". However, their model takes no account of the contribution of this averaged out value to blood lead and it cannot be found from the model as it is not defined for a zero air lead. In the light of these criticisms the Goldsmith-Hexter model would appear to provide little useful information regarding the relationship between blood lead level and lead exposure. (See Appendix II).

A second statistical model was developed by the National Academy of Sciences in their 1972 Report on Atmospheric lead[8].

The NAS proposed a semi-logarithmic model in order to predict the blood leads of populations exposed to a given air lead concentration. They assumed that the average American diet contributed 300 ug/day of lead, of which 10% was absorbed by a reference man. The use of the reference man concept was extended to include pulmonary ventilation, 23 m^3/day and lung retention of the airborne lead aerosol, 30-37%. The NAS model was of the form:

Blood lead = m log (Pb absorbed daily) + c

Using the parameters outlined above, the NAS carried out regression analysis on the Three-City Study data, as employed by Goldsmith and Hexter, to which they added two additional points shown in Table 5-3. Taking the 30% value for lung lead retention, the NAS produced the following model:

Blood lead = m log (30 + 6.9(Air lead)) + c

with values of 54.76 and -69.21 for m and c respectively, and a correlation coefficient of 0.961. However, if the values in Table 5-3 are deleted, as they are the product of artificially high dietary lead levels, the correlation coefficient drops to 0.835, similar to that of Goldsmith and Hexter. The values of m and c also alter to 45.63 and -54.05 respectively. The output of this model is shown in Table 5-4 and Figure 5-2. In this case, however, it should again be noted that for a zero lead exposure, the function is not defined. Also, for a zero air lead and dietary lead levels of 180 ug Pb/day, negative blood leads are output by the model. (The same holds true of a unitary air lead and dietary leads below 110 ug Pb/day). In the light of this evidence one must conclude that the NAS model may only be used over a limited range of lead exposures and cannot be considered generally applicable. To these criticisms

Table 5-3

Blood lead ug/100 ml	Air lead ug/m^3	Total Daily lead Assimilation ug
[a]48	2.0	148.8
[b]63	2.0	221.3

Daily oral lead intake for:

a) 1350 ug/day

b(2075 ug/day

Table 5-4

Air lead Concentration ug/m^3	Blood lead Concentration ug/100 ml
0.00	11.68
0.25	13.01
0.50	14.27
0.75	15.46
1.00	16.60
1.25	17.69
1.50	18.73
1.75	19.72
2.00	20.68
2.25	21.60
2.50	22.48
2.75	23.33
3.00	24.16

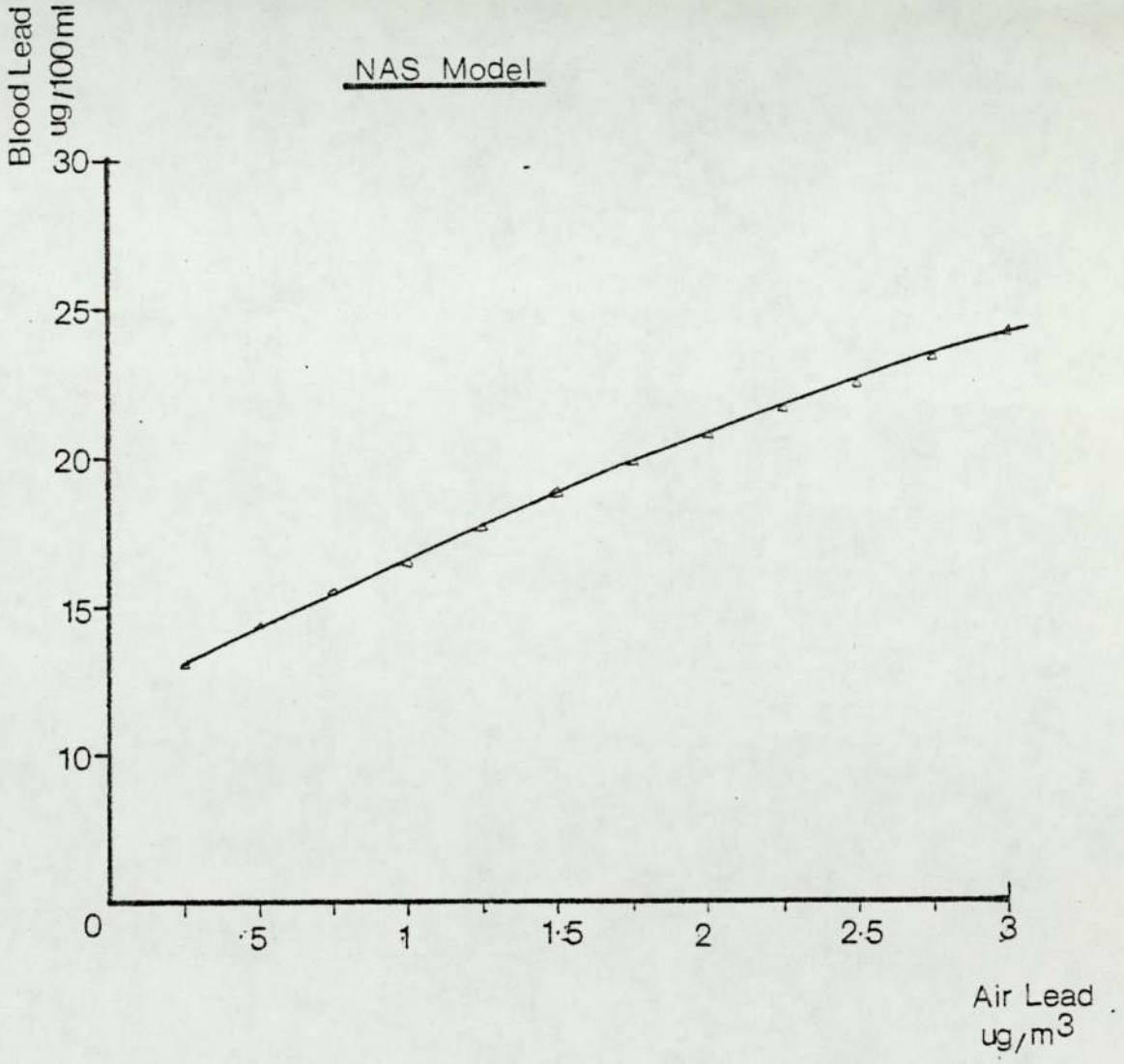
NAS Model
Blood Lead
ug/100ml
30
25
20
15
10
0
·5
1
1·5
2
2·5
3
Air Lead
ug/m3

Figure 5-2

must be added the fact that the NAS employed the Three-City Study data which is open to doubt on a number of points, described previously. The data shown in Table 5-3 is the product of abnormally high levels of dietary lead and cannot be considered representative of the environmental exposure represented by the remaining data employed, shown in Table 5-1, and it does not seem consistent to merge these two data sets. The obvious improvement of the NAS model over that of Goldsmith and Hexter is the inclusion of a term relating to dietary lead exposure, but overall the model appears to be of limited value. (See Appendix II).

A further statistical model was described by Knelson et al., 1972[9]. In this case, adult male volunteers were exposed to a lead aerosol of 10.9 ug/m^3 and 3.2 ug/m^3 for periods of up to eighteen weeks. These men were in fact prisoners in a U.S. Penitentiary. Initial measurements of the mens' blood leads indicated a mean base-line blood lead level of 19.6 ug Pb/100 ml blood. The volunteers' blood leads were periodically measured and the mean increases in blood lead were calculated. Table 5-5 shows the notation used, and the Knelson model. The object of this model was to predict the increase in blood lead brought about by exposure to an air lead elevated above the subjects' normal level. Experimental values for IBL and BB, for each of the two exposure levels, were measured over the time period and the equations shown in Table 5-5 produced by regression analysis. These equations represented the extreme points of an exposure continuum and in order to calculate increases in blood lead concentration occurring as a result of intermediate exposure levels, the model shown in Table 5-5 was

Table 5-5

IBL, ug/100 ml	Predicted rise in blood lead concentration
BB, mg	Potential rise in total body burden of lead
L, ug/m^3	Air lead concentration
V, m^3/day	Pulmonary Ventilation
R	Fraction of inhaled lead retained in the lung
D, days	Duration of exposure

Values assigned to the parameters were:

$V = 15\ m^3/day$

$R = 37\%$

Knelson et al. employed a blood volume V = 4.9 litres.

The subscripts H and L refer to the high and low exposure groups respectively.

$$BB = L \cdot V \cdot R \cdot D \cdot 10^{-3}$$

$$IBL_H = m_H + b_H \log_{10} BB$$

$$IBL_L = m_L + b_L \log_{10} BB$$

$$IBL_H = 6.80318 + 10.8101 \log_{10} BB$$

$$IBL_L = 4.283 + 3.774 \log_{10} BB$$

$$m = m_H - m_L$$

$$b = b_H - b_L$$

$$L_H = 10.9\ ug/m^3$$

$$L_L = 3.2\ ug/m^3$$

$$L = L_H - L_L$$

Table 5-5 cont.

Knelson Model

$$IBL = \frac{L - L_L}{L} \quad m + m_L + \left(\frac{L - L_L}{L} + b_L \right) \log_{10} BB$$

Simplifying and Substituting:

$$IBL = 0.327L + 3.236 + 0.914L + 0.85 \log_{10} BB$$

constructed. Obviously if a zero value of BB exists the model is undefined, but this criticism does not apply as BB will never be zero as the model is designed to predict the response to an increase in air lead concentration over time; i.e. BB would be zero if there was no increase, but then the model would not be used. Also the fact that the dietary contribution to blood lead is not taken into account is irrelevant as the model is designed to predict the increase in blood lead due solely to an increase in air lead concentration, although for the results to be valid it must be assumed that the dietary lead intake remains constant over the time period being measured. If this is not the case, the sum of the base-level blood lead and IBL will not be the actual blood lead measured at the end of the time period. Knelson's experimental work showed that towards the end of the exposure about 10% of the potential increase in body burden could be accounted for by increased total blood lead content. This agrees well with the findings of Schroeder and Tipton, 1968[10], that, as equilibrium is approached, 90% of the total body burden of lead is contained in the skeleton. However, the study is open to two major criticisms. Firstly how well prisoners inhaling a manufactured lead aerosol represent the average male inhaling environmental lead levels remains open to question. Secondly, both the above levels of airborne lead are higher than those normally found in the environment. The slope and intercept of the function are dependent on the air and blood lead measurements, and the intermediate function produced, Knelsons' Model, would possibly be inapplicable outside the 3.2. - 10.9 ug/m^3 range. For the U.K., a range of 0.5 - 3.0 ug/m^3 would be more appropriate

in which case the range of IBL measured experimentally would be so small as to make valid regression analysis, to produce the intermediate function, impossible. It is therefore difficult to apply this model to environmental levels of airborne lead exposure which are generally low, and usually constant, giving no increase to measure. However, it could be used if a new source of airborne lead was to appear in the environment; e.g. to predict possible increases in the blood leads of a population living near to a proposed lead smelter or motorway.

Although statistical models are usually developed from one set of experimental data, their designers suggest that the form of the model, e.g. log-log, semi-log or linear, should apply to all epidemiological data. It is most unlikely that the values for the slope and intercept developed from one data set would apply to another, unless both sets were from the same, or very similar, populations. The scarcity of large-scale epidemiological studies make extensive testing of statistical models difficult and small-scale studies do not average out individual variation in physiological parameters, such as lung retention of lead aerosols or G-I tract absorbtion of lead, to permit the valid use of a statistical model. Experimental error in the analysis of the epidemiological data may affect a statistical model. Although the form of the model remains unchanged, the model itself, its slope and intercept, are dependent on the data set. For example, it is possible to conceive of a totally erroneous study whose results lead to the development of a model predicting decreasing blood leads with increasing air leads. Thus it can be seen that the

'goodness' of a statistical model depends upon the experimental accuracy of the data on which it is based; i.e. a statistically good model may possibly not be a very good representation of the relationship between exposure to lead and blood lead concentration. To this problem are added all the difficulties inherent in a large-scale epidemiological study. Blood and air lead values are technically difficult to obtain, especially at the low levels encountered in the environment, unless scrupulous laboratory techniques are rigidly adhered to. Also, in a large study, it is usual to employ the services of more than one laboratory for the blood and air lead assays in which case the problem of inter-laboratory variation arises; i.e. the same sample sent to two different laboratories produces two different results. With regard to air lead sampling, before the introduction of personal air sample the air lead was measured for an area, perhaps several square miles, and everyone in that area is assumed to be exposed to that air lead level. Detailed sampling has shown considerable variation within a given area[11] and so this technique is open to some criticism. Naturally the size of the sample population is also important, because if it is too small the results may not be statistically applicable to the population generally. Hence, taking these difficulties into account, one may conclude that statistical models are relatively simple to construct but there are difficulties associated with their general application.

Simulation models provide a more acceptable solution to the question of the relationship between lead exposure and blood lead concentration. This occurs for two main reasons; firstly,

as mentioned earlier, simulation models are more testable than their statistical counterparts as they may employ both epidemiological studies and the results of laboratory work for their validation and secondly they are open to iterative improvement in the light of on-going research in any of the fields associated with their parameters, e.g. the elimination of lead from the blood stream. For statistical models the various physiological parameter associated with a simulation model are combined, irretrievably, in the slope and intercept. With regard to the physiological parameters employed in a simulation model, various standard man values have been derived over a considerable period of time, notably those described in the various ICRP reports. These are of course open to criticism but at least provide a starting point for the calibration of a simulation model.

Lutz et al., 1970[12], suggested a first order dynamic model which enabled the total body burden of lead to be calculated, for various lead inputs along with its distribution in the blood, bone, liver, kidney, and lung. Table 5-6 shows the notation and model developed by Lutz and his co-workers. The first point to note is that Lutz fails to define one of the terms employed, namley G. An intelligent guess would suggest that it might be the remaining fraction of soluble lead, i.e. 1-E, but there can be no certainty regarding this. Later in his paper it would also appear that the terms representing soluble and insoluble airborne lead, C_{AS} and C_{AI}, are combined into a single value, C_A, although this is not explicitly stated. Lutz considers his model to be deterministic, i.e. mechanisms relating the various components are suggested and appropriate mathematical functions are derived to represent

Table 5-6

Notation

Suffixes:

l	- lung	b	- bone
bl	- blood	li	- liver
tb	- total body	k	- kidney

$\dot{Y}_{suffix}$	Derivative with respect to time of the lead quantity at the suffix location in ug/day.
Y_{suffix}	Quantity of lead at time t at the suffix location in ug.
C_{AI}	Concentration of airborne, insoluble lead compounds in ug/cm^3.
C_{AS}	Concentration of airborne, soluble lead compounds in ug/cm^3.
B	Pulmonary ventilation in cm^3/day.
D	Fraction of insoluble lead retained in the lung.
E	- ditto - soluble lead
F	Fraction of ingested lead which reaches the blood.
G	Not defined in Lutz's paper.
λ_{suffix}	Elimination rate coefficient for lead from the suffix location in $days^{-1}$.
$(F_2)_{suffix}$	Fraction of lead transferred from the blood to the suffix location.
$(F_a)_{suffix}$	Fraction of inhaled lead reaching the suffix location.
$(F_w)_{suffix}$	Fraction of ingested lead reaching the suffix location.

Table 5-6 continued

$$\dot{Y}_1 = DBC_{AI} - \lambda_1 Y_1$$

$$\dot{Y}_{b1} = (F_1E + G)\, BC_{AS} + P_1FBC_{AI} + F_1 \sum_{n=1}^{N} I_N C_N + \lambda_1 Y_1 - \lambda_{b1} Y_{b1}$$

$$\dot{Y}_{tb} = (Fw)_{tb} \left(\sum_{n=1}^{N} I_n C_n + FBC_{AI} \right) + (Fa)_{tb} BC_{AS} + (F_2)_{tb} \lambda_1 Y_1 - \lambda_{tb} Y_{tb}$$

$$\dot{Y}_b = (Fw)_b \left(\sum_{n=1}^{N} I_n C_n + FBC_{AI} \right) + (Fa)_b BC_{AS} + (F_2)_b \lambda_1 Y_1 - \lambda_b Y_b$$

$$\dot{Y}_{li} = (Fw)_{li} \left(\sum_{n=1}^{N} I_n C_n + FBC_{AI} \right) + (Fa)_{li} BC_{AS} + (F_2)_{li} \lambda_1 Y_1 - \lambda_{li} Y_{li}$$

$$\dot{Y}_k = (Fw)_k \left(\sum_{n=1}^{N} I_n C_n + FBC_{AI} \right) + (Fa)_k BC_{AS} + (F_2)_k \lambda_1 Y_1 - \lambda_k Y_k$$

them. Thus the Lutz model was developed from a purely hypothetical basis. Further examination revealed that the source of reference cited by Lutz for all the other parameters, the 1959 ICRP report[1], failed to provide values for D and λ_{bl}. Lutz stated that a value for λ_{bl} was obtained by least squares regression analysis of reported experimental data on blood lead levels as a function of inhaled and ingested lead but did not reference this data. However, an estimation of the values employed may be obtained using the following approach and the results of the model as given by Lutz, shown in Table 5-7.

In dynamic equilibrium conditions:

$$\dot{Y}_l = \dot{Y}_{bl} = \dot{Y}_{tb} = \dot{Y}_b = \dot{Y}_{li} = \dot{Y}_k = 0$$

Hence, from Table 5-6,

$$\lambda_l Y_l = DBC_{AI}$$

Substituting, again from Table 5-6,

$$0 = (F_l E + G) BC_{AS} + F_l FBC_{AI} + F_l \sum_{n=1}^{N} I_n C_n + DBC_{AI} - \lambda_{bl} Y_{bl}$$

It is reasonable to assume that under normal environmental conditions the value of C_{AS} will tend to zero. Thus:

$$0 = F_l FBC_{AI} + F_l \sum_{n=1}^{N} I_n C_n + DBC_{AI} - \lambda_{bl} Y_{bl}$$

Substituting Lutz's results, from Table 5-7, this latter equation may be solved twice, as a pair of simultaneous equations in two unknowns. This gives values for λ_{bl} and D of 0.03 and 16% respectively. The value for λ_{bl} agrees with other values described in the literature[13,14,15,16,20,24], but a lung lead-retention factor of 16% is roughly half what one would have expected. This parameter is normally quoted as having a value between 30% and 40%. The Lutz model sug-

Table 5-7

Estimated average respiratory exposure ug/m^3	Measured Mean Blood lead level ug/100 ml	Predicted Blood lead level ug/100 ml
0.5	16	16.5
1.0	21	17.6

The first two columns of this table are taken from the Three-City Study. Lutz assumed a dietary intake of 300 ug Pb/day with a 10% absorption factor, i.e. 30 ug Pb/day.

gests a rise in blood lead of 2.2 ug Pb/100 ml blood in response to a 1 ug/m^3 rise in the airborne lead concentration. However, as Lutz validates, and if he calibrated, his model against the results of the Three-City Study[5], as used by Goldsmith and Hexter[4] and the NAS[8], the discrepancy in the lung lead-retention factor may arise from the doubtful accuracy of this data.

A similar approach was adopted by Robinowitz et al., 1973[13], although he employed a three compartment model as shown in Figure 5-3, as opposed to Lutz's six compartment design. At this time Robinowitz considered the bone to be a time-independent compartment and a sink for lead from the blood. His technique was to feed a healthy adult male a constant low lead diet whilst he lived in a metabolic unit for 160 days. For the first 104 days the diet was supplemented with lead-204 nitrate to increase the total dietary lead intake to the subject's approximate intake before the study. Then the lead-204 nitrate was replaced with lead-207 nitrate for 10 days. For the next 46 days the subject received only the low lead diet. The concentration and isotopic composition of lead were determined serially in the diet, faeces, blood, urine, facial hair and atmosphere by mass spectrometric isotope dilution analysis. From these results Rabinowitz concluded that two-thirds of the blood lead was dietary in origin whilst one-third was inhaled. The equations representing the kinetics of his model are shown in Table 5-8, along with the appropriate notation. This table refers to Figure 5-3. This first study considered one subject, but Rabinowitz later extended his work to two subjects, 1974[14], and five subjects, 1976[15] and 1977[16]. This latter work confirmed Rabinowitz's earlier conclusion regarding a 2:1

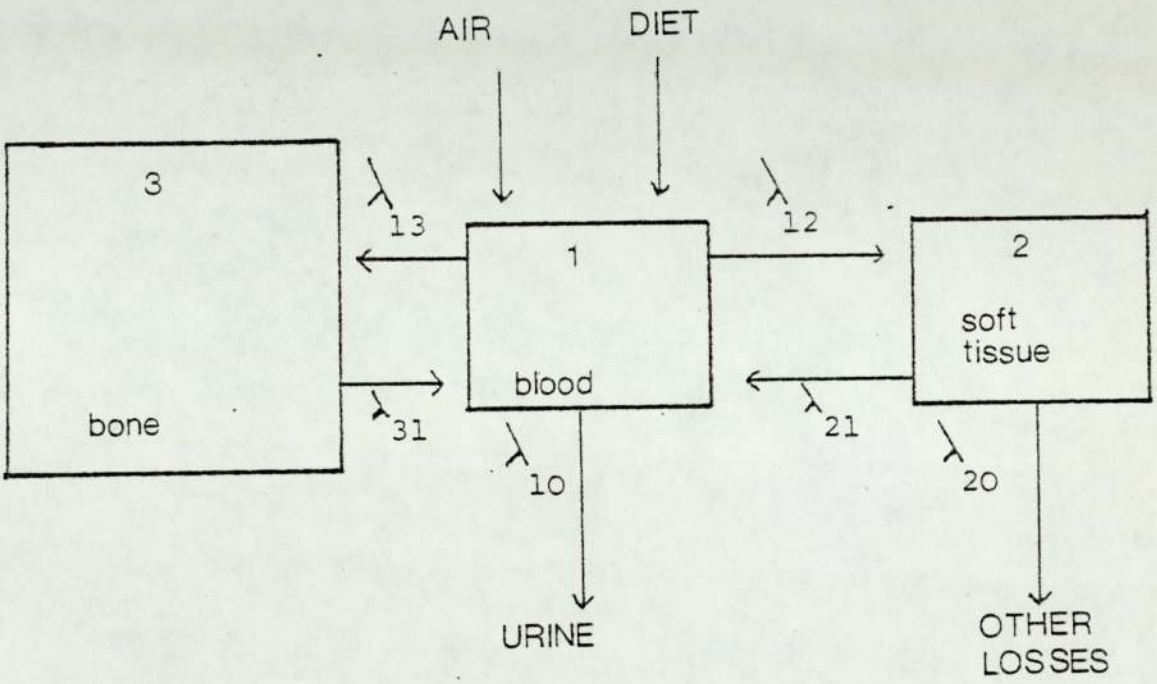

Figure 5-3

Rabinowitz et al., 1973

Table 5-8

$$\frac{dq_1}{dt} = \frac{A}{Q_1} - \lambda_1 q_1 + \lambda_{21} \frac{Q_2}{Q_1} q_2$$

$$\frac{dq_2}{dt} = \frac{Q1}{Q_2} \lambda_{12} q_1 - \lambda_2 q_2$$

Notation

q_i	Concentration of ^{204}Pb in compartment i . (ug/kg).
λ_{ij}	Exchange constant from compartment i to compartment j (days).
Q_i	Mass of compartment i (kg).
λ_i	Sum over j of λ_{ij} (days).
A	Quantity of ^{204}Pb absorbed daily from the gut (ug).

ratio between the contributions of ingested and inhaled lead to blood lead. A complete analysis of the Rabinowitz Three-Compartment Model is given in the DSc Thesis of Rowe, 1976[17], and although this refers to the 1974 work it may still be considered to give a full explanation of the model. Rowe also introduces the bone as an active compartment rather than as a time independent sink for blood lead. Table 5-9 shows the model and notation, slightly different from that of Rabinowitz, employed by Rowe, and refers to Figure 5-4. It was decided to look at this model under conditions of dynamic equilibrium as these best represent long-term exposure to environmental levels of lead. However at this point two anomalies occurred in the Rowe thesis. Firstly, Rowe employs what he defines as "Instantaneous, first-order exchange constants from compartment i to compartment j, k_{ij}" whose units are ug/day as shown in Table 5-9. This is in contrast to the more usual elimination rate constant, measured in $days^{-1}$, as used by Rabinowitz et al[14]. Rowe gives no further explanation of this term although his results appear to be dimensionally correct. Secondly, Rowe cites the following equation relating to bone, Compartment 3,:

$$\frac{dq_3}{dt} = K_{13} \frac{Q_1}{Q_3} - K_{31} q_3$$

Analysis from first principles would suggest the following equation:

$$\frac{dq_3}{dt} = K_{13} \frac{Q_1}{Q_3} q_1 - K_{31} q_3$$

It is quite possible, however, that this latter point arises from a misprint rather than from an error on Rowe's part.

Table 5-9

$$\frac{dq_1}{dt} = \frac{A}{Q_1} - (K_{10} + K_{12} + K_{13})\, q_1 + K_{21} \frac{Q_2}{Q_1} q_2 + K_{31} \frac{Q_3}{Q_1} q_3$$

$$\frac{dq_2}{dt} = K_{12} \frac{Q_1}{Q_2} q_1 - (K_{20} + K_{21})\, q_2$$

$$\frac{dq_3}{dt} = K_{13} \frac{Q_1}{Q_3} - K_{31}\, q_3$$

Notation:

A	Pb absorbed from gut and lungs, (ug/day).
q_i	Concentration of Pb in Compartment i, (ug/kg).
Q_i	Mass of Compartment i, (kg).
K_{ij}	Instantaneous, first-order exchange constants from Compartment i to Compartment j, (ug/day).

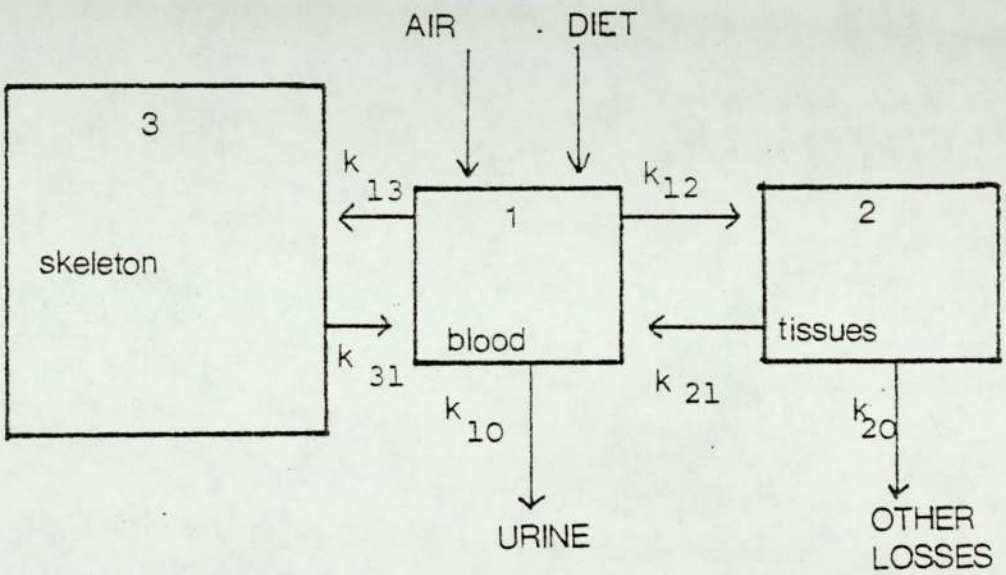

Figure 5-4

Rowe, 1976

Nevertheless, to avoid errors, the terms for Compartment 3 were ignored and the model was employed in the same way as by Rabinowitz with Compartment 3 being assumed time independent. The contribution of Compartment 3 to blood is extremely small and the model's output is not greatly affected. Thus for dynamic equilibrium conditions it may be said:

$$\frac{dq_1}{dt} = \frac{dq_2}{dt} = 0$$

Hence:

$$0 = \frac{A}{Q_1} - (K_{10} + K_{12} + K_{13})\, q_1 + K_{21} \frac{Q_2}{Q_1} q_2$$

$$0 = K_{12} \frac{Q_1}{Q_2} q_1 - (K_{20} + K_2)\, q_2$$

Substituting, the upper equation becomes:

$$0 = \frac{A}{Q_1} - (K_{10} + K_{12} + K_{13})\, q_1 + \frac{K_{12}\, K_{21}}{K_{20} + K_{21}} q_1$$

Rowe cites values for the various parameters taken from Rabinowitz and Koppel, 1974[14] and these are shown in Table 5-10, again referring to Figure 5-4. From these it is possible to calculate the blood lead concentration, in ug/kg for Compartment 1 and it is then an easy matter to convert this to a blood lead level expressed in ug Pb/100 ml blood. However, A is a complex term representing the daily absorbtion of lead from the lungs and gastro-intestinal tract. The values cited by Rabinowitz for lung and gut retention/absorption were 40% lung lead retention factor, with a pulmonary ventilation of 20 m^3/day, and between 6-9% gut absorption of lead. Substituting these values into the steady-state equation for

Table 5-10

Parameter	Subject A	Subject B
lead in Compartment 1 (ug)	1830 ± 75	1825 ± 55
lead in Comparment 2 (ug)	760 ± 100	900 ± 100
K_{10} (ug/day)	0.020 ± 0.002	0.015 ± 0.002
K_{12} (ug/day)	0.010 ± 0.003	0.006 ± 0.002
K_{13} (ug/day)	0.005 ± 0.005	0.003 ± 0.002
K_{21} (ug/day)	0.020 ± 0.010	0.0002 ± 0.0001
K_{20} (ug/day)	0.050 ± 0.020	0.030 ± 0.010

Q_1 Mass of Compartment, = 7.23 kg

A Total, daily Pb absorption. (ug/day)

The unites of K are those employed by Rowe[17], as opposed to Rabinowitz[14].

Compartment 1 produces a blood lead level of 34 ug Pb/100 ml blood, using the lead exposure suggested by Rabinowitz, 1974[14].

Taking the high lung retention factor coupled with the high dietary lead intake needed to produce the lead absorption shown, 50 ug Pb/day, this is an acceptable result. It should be noted that mean values for K_{ij}, taken from Table 5-10 were employed for the purposes of this calculation. When values suggested by more recent research[18,19] are substituted in the model, a blood lead level of 17.6 ug Pb/100 ml is produced for an exposure of 1 ug Pb/m^3 inhaled and 200 ug Pb/day ingested. This result is within the expected physiological range for the above exposure. A rise in airborne lead of 1 ug/m^3 produces a rise in blood lead of 5.5 ug pb/100 ml blood, using the original Rabinowitz parameters. This quite high rise may be accounted for by the high values suggested by Rabinowitz for pulmonary ventilation, lung lead-retention and daily lead intake from the diet, and obviously the model may be re-calibrated in the light of more recent research. Whereas the Lutz model was developed to provide the U.S. Environmental Health Service with a management tool, the chief interest of Rabinowitz appears to have been the simulation of human lead metabolism. The two models were developed from entirely different backgrounds, Lutz from pure theory and Rabinowitz from detailed laboratory work, and yet they both apply to the same subject - an adult male. Rowe re-calibrated the Rabinowitz model for children, but he did so in the light of epidemiological studies carried out on children and simply fitted the model to the data. Values for the model's parameters for children were therefore statistically, rather than experimentally, obtained. This emphasises the paucity of data for groups other than

adult males and hence the result that most simulation models are developed for adult males.

This holds true for Chamberlain, 1978[20], who suggests a possible scheme for the uptake and excretion of lead based upon his most recent work. Chamberlain followed 203-Pb through the body; its removal from the lung, its removal from blood and excretion, and also its uptake from the gut. From this work he concluded that, for the average urban dweller, 50% of the inhaled lead aerosol is retained in the lung, of which 55% is transferred to the blood, the remainder becoming attached to soft tissue or bone. This 50% retention factor is the highest cited in the literature, but is balanced to some extent by the adoption of a 15 m^3/day pulmonary ventilation and 55% lung-blood lead transfer coefficient. Absorption from the gut is stated to range from 7% to 12%. Chamberlain does not outline a specific model, but rather tabulates lead input and excretion related by transfer coefficients and concludes that a rise in airborne lead of 1 ug/m^3 would lead to a rise in blood lead level of 2 ug Pb/100 ml blood. Chamberlain criticises the Rabinowitz model on two counts. Firstly he argues that Compartment 1 should in fact be two compartments for blood plasma and red cells respectively, in the light of his 55% lung-blood transfer coefficient. However, this brings about a logical inconsistency. If only 55% of the lead absorbed through the lungs is transferred to the blood (red cells), why is the same factor not applied to lead absorbed through the gut? Chamberlain does not offer any explanation for this discrepancy. Chamberlain also finds that urinary and endogenous fecal excretion commence simultaneously whereas

Rabinowitz and his co-workers suggest that fecal excretion is delayed by about forty days with respect to urinary excretion, due to the fact that fecal excretion only occurs from Compartment 2. Although Chamberlain does not suggest a model, his proposed intake-excretion scheme implicitly requires a first-order dynamic mechanism similar to those of Lutz and Rabinowitz. The Lutz model was developed to provide a management tool, the Rabinowitz model to simulate lead metabolism but the work of Chamberlain is a combination of the two approaches. Although it is not explicitly suggested that the intake-excretion scheme be used to provide information for the decision-maker, it is included in a paper entitled, "Investigations into lead from Motor Vehicles" produced to provide information, presumably for the policy-maker, on the effects on humans of lead emitted from motor vehicles.

In conclusion the simulation model succeeds over its statistical counterpart by the nature of its construction. The model can be tested against the results of epidemiological studies, and at the same time each of the model's parameters may be tested against the results of laboratory work on the appropriate facet of lead metabolism, e.g. lead uptake from the gut. Furthermore, one may identify those parameters critical to the accuracy of the model, carry out further research in those areas, and subject the model to iterative improvement. Theoretically one should be able to apply the model to any population's lead exposure once it has been calibrated and suitable values for the various parameters found. This is a much more difficult, perhaps impossible task, for a statistical model as the many parameters involved are cascaded

together in the slope and intercept of the function. Thus the simulation model provides a more general, and more easily, applicable relationship between lead exposure and blood lead concentration than a statistical alternative.

In the light of these arguments it was decided to develop a simulation model to provide an index of the effectiveness of the strategies discussed in Section 7. The output of this model would be used in the production of a cost-effective ranking of possible control strategies for environmental lead, as outlined in Section 4. The model developed within the Technology Policy Unit (TPU) for this purpose was based on the Lutz work, and the equation and notation are shown in Table 5-11. In developing this model an attempt has been made to reflect current research in the field of the relationship between lead exposure and blood lead concentration. To this end the most recent values available have been employed for the various parameters, and where no suitable value could be found, parameters have been derived from the appropriate research. These are shown in Table 5-12, together with their source of reference. (See Appendix 1)

From Lee et al., 1971[21], 5% of the average urban lead aerosol has a mean equivalent diameter greater than 0.5 u. These larger particles tend to become trapped in the upper lung and are transported to the back of the throat by ciliary action from where they are subsequently swallowed. The mean retention rate for these particles, given in Danielson, 1970[22], is 80-95%. If it is assumed that all particles larger than 0.5 u which are retained the lung are later transferred to the throat, a value of 0.044 is obtained for F, the lung-gut

Table 5-11

$$(PbB) = \frac{F, F\ B\ (PbA) + F_1\ (PbG) + D\ B\ (PbA)}{\lambda\ K}$$

Notation:

λ	Elimination rate coefficient of lead from blood, $(days^{-1})$.
F_1	Fraction of ingested lead absorbed by the gastro-intestinal tract.
F	Fraction of airborne lead ejected from the lung and subsequently swallowed.
B	Pulmonary ventilation, (m^3/day).
PbA	Airborne lead concentration, (ug/m^3).
PbG	Daily lead intake from the diet, (ug/day).
D	Fraction of inhaled lead retained in the lung.
K	Blood Volume in ug/100 ml units.
PbB	Blood lead concentration, (ug/100 ml).

Table 5-12

Parameter	Value	Source of Reference
*λ	0.033	Chamberlain et. al, 1975,[24] 1978[20], Griffin et. al, 1975[20], Rabinowitz 1973[13], 1974[14], 1976[15], 1977[16].
B	15 m^3/day	Chamberlain et. al, 1978[20].
D	0.37	NAS, 1972[8].
F_1	0.10	Kehoe, 1961
*F	0.044	Danielson, 1970[22] and Lee 1971[21].
K	52 x 100 ml	ICRP, 1975[2].

* Those parameters marked with an asterisk are derived rather than cited directly. An explanation of their derivation is given in the text. (See Appendix 1).

transfer coefficient; i.e. the fraction of lead inhaled that is subsequently swallowed.

The elimination rate constant of lead from blood, λ, is equivalent to the inverse of the mean life of lead in blood, or ln 2 divided by the half-life of lead in blood, both lives measured in days. Reviewing the literature for values of the mean, and half,-life of lead in blood suggests a value for λ of 0.033 days. (See Appendix 1).

Inserting these values into the model produces the output shown in Table 5-13 and Figure 5-5. As can be seen, a rise in air lead concentration of 1 ug/m^3 produces a rise in blood lead of 3.27 ug Pb/100 ml blood. It should be noted that these results do not represent how a standard man's blood lead level responds to an airborne lead level rising from zero to 3 ug Pb/m^3, but rather the blood lead level that will be achieved, under conditions of dynamic equilibrium, in response to a constant airborne lead exposure at any point along the range, given constant dietary lead exposure.

Having constructed the model, it was required to test it. To perform this operation the output from the model would be compared with the blood lead values from an epidemiological study, having used the air lead values from the study, along with a suitable value for dietary lead intake, as inputs to the model. The dietary lead value would preferably be an experimentally measured value for the same population from which the epidemiological study was taken, but a value could be estimated from regression analysis of the epidemiological data. The t-test for paired observations would be used to indicate a significant difference, if any, between the experi-

Table 5-13

Airborne lead Concentration ug/m^3	Predicted Blood lead level ug Pb/100 ml
0.00	10.49
0.25	11.31
0.50	12.13
0.75	12.94
1.00	13.76
1.25	14.58
1.50	15.40
1.75	16.22
2.00	17.03
2.25	17.85
2.50	18.67
2.75	19.49
3.00	20.31

PbG was set to 180 ug Pb/day, a value suggested by the MAFF[18] survey.

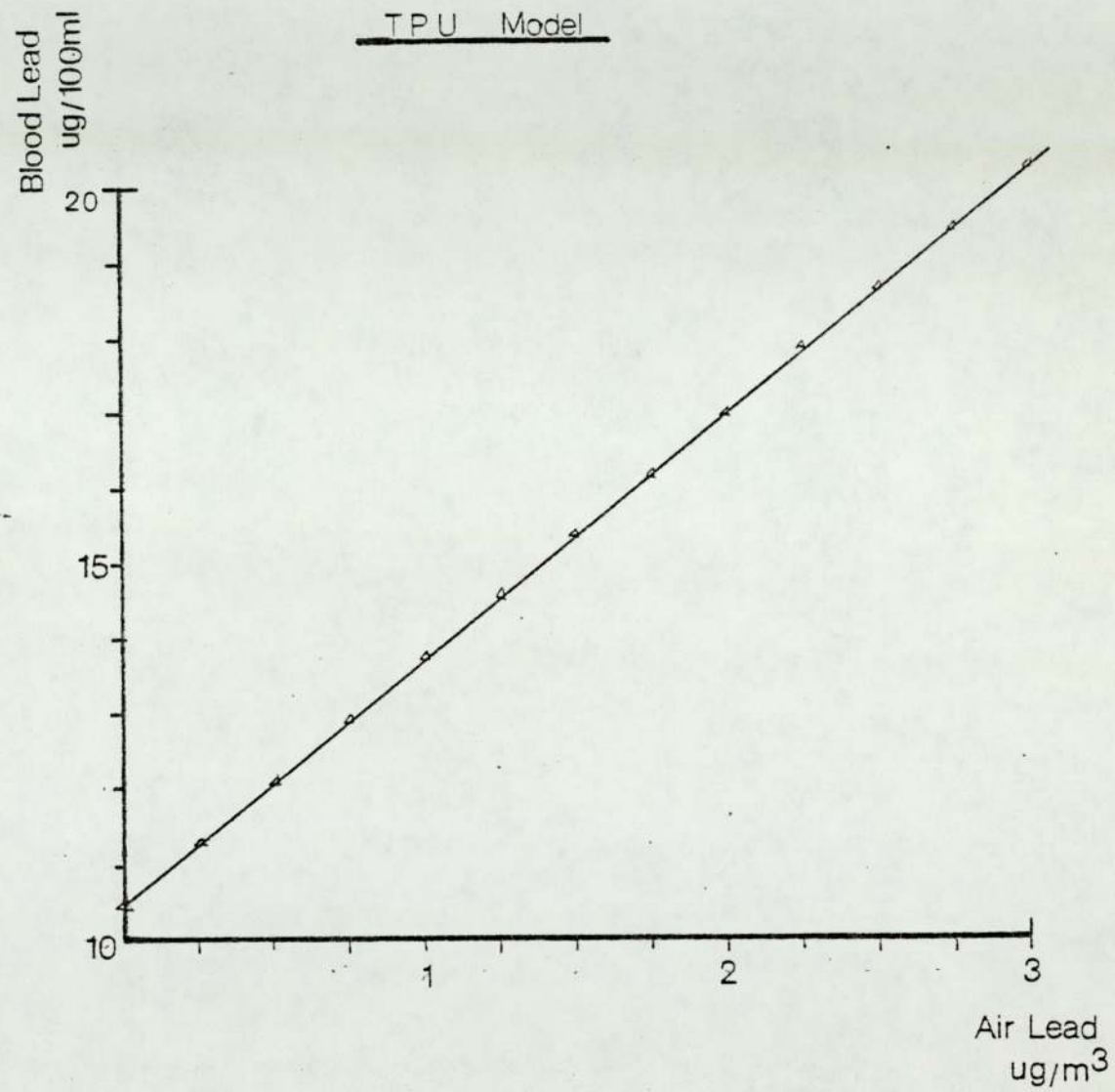

Figure 5-5

mentally measured blood level values and those predicted by the model. One such study suitable for this purpose was that of Tsuchiya et al., 1975[23], which involved three thousand Japanese Policemen. This study provided experimental values for PbA and PbB, a value for PbG was obtained from Horiuchi, 1965,[25] who estimated a dietary exposure of 280 ug Pb/day for Japanese adults. In this instance the t-test indicated no significant difference between the measured data and the model's output, i.e. the model provided an excellent fit to the data. (See Appendix 1).

In conclusion this model is based upon the concept of a standard man , aged between 25 and 50 years. Individual response to environmental lead exposure varies enormously and so the model's output is unlikely to be accurate for a specific man, unless his physiological parameters for lead metabolism and lead exposure had been measured accurately. However, it should be practical to calibrate the model for specific populations, from work carried out on these populations' exposure to lead in the environment.

In contrast to the EPA's use of modelling and the standard man concept, discussed in Section 3, from which they developed their 'maximum permissable average air lead concentration of 2 ug Pb/m^3, an absolute value, the TPU's model will be used to produce a cost-effective index ranking, a series of relative values. Thus even if the model is inaccurate in its representation of the lead exposure-blood lead relationship, the relative cost-effect of the proposed lead control strategies will remain unaltered. As mentioned previously, those parameters significant to the model's accuracy, and to the cost-

effective ranking, may be identified and further research carried out to permit the model to undergo iterative improvement. This process cannot be applied to the EPA's model as individual parameters cannot be identified. In this case improvement can only be obtained by carrying out further large-scale epidemiological studies to confirm the form of the relationship, e.g. semi-log etc., and carrying out repeated analysis to produce long-term average values for the slope and intercept of the function.

These latter points indicate the inherent superiority of the simulation model over its statistical counterpart. Unlike the latter, the former may be easily altered to reflect state of the art knowledge of human lead metabolism. Combining this with the use of the model, albeit an imperfect one, to produce a relative value such as a cost-effective index, modelling may provide the policy-maker with a powerful, effective tool to aid decision-making.

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SECTION 6

The Effects and Impacts of Strategies to Reduce Environmental Lead by Controlling Lead Emissions from Motor Vehicles

The strategies under discussion in this project were outlined in Section 4, and in this Section it is proposed to assess their impact on the Refinery-Engine - Environment System; i.e. their effects on refinery operation, engine efficiency, and human blood lead concentrations as represented by the standard man model developed in Section 5. It may be useful at this point to reiterate the philosophy of using the blood lead of a standard man as a surrogate for the population's blood lead distribution. Firstly the distribution of blood lead concentrations for the total population is not known and secondly, if it were, the effect of any strategies under consideration upon this distribution could not be calculated because the distribution of those factors which control the individual's blood lead concentration, e.g. airborne and dietary lead exposure and lead absorption and excretion rates, is equally unknown for the population as a whole. This lack of knowledge leaves no alternative but to use the standard man approach in order to compare the effectiveness of strategies, in terms of the reduction they bring about in blood lead concentration. At present there is no absolutely conclusive evidence that environmental exposure to lead is harmful, but, if this should be shown to be the case, then blood lead would be an indicator of harm as it reflects current exposure to lead, from all sources. In terms of minimising the possible harm to the population, especially suspected lead sensitive risk groups, it may be necessary to take action to reduce lead exposure on an 'insurance policy' basis, i.e. before conclusive evidence of lead's harm is found. To carry out a policy study on the

basis of the above argument, the effectiveness of a number of strategies for reducing environmental lead exposure has to be compared using the standard man approach at present because of the impossibility of assessing the distributive effects mentioned previously. Also, as described in Section 5, the model may be analysed in terms of those parameters which are critical to the cost-effective ranking and knowledge of these factors may undergo iterative improvement by means of further clinical and epidemiological research. These then are the reasons for adopting the standard man approach in calculating the effectiveness of the strategies in reducing lead concentrations. Taking these into account, the impact of those strategies selected for examination will now be assessed, not only for their effectiveness, but also in terms of how they affect refinery operations and engine performance. However, only the primary effects of the strategies will be analysed in depth, secondary effects being described more briefly; secondary effects being defined as those which are not necessarily brought about by the implementation of a particular strategy. The main reason for this is one of scale; if all the ramifications of each strategy were covered in detail, it would be beyond the scope of the project brief. Also, however, there is very little literature available on these effects and so it is very difficult to build up a picture of the strategies[1] impacts in these areas. This point will be brought out more fully when it arises within the strategy analyses.

The lead added to gasoline, initially as the organic liquid compounds tetraethyl and tetramethyl - lead, is emitted from the exhaust systems of motor vehicles as

inorganic halides, oxides, carborates and sulphates of lead. Approximately 75% of the lead initially added is emitted[1], the remaining percentage being trapped in the engine's sump oil and exhaust system. A small amount of organic lead also escapes to the environment, some 0.1-4.0% of the total airborne lead[2]. The concentration is somewhat higher near filling stations and in enclosed spaces such as multi-storey car parks. However, organic lead compounds are photo reactive and disappear rapidly from the atmosphere.

In excess of 90%[3,4,5,6] of all atmospheric lead is the result of the combustion of motor fuels containing lead additives. The above two factors, that 75% of the lead added to gasoline is emitted and that these emissions account for at least 90% of all airborne lead, will be taken as constant values in the calculation of the effectiveness of the various strategies under consideration.

To return to the concept of measuring the effectiveness of the various strategies under consideration, it would be useful to measure this in terms of the reduction in harm caused by lead pollution which they bring about. However, this is impossible due to the nature of the harm brought about by exposure to lead at environmental levels not being sufficiently understood as to permit its direct measurement. This being the case, it is therefore necessary to employ an indicator of effectiveness, or harm, which in this case was chosen to be blood lead concentration, for the reasons outlined in Section 4. Furthermore the use of blood lead levels as the indicator of the strategies[1] effectiveness, in terms of blood lead's value as an indicator of the harm caused by

lead exposure, permits this policy study to be compared directly with clinical studies to determine the risks to the population caused by exposure to environmental lead, because these latter studies normally relate various forms of lead exposure to blood lead concentration.

Having obtained a means of determining the effectiveness of each proposed strategy, it should be reiterated at this point how this information will be used. Previous studies generated absolute criteria for the control of lead exposure using a similar, although less sophisticated standard man approach. However, this work is open to a number of criticisms from a policy viewpoint. Firstly there are many unknown factors associated with the population's exposure to environmental lead; e.g. the distribution of blood lead concentrations and lead uptake and absorbtion rates throughout the population. These may vary considerably from those of a standard man, and hence, in the light of future research, exposure criteria developed from a standard man may well be highly inaccurate. However, in this study, the cost and effect of each strategy will be compared in terms of a cost-effective ranking. Thus, any inaccuracies contained in the current modelling of the strategies' effects may not affect the ranking position of the strategies. This brief review indicates how the information produced by the model, the strategies' effectiveness, will be used, although the cost-effective ranking of the proposed strategies will be dealt with fully in Section 8.

The same methodology will be employed in assessing the potential effect of each strategy; the reduction in airborne lead from current levels will be calculated and this will be

input to the blood lead model in order to obtain the new blood lead concentration brought about by the strategy, and details will be given of the technological effects and requirements of the strategy, although the costs will be discussed in Section 7. The reduction in blood lead concentration over current levels, in ug Pb/100 ml blood, will be termed the effect of the strategy.

Table 6-1 shows the base run of the model to give the current standard man blood lead level. This is based on airborne lead levels brought about by the current concentration of lead in gasoline 0.45 g Pb/l[8], and levels of lead in the total diet, solids and liquid, described in the MAFF report[9]. The actual airborne lead concentrations are taken from the report on lead pollution in Birmingham[10], and are representative of values found elsewhere. Blood leads suggested by the model are shown for these values, and these are of the same order as those found in the Birmingham study mentioned above. Figure 6-1 shows a graphic representation of lead exposure as outlined in this study. Again it is noted that all blood leads and physiological parameters mentioned in this Section are from male subjects, or the standard man. Having defined the base case, the six strategies described briefly in Section 4 will now be dealt with in detail, calculating their effects and describing the technology required for their implementation.

The first strategy to be considered is the total elimination of lead antiknock additives from gasoline. This would lead to an eventual 90% reduction in airborne lead concentrations as shown below:

Table 6-1

Typical Lead Exposure Parameters for U.K.

Airborne Lead Concentration[8] - 1 - 2 ug Pb/m^3

Dietary Lead Concentration[9] - 180 ug Pb/day

Airborne Lead ug Pb/m^3	Blood Lead ug Pb/100 ml
1.0	13.76
2.0	17.03

Mean Blood Leads[10]

(Male)

Venous	16.0	ug Pb/100 ml
Capilliary	18.8	

The subjects[10] lived near a major motorway interchange and would be exposed to lead concentrations of the order of 2 ug/m^3.

Airborne Lead Concentration ug Pb/m^3

Current	After Strategy
2.0	0.2

This reduction would bring about the following change in blood lead concentration as predicted by the TPU model for a standard man:

Blood Lead Concentration ug/Pb/100 ml

Current	After Strategy
17.03	11.14

Hence, the above strategy brings about a 5.89 ug/100 ml reduction in blood lead, for a standard man. However, what is not clear from the above is the time scale over which the reduction will occur, which is dependent on the technical factors associated with the strategy.

As described fully in Section 2, lead alkyl compounds are added to gasoline to prevent knock in the engine. They achieve this goal by raising the octane quality of the fuel by up to 6 Research Octane Numbers; i.e. 4-STAR fuel is refined to 92 RON from the crude oil and lead additives are used to raise this value to 98 RON[11]. It would not be possible to change to lead free fuel operation overnight for two reasons. Firstly the refineries could not supply sufficient fuel of adequate octane quality, 4-STAR, to satisfy the demand, and conversely a large proportion of the car population could not operate successfully on a lower grade of fuel than that for which they were designed.

Secondly, the production of lead-free fuel would inevitably require an increase in crude oil consumption, to maintain octane quality, the availability of which would depend on the current market supply and demand situation.

To examine the first of these factors, it is useful to look at the U.K. fuel supply distribution, as shown in Table 6-2[12]. As can be seen, the vast majority of gasoline consumed within the U.K. is 4-STAR and this is, in fact, the only grade with a positive growth trend. Overall fuel demand is also rising, although the figures for 1974/75 reflect the action taken by the OPEC organisation at that time. This also partially accounts for the decrease of 5-STAR fuel with its high crude oil consumption. However, advances in engine technology must also account in part for the drop in demand for this extra high octane fuel, 100+ RON[13]. Hence, there appears to be a 75%/25% split between 4-STAR/other Grades in the U.K. market. This agrees with the information calculated by CONCAWE[14] of 75% Premium, at 98 RON, and 25% Regular, at 92 RON. The terms Premium and Regular correspond with the U.K. 4-STAR and 3/2-STAR grades of gasoline. Hence, if lead free gasoline were to be introduced immediately, up to 75% of U.K. vehicles would be adversely affected due to running on gasoline of a lower octane quality than that for which they were designed, unless current octane quality was to be maintained. The option which would bring about the smallest perturbation in the Refinery - Engine System would be to introduce new engine designs which would operate successfully on 2-STAR fuel. This has an octane quality of 91-92 RON and gasoline may be refined to that standard without substantial

Table 6-2

YEAR	5-STAR	4-STAR	3-STAR	2-STAR	TOTAL
			TONNES		
1972	2,451,212	7,889,724	2,176,449	3,130,193	15,647,578
1973	2,772,457	8,582,909	2,093,185	3,210,770	16,659,321
1974	2,077,700	9,458,100	1,901,900	2,785,400	16,223,100
1975	1,665,210	10,025,260	1,809,680	2,624,660	16,124,810
1976	1,404,700	11,144,800	1,714,700	2,614,400	16,878,600
1977	744,300	12,671,400	1,487,200	2,432,800	17,335,700
1978	198,600	14,701,900	1,214,900	2,233,000	18,348,400
r	-0.96	0.98	-0.98	-0.96	0.87
1979	-re value	14,967,758	1,159,171	2,087,529	18,189,748
1980	obtained	16,049,908	1,006,178	1,929,725	18,550,845
1981	zero production	17,132,058	653,184	1,771,921	18,911,942
1982		18,214,209	700,191	1,614,116	19,273,039
1983		19,296,359	547,197	1,456,312	19,634,137

Data is available up to 1978. After this, data was produced by trend line analysis, the correlation for the linear trend line for each grade being shown against 'r'

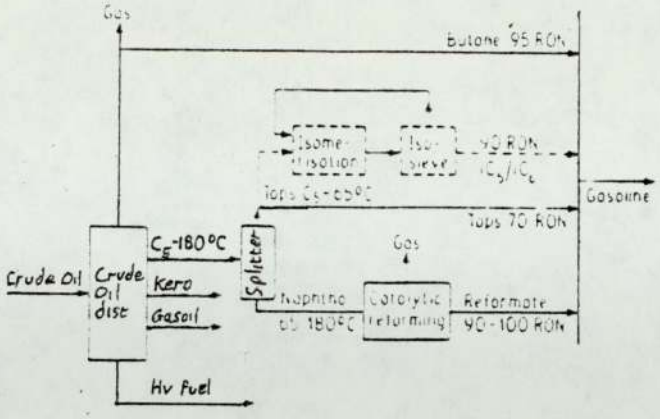
changes in refinery operations or crude oil consumption. However, there is an enormous barrier to introducing radically new engine designs in terms of research and development costs. Furthermore, alterations in vehicle design would not be limited to engine design. If lead antiknock additives were to be eliminated from gasoline, the resultant loss of 6 to 8 RON would mean that lower efficiency, lower compression engines would have to be employed, unless gasoline octane quality was maintained via more severe refinery operations. To maintain current vehicle performance in the former situation would require radical changes in vehicle design; power plant, chassis, body, transmission, accommodation etc. Currently a period of approximately five years elapsesbetween the design of a new car and its first production. However, this figure applies to traditional vehicles which employ much of the technology and often many of the parts, such as engines and transmission, from previous generations of machines. For the radically new designs required throughout any manufacturer's complete range, as required by the above approach, a much longer period would probably be necessary. There would not appear to be any information in the literature regarding this option, perhaps as it has not yet been considered realistic by the automobile industry. This industry also puts forward theoretical as well as financial arguments against the use of lower octane fuelled vehicles as follows, although detailed analysis of these problems was considered beyond the scope of this project. The lower compression ratio engines designed by the motor industry to operate efficiently on 2-STAR fuel will be less efficient overall than engines

designed to operate on 4-STAR fuel. This objection to the above approach is founded in the laws of thermodynamics - the higher the compression ratio the greater will be the thermal efficiency of the engine. It is argued that, in the case of an increasingly scarce resource such as petroleum, it ought to be used as efficiently as possible, and with respect to the consumption of gasoline by internal combustion engines this requires the use of the most thermally efficient designs available and therefore the compression ratio of the engines should be kept high and lead free fuel of sufficiently high octane quality produced to enable their efficient operation. This latter approach, introducing lead free gasoline and maintaing octane quality, has already been adopted in the U.S. However, it immediately introduces an economic penalty, to be discussed in Section 7, and a technical problem. The lead additives presently in use make up the final six numbers in the fuel's RON rating. If lead free fuel is to be used, more than six octane numbers have to be made up using high octane blending components at the refinery. That is lead free 4-STAR fuel will have to be ~102 RON as opposed to the current ~99 RON leaded variety[15]. This phenomenon, the lead-road bonus, was described in detail in Section 2. The introduction of lead free gasoline with no change in the compression ratio of vehicle engines would require new refinery plant to produce higher volumes of high octane blending stocks and increases in crude oil consumption from which to extract these new components. There is a further difficulty associated with the refinery technology employed. At present European refineries, including those in the U.K., extract approximately

17%-25% of gasoline per barrel of crude oil. In the U.S. roughly 50%[16] of the barrel is converted to gasoline using the technology that would be required in Europe to produce lead free gasoline to satisfy the current total demand. This approach, however, would lead to a surfeit of gasoline and petro-chemical products unless there was to be a reduction in the number of refineries in Europe, with associated redundancy problems. Furthermore, most European refiners operate on Europe-wide basis which would be subject to major disruption if the number and/or location of their refineries were to be altered. Very little research has been done in this area by the refiners, they having concentrated on ascertaining the costs of producing lead-free petrol, and it is beyond the brief of this study to carry out such research. However, it is mentioned because should a lead elimination strategy be implemented, these difficulties may eventually be encountered.

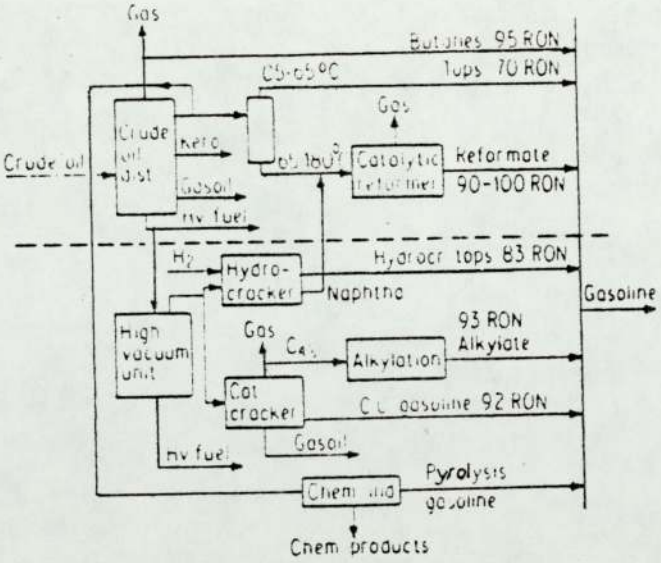
The production of lead free premium gasoline would require the following changes at a typical European refinery; Figure 6-1[11] shows the two types of refinery commonly found in Europe. The first, 6-1 (a), is the hydroskimming, or simple, refinery so called because it does not possess the high-vacuum distillation unit and cracking units found in the complex refinery, 6-1 (b). The distillation unit provides the heavy feedstocks required by the Hydro - and Catalytic Cracker units enabling the complex refinery to extract 25% gasoline from the barrel of crude oil, as opposed to the hydroskimming refinery's 17%. The finished gasoline in a hydroskimming refinery is blended from three components, butane, tops and reformate, as shown in Figure 6-1 (a).

(a)



Hydroskimming Refinery

(b)



Complex Refinery

Figure 6-1

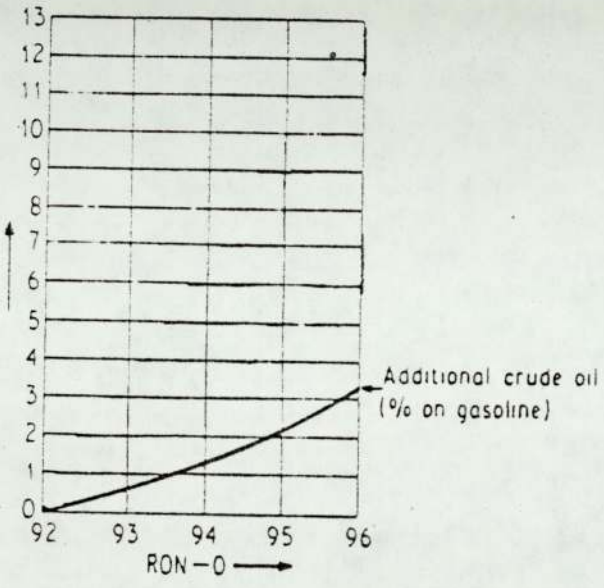
From van Gulick, 1975

The amount of butane that can be blended is limited by vapour pressure requirements and the ratio of tops/reformate is constrained between limits set by the minimum and maximum mid-range volatility of the gasoline. At a given gasoline volatility the tops/reformate ratio is fixed and the RON required of the reformate is then set by the RON of the gasoline required before the addition of lead. Raising the RON of the unleaded gasoline, an obvious requirement for the production of lead free fuel, must then be achieved by raising the RON of the reformate. This involves a reduced reformate yield and an increase in the energy consumption of the reformer units. Gasoline output can only be maintained by providing additional reformer feedstocks by processing more crude oil. Also high gasoline volatility requirements clash with high RON requirements. For a high gasoline volatility, a large percentage of low octane tops has to be blended which may force a reformate RON quality exceeding the technically achievable level. Improvements may be made via isomerisation, dotted scheme in Figure 6-1 (a), which may increase the RON of light tops by up to 20 units.

The complex refinery, Figure 6-1 (b), increases gasoline output at the expense of fuel oil. The hydro-cracker provides tops of a reasonable octane quality and naptha equivalent to medium straight run naptha. The hydro-cracked naptha must therefore be reprocessed in a catalytic reformer. The other conversion unit, a catalytic cracker, produces a gasoline fraction containing 40% olefins. These light olefins may be processed with iso-butane to produce a high RON alkylate. The complex refinery is also linked to the chemical industry

via its use of pyrolysis gasoline, a by-product of ethylene production. The complex refinery, having more blending components than the hydroskimming refinery, has somewhat more flexibility to adjust gasoline volatility. Hence, although equally dependant on catalytic reformate to achieve gasoline RON requirements, the complex refinery is in a better position to balance volume output. A higher gasoline RON, as required by lead free gasoline, at constant volume production of all products, including gasoline, requires a higher reformate RON and more naptha reformer feedstock. The additional naptha may be obtained by raising the high vacuum distillation and hydro-cracker intake. More crude oil will be required to correct the loss of fuel oil output that would otherwise result. Extra crude oil will also be required to provide process energy for the increased operation of the high vacuum distillation, hydro-cracker, reformer and support plant. Extra processing of crude oil in the crude distiller will also require an increase in process energy. If the balancing within a particular refinery can be accomplished to perfection, the gasoline RON may be raised while the output of all products remains constant. The extra amount of crude oil processed will then be used exclusively to provide the extra process energy requirements. However, extra plant capacity will also have to be installed for the crude oil distiller, vacuum distillation, hydro-cracking, reforming and support plant.

As has been mentioned, a lead-free gasoline strategy will require an increase in crude oil consumption for the various reasons outlined above. Figure 6-2 shows one estimate[11] for the necessary increase in crude oil consumption. Other values ranging from 3%-8% are cited in the CONCAWE report[17].



% Increase in Crude Oil Required for Lead Free Gasoline

Figure 6-2

Adapted from van Gulick, 1975

Turning away from the refinery component of the Refinery-Engine System, there is one difficulty with engine operation on lead free gasoline, that of valve seat wear. Engines which are designed to operate on diesel fuel or Liquefied Petroleum Gas (LPG) are fitted with valve seats manufactured from a wear resistant alloy because it is recognised that, there being no lead in their fuels, there will be no lead-lubrication of the valve seating interface[18]. To what degree this problem would affect European engines currently on the road has not been generally assessed, although serious valve recession difficulties might occur as a result of operating these engines on lead free fuel.

To sum up then, the introduction of lead free fuel would require major re-organisation of the refining industry which would take at least five years, this being approximately the length of time between deciding to construct new refinery plant and its commissioning. The production of lead free fuel requires an increase in crude oil consumption for process energy, and this is raised still further if current octane quality standards, BS 4040[19], are to be maintained. With respect to vehicles using this fuel, there may be mechanical difficulties associated with the absence of lead from the fuel. Again, it takes about five years from the initial design to first production for a new car, and so it would appear that a lead free gasoline strategy would take at least five years to implement.

The next two strategies may be considered as variations on a theme, and as such will be dealt with together. Both involve the reduction of the amount of lead additives employed

to prevent knock, as opposed to their elimination, and have the following effect on airborne lead concentrations:

Lead Additive Concentration ug Pb/l		Airborne Lead Concentration ug Pb/m^3	
Current	Proposed	Current	After Strategy
0.45	0.40	2.0	1.8
0.45	0.15	2.0	0.8

These reductions were calculated using the parameters of 75% of the lead added to gasoline being emitted, and these emissions accounting for 90% of the lead present in the atmosphere. The reductions shown above produce the following changes in the blood lead of a standard man according to the TPU model:

	Airborne lead ug/m^3	Standard Man's Blood lead ug/100 ml
Current	2	17.03
Strategy 2	1.8	16.38
Strategy 3	0.8	13.11

Thus, reducing the amount of lead additives in gasoline from 0.45 g/l to 0.40 g/l leads to a 0.65 ug Pb/100 ml reduction in blood lead, and reducing from 0.40 g/l to 0.15 g/l gives a 3.92 ug Pb/100 ml reduction in blood lead concentration. The first of these two options is proposed legislation for the EEC nations, whilst the latter has so far been adopted, in Europe, by Germany[17] and is due to be introduced by Sweden in 1980/81.

With both of these strategies, problems with engine valve

seat wear will be avoided, the 0.15 g Pb/l level being sufficient to provide adequate valve seat lubrication. However, the strategies will have an effect on refinery operations. Once again if vehicle engine designs could be altered to run on Regular (2-STAR) fuel there would be few, if any, refinery problems. However, there is a lead time of thirteen years for this approach - five years to the first suitable production vehicles and seven to eight years to renew the car population. Apart from the lengthy time scale, there are further objections to this approach described fully earlier in this Section. From the refinery viewpoint the impacts of these two strategies will be similar to that of the lead elimination strategy, although reduced in magnitude. With respect to the hydroskimming refinery, the reduction in lead could be compensated for by increasing the severity of the catalytic reforming operation, although this provides only partial compensation. The effects of this action are three-fold. Firstly, the yield of reformate is reduced and that of gas increased; secondly the maximum possible throughput of the reformer unit is reduced and thirdly there is an increased usage of crude for process energy. However, there is a limit to which severity can be increased. Some older units would not be able to make any compensation at all, and even more recent plant may experience throughput reductions of up to 50%. This results in an increased demand for reformer feed-stock, higher gas production for which there may be no demand, and the necessity for extra plant to compensate for the reduced throughput due to the higher severity of reformer operation. A hydroskimming refinery would experience severe

difficulties in producing 4-STAR fuel with a lead content of 0.15 g Pb/l, and major plant additions and alterations would be required to achieve this goal[21]. The complex refinery copes with the reduction in lead additive concentration in a different manner. Straight run gasoline is replaced by reformate to maintain octane quality, this action being made possible by the increased flexibility of the plant. However, a reduction in lead additive concentration to 0.15 Pb/l requires an increase in reforming severity, with the associated disadvantages mentioned previously. The alkylation plant compensates for this effect to a certain extent, but there is an increased usage of crude oil for process energy and feedstocks. Table 6-3[A] shows the drop in octane quality brought about by these reductions in lead additive concentration. The increase in crude oil consumption to implement the lesser of the two reductions, 0.45-0.4 g Pb/l, has been reported as from 1-4% of current consumption, whereas the reduction from 0.45-0.15 g Pb/l is reported to require a 3-8% increase in crude oil usage[21].

In conclusion, strategies involving the reduction or elimination of lead antiknock additives from gasoline all require modification to, or additional, refinery plant and an increased crude oil consumption. The degree to which these effects occur rises as progression is made from a small reduction in, to the total elimination of, lead additives from gasoline, in addition to which the elimination of lead additives from gasoline may require modification of engine valve seats in order to avoid wear problems. As the previous paragraphs show, albeit briefly, altering the composition of

Table 6-3[17]

Lead Additive Concentration g Pb/l	RON increase supplied		
0.45	5.7	0.5	3.4
0.40	5.2		
0.15	2.3		

gasoline involves interfering with a highly complex system with the inevitable result that perturbations spread throughout the entire system. Hence, the gasoline manufacturers, the motor manufacturers and particularly the alkyl manufacturers would prefer an approach which, whilst reducing lead emissions to the atmosphere, did not require the elimination, or reduction, of lead additives in gasoline.

This requirement appears to be satisfied by the lead trap, a device installed in the vehicles exhaust system which partially eliminates lead particulate emissions to the environment. The 1974 TRRL report[22] describes a lead trap and its effectiveness. The traps consisted of stainless steel wire, with a volume approximately five times that of the engine capacity: i.e. a one litre engine would be fitted with a five litre trap. It was reported that a number of factors affected the performance of the trap whilst in use. These included changes in exhaust gas temperature, gas velocity and exhaust path length. A low velocity and long path tended to improve the trap's efficiency, low temperature assisting this affect. Hence, the siting of the trap within a given vehicle's exhaust system will have an effect on the trap's efficiency. In terms of the vehicles efficiency, engine test bed studies showed that the above design of lead trap had no significant effect on power output, gaseous emissions, noise or exhaust gas back pressure and thus there was no fuel penalty, in terms of gasoline economy, associated with the trap. The trap had a life of at least 24,000 miles and showed no signs of having reached the end of its useful life at 36,000 miles. However, extensive high speed operation may reduce the trap's lifetime

due to failure of the steel wire trapping material as a result of exposure to high temperatures. A purging effect was noticed when operating conditions became more severe than those under which the lead was originally retained; i.e. when a vehicle used mainly in urban conditions was taken on to a motorway for high speed operation. The trap was then subjected to higher gas temperatures and velocities and some of the lead retrieved in the system was emitted until a new equilibrium between lead supplied from the engine and lead retained by the trap had been achieved. The efficiency of the trap was such that it led to a 18% to 41% reduction in lead emissions, assuming 70% emission of lead for an untrapped vehicle.

The U.K. manufacturer of lead antiknock compounds for gasoline, the Associated Octel Company, has carried out extensive research into the effectiveness of lead particulate traps for automobiles. They produced a Situation Report in 1977[23] which included traps of a more sophisticated design than those covered in the TRRL study. The following factors have been noted regarding exhaust gas filters:

1) The reductions in lead emissions attributed to lead traps range widely from the very poor to excellent.
2) There is resistance from the motor manufacturing industry to fitting an unproven device which might mar the performance of their products.
3) Emission control authorities are skeptical about the efficiency of lead traps and are concerned about their legislative control, specifications and monitoring.

For these reasons the Octel Company conducted their survey and provided reassurance on each of the above points. However, it must be remembered that the company has a vested interest in maintaining current levels of lead in gasoline, as they are the sole U.K. supplier of this product and their income would be adversely affected by compulsory lead reductions. With respect to the lead particulate traps themselves it was observed that the higher the operating severity, i.e. speed, load and engine capacity, the higher the lead emissions. Further, the design of the exhaust system was related to exhaust lead emissions, generally the larger the exhaust system the lower the lead emissions. The driving history of the vehicle prior to its being tested with a lead trap fitted was also found to be important; e.g. mileage and driving severity in comparison with test conditions. Lead emissions increase as exhaust system mileage increases and a significant increase in emission level occurs if the test operating conditions are more severe than those employed prior to the test. The converse is also true, there being a reduction in lead emissions if test conditions are less severe than those to which the vehicle was previously subjected. Finally Octel reported that lead emissions were markedly higher if the engine was started from cold, as opposed to a hot start, this being due to lead being released from the trapping material as a result of thermal shock. It was pointed out that the above parameters have to be controlled if test results are to be comparable.

Two types of lead trap were examined, axial and radial, the filter boxes replacing the resonator and silencer found in

many European vehicle exhaust systems, three examples of which are shown in Figure 6-3. Figure 6-4 shows the designs of the two traps described in the study. In use the filters replace the resonator and silencer boxes, as shown in Figure 6-3, the filter material replacing the normal sound absorbing material found in these units. Compared with normal exhaust systems, filtered exhaust systems show an increase in back pressure although this does not appear to have any significant effect on vehicle operation[22]. Good reductions in lead emissions have been achieved by such filters on a variety of European vehicles tested by independant authorities[24,25]. To reduce the back pressure effect found with the axial design, the radial design was produced which presents a greater surface area to the exhaust gas flow and hence offers lower back pressure, as shown in Figure 6-4. The radial filters, however, usually have a lower catchment than their axial counterparts. The filter matrix, steel wool, is coated with alumina impregnated with phosphate in order to eliminate the purge effect which occurs under severe operating conditions. A further advantage of the availability of two types of filter was that overall filter systems could be designed to match the back pressure characteristics of most standard exhaust systems. The filters themselves are constructed from normal exhaust material, mild steel, but stainless steel may be substituted if the full lifetime of the filter is to be exploited. The efficiencies of the filters are shown in Table 6-4[23], the single filters being used in small vehicles whose exhaust systems would not accommodate a twin filter design.

Typical European Exhaust Systems

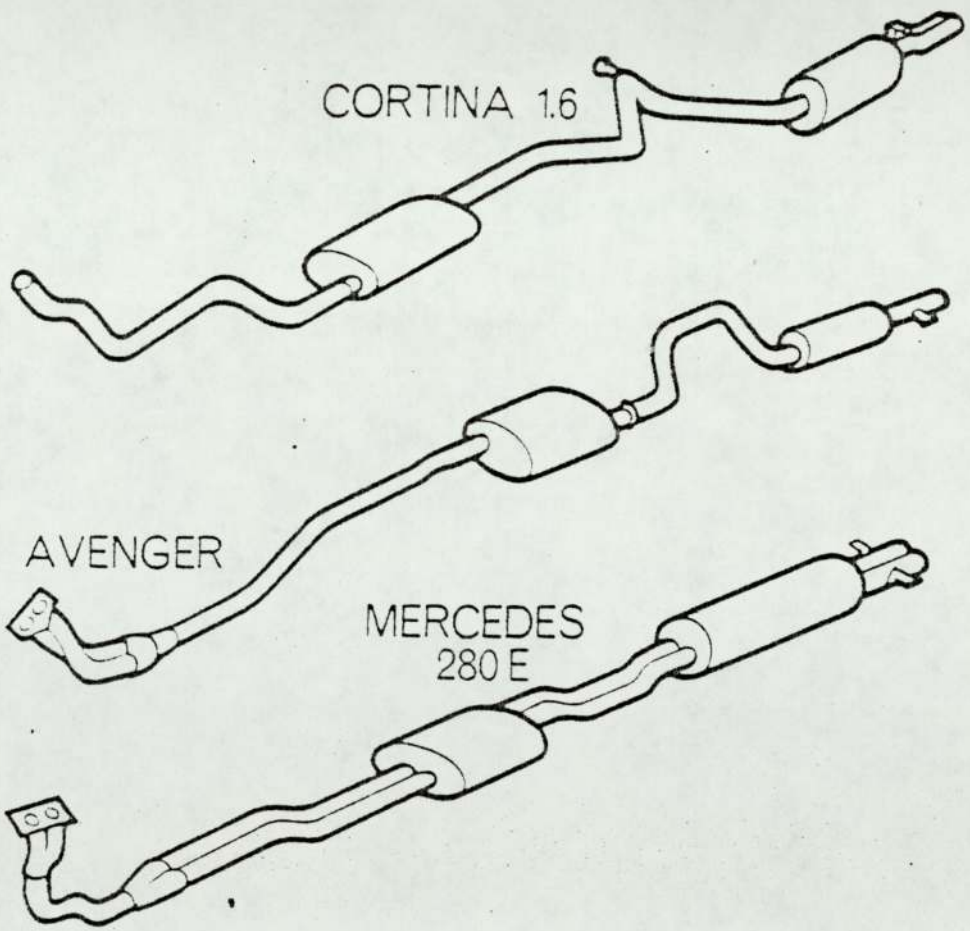
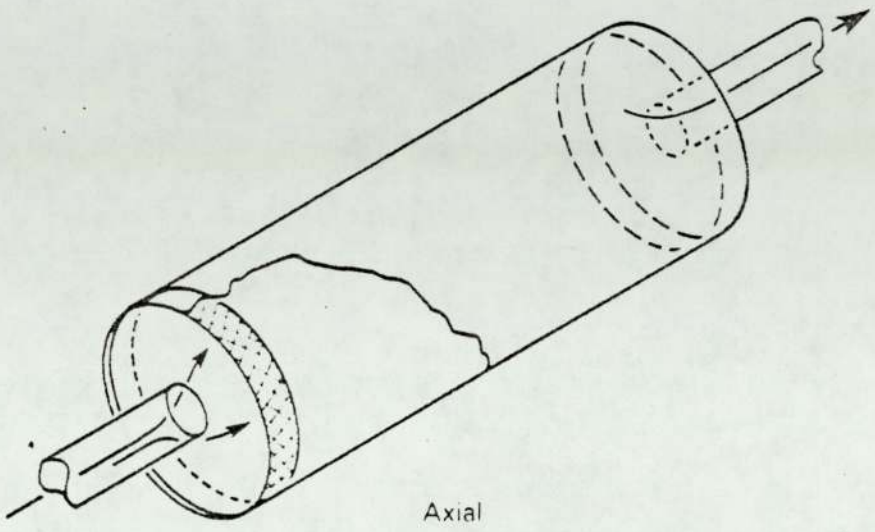


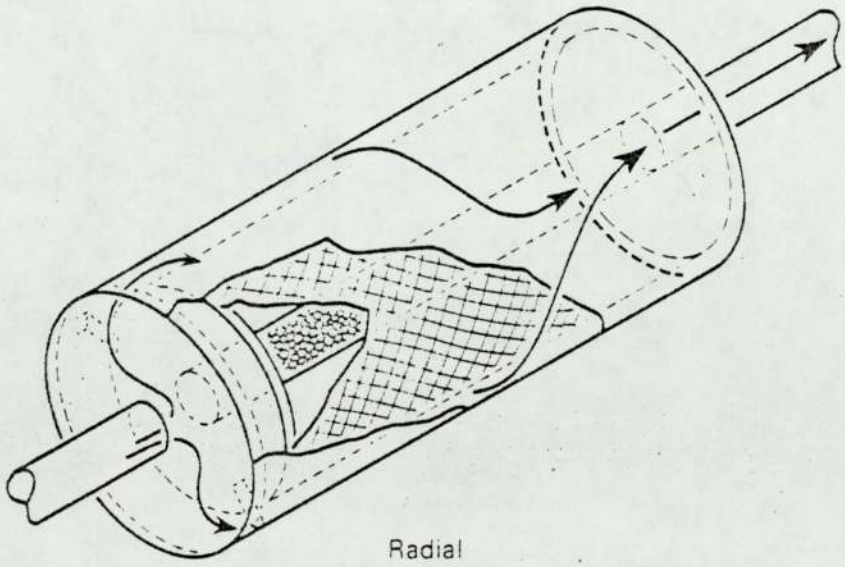
Figure 6-3

Adapted from Campbell, 1977

Lead Exhaust Traps



Axial



Radial

Figure 6-4

Adapted from Campbell, 1977

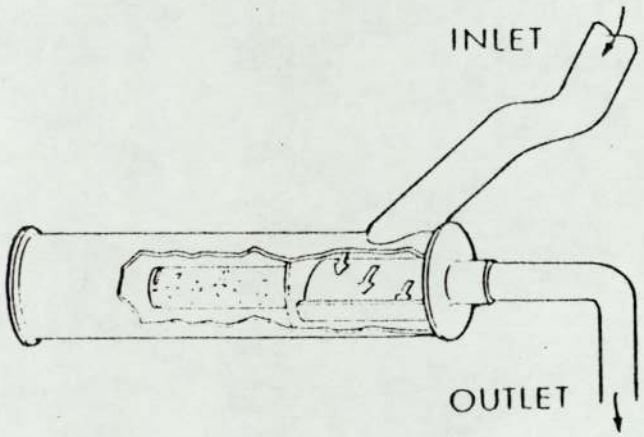
Table 6-4[23]

	Single		Twin	
	Radical	Axial	Radical	Axial
% Reduction in lead Emissions	50	57	65	70

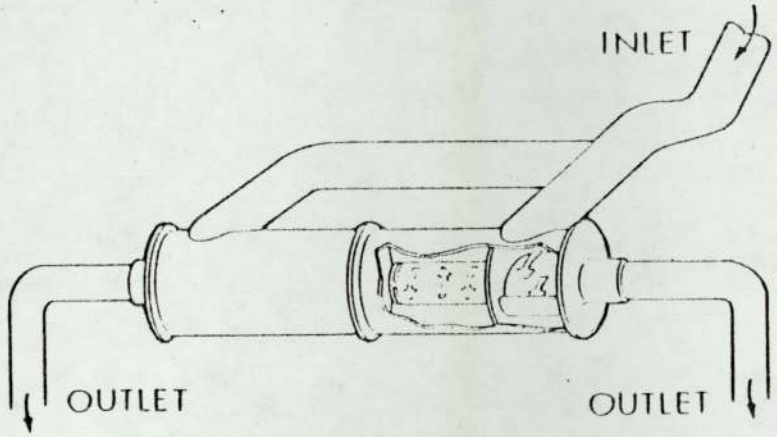
The other main manufacturer of lead antiknock additives, the Ethyl Corporation, has developed its Tangential Anchored - Vortex Trap (TAV) as shown in Figure 6-5[26]. This trap incorporates heat transfer, agglomeration and inertial separation of particles in a single unit giving reductions in lead emissions of up to 75%[16]. The results of the Ethyl Corporation's work on anch red vortex traps was used as a basis for the TAV. Metal building lath is cut up and used as the trap matrix filling both entry and collection chambers. This metal acts as a cooling and agglomeration medium in the inlet section and prevents particle escape into the gas flow in the collection chamber. Traps have been designed with both single and twin inlets, as shown in Figure 6-5. In operation the lead particles enter the trap at a tangent to the circumference resulting in an increased residence time. Small particles are deposited on the lath and grow to sizes which can be separated by the inertial separator tube. These particles, released from the lath, are carried to the wall of the anchored-vortex tube where they are dumped into the collection chamber through slots in the tube wall. The exhaust gases exit through the outlet tube. As is apparent, both in design and operation this is a more sophisticated design that the Octel Company's simple, steel wool lined trap, which accounts for its somewhat higher efficiency. Table 6-5 shows the reduction in lead emissions for a number of European cars achieved by the TAV trap[26]. For U.S. vehicles a reduction in lead emissions of 74% was recorded, as opposed to a mean value of 67.7% for the European vehicles. As mentioned in Table 6-5, however, the values obtained for European vehicles

Figure 6-5

SINGLE OUTLET



DUAL OUTLET



Tangential Anchored Vortex Traps

From Lenane, 1977

Table 6-5[26]

Type of Vehicle	Mean % Reduction in Pb Emissions
Fiat 124 Sport	75.6
Fiat 126	64.3
Fiat 128	79.0
BMW 2002	53.0
Renault 12 TL	44.4
Volvo 244 DL	87.7

It should be noted that the mean % reduction in lead emissions are the averages for a variety of test cycles, assuming a 75% emission where the untrapped value is unknown.

are mean values produced from a variety of test cycles and may be subject to error due to incompatability between test cycle results. The mean value cited for reduction in lead emissions by the TAV trap is 72.5%. The TAV trap like its Octel designed counterpart is designed to fit in place of conventional exhaust systems, the resonator and silencer units being replaced by the trapping devices. Unlike the U.K. twin traps, the TAV design does not replace both the above units with traps but rather inserts a further agglomerator in the forward unit, this being followed by the TAV trap. This raises the particle size before they reach the trap, improving the trap's efficiency.

Having outlined the technical aspects of reducing environmental lead by the use of particulate traps, it is necessary to calculate the effectiveness of this strategy. For the sake of clarity the mean efficiency of the various types of trap has been employed for this calculation as shown in Table 6-6. As the TRRL results were based on early designs of trap, a second value has been calculated ignoring these early results. Finally, the highest possible reduction suggested by the values in Table 6-6 have also been calculated, again ignoring the TRRL results. However, it must be noted that the physical design of the traps which achieved these high reductions in lead emissions may render them unsuitable for certain vehicles. These figures result in the following reductions in airborne lead concentrations as a result of employing lead particulate traps:

Table 6-6

Type of Trap		% Reduction in Pb Emissions	
TRRL		29.5	(18-41%)
Single Axial Twin Axial Single Radial Twin Radial	Octel	60.5	(50-70)
TAV	Ethyl	67.7	(44.4-87.7)

Mean Reduction in Pb Emissions - 52.6%

Mean Reductions Ignoring TRRL results - 64.1%

Highest Possible Mean Reduction Ignoring TRRL Result - 78.9%

Current Airborne lead ug/m^3	% Reduction in Pb Emissions	Resulting Airborne lead ug/m^3
a) 2	52.6	1.1
b) 2	64.1	0.8
c) 2	78.9	0.6

The effectiveness of the strategy, measured in terms of the reduction in the blood lead concentration of a standard man is therefore:

	Blood Lead Concentration ug Pb/100 ml	
	Current	After Strategy
a)	17.03	14.09
b)	17.03	13.11
c)	17.03	12.45

Hence, the strategy brings about reductions in the blood lead of a standard man of 2.94, 3.92 and 4.58 ug Pb/100 ml depending upon the efficiency of the lead traps. The effects of these variations in efficiency will be discussed fully in Section 8.

The final two strategies to be examined are combinations of the techniques already described, firstly reducing the lead additive concentration from 0.45 - 0.4 g Pb/l and then fitting exhaust gas filters and secondly reducing the lead additive concentration from 0.45 - 0.15 g Pb/l and fitting exhaust gas filters. The former strategy represents an option which might be implemented after the proposed EEC legislation in 1980[27]. whilst the latter represents an option to minimise lead emissions from automobiles while constrained to keep sufficient lead in gasoline to prevent engine valve damage. The effect-

iveness of the strategies will be calculated after both phases have occurred, i.e. both lead reduction and filter fitting, although it is obvious that fitting of exhaust gas filters could commence in advance of the reduction of lead concentrations in gasoline.

The reductions which would be brought about by the first of these strategies, lead reduction to 0.4 g Pb/l and the fitting of traps, are as follows:

	Airborne Lead Concentration ug/m^3	
Current	Post Additive Reduction	Post Filters
2	1.8	0.96
2	1.8	0.77
2	1.8	0.54

Three values are given relating to the range of filter efficiencies shown in Table 6-6. The second strategy, lead reduction to 0.15 g Pb/l and subsequent fitting of filters, gives the following reductions in airborne lead concentrations:

	Airborne Lead Concentration ug/m^3	
Current	Post Additive Reduction	Post Filters
2	0.8	0.48
2	0.8	0.42
2	0.8	0.33

Again three values are calculated relating to the range of filter efficiencies given in Table 6-6. In all cases it is recognised that currently 0.2 ug Pb/m^3 is from sources other

than the combustion of leaded fuel and will remain unaffected by the above strategies. The effectiveness of the strategies is then:

Standard Man's Blood Lead Concentration

Ug Pb/100 ml

Current	Post Strategy
17.03	13.63 - 12.26
17.03	12.06 - 11.57

The range of blood leads relates again to the range of efficiencies of exhaust gas filters in Table 6-6. Hence, these latter two strategies give reductions in blood lead ranging from 3.4 - 5.46 ug Pb/100 ml. Table 6-7 shows the strategies together with the reduction and % reduction in blood lead which they bring about. The effectiveness of the strategies as given in this table will be related to the costs of each strategy, calculated in Section 7, to give the cost-effective ranking, in Section 8. At that point the effect of various factors, such as the range of reported efficiencies for lead traps, will be discussed in the light of their bearing on the cost-effective ranking of the strategies.

Table 6-7

Strategy	Standard Man's Blood Lead Concentration ug/100 ml Reduction	% Reduction
1) Total elimination of lead additives from gasoline.	5.89	34.6
2) Reduction of lead additive concentration from 0.45 g Pb/l to 0.4 g Pb/l	0.65	3.8
3) Ditto from 0.45 g Pb/ l to 0.15 g Pb/l	3.92	23.0
4) Fitting exhaust gas filters (lead traps) to automobiles	2.94-4.58	17.3-26.9
5) Reducing additive concentration to 0.4 g Pb/l (2) and fitting lead traps.	3.4 -4.77	20.0-28.0
6) Reducing additive concentration to 0.15 g Pb/l (3) and fitting lead traps.	4.97-5.46	29.2-32.1

Also see Figure 6-6.

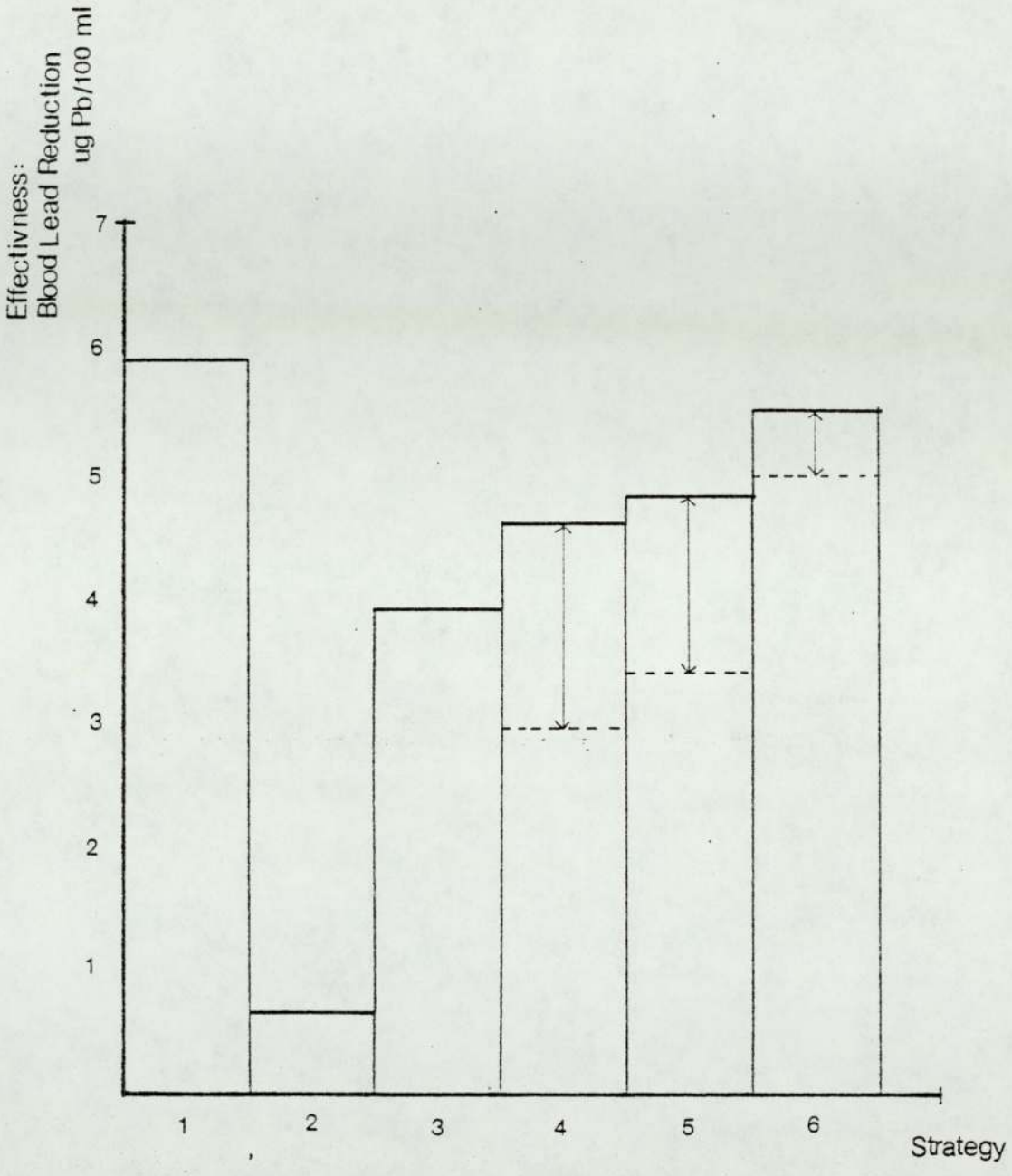

Effectivness:
Blood Lead Reduction
ug Pb/100 ml
7
6
5
4
3
2
1
1
2
3
4
5
6
Strategy

Figure 6-6

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SECTION 7

The Costs of Strategies to Reduce Environmental Lead by Controlling Lead Emissions from Motor Vehicles

This Section deals with the costs of the strategies described in Section 4. The six options under consideration fall into two groups, according to the method they employ to reduce lead in the environment. The first three strategies involve the reduction or elimination of the lead antiknock additives currently added to gasoline and these form the first of the groups mentioned above. The fourth strategy reduces environmental lead, not by eliminating the lead compounds from the vehicles' fuel but by trapping the particulate lead in the exhaust system before it escapes into the atmosphere using a filter system, and forms the second main group. The final two options are combinatorial techniques which first reduce the quantity of lead added to gasoline and then further reduce lead emissions from the vehicles' exhaust systems using lead particulate traps. Table 7-1 gives a list of the strategies under consideration in this thesis.

Calculation of the costs of these strategies is made difficult by the wide variation in the manner by which different estimates of costs involved are obtained. An early estimate of the cost of reducing the lead content of gasoline was given by the U.K. Secretary of State for Energy in 1974. He stated that to reduce the lead concentration in gasoline from 0.64 to 0.55 g Pb/l would cost the country £10 million per annum if the then current quantities and qualities of gasolines were maintained[1]. This figure illustrates one of the difficulties in obtaining the costs of lead reduction or elimination; total costs are often cited without giving a breakdown of the contribution of the various

Table 7-1

Strategy 1:	The elimination of all lead additives from gasoline.
Strategy 2:	The reduction of lead additive concentration in gasoline from the current level of 0.45 g Pb/l to 0.40 g Pb/l.
Strategy 3:	The reduction of lead additive concentration in gasoline from 0.45 g Pb/l to 0.15 g Pb/l
Strategy 4:	Fitting all vehicles with efficient lead particulate traps in the exhaust system.
Strategy 5:	The reduction in lead additive concentration of Strategy 2, 0.45 - 0.4 g Pb/l, combined with the fitting of exhaust gas filters.
Strategy 6:	The reduction of lead additive concentration of Strategy 3, 0.45 - 0.15 g Pb/l, combined with the fitting of exhaust gas filters.

cost components, primarily production and investment costs in the above case. Furthermore the accuracy of such figures is open to doubt because the methodology employed to obtain the various cost elements is rarely given; e.g. were all costs calculated to one monetary value, such as 1976 dollars? In this study, wherever possible all costs were obtained or converted to, the same monetary units. It is also important, with respect to gasoline costs to obtain all values from European standards, due to the differing production methods in Europe as opposed to the U.S.A., i.e. in Europe 20-25% of the barrel of crude oil is produced as gasoline as opposed to 50% in the U.S.A. Throughout this section, all cost calculations assume that European data may be applied to the U.K. situation, as data is usually available for a typical European refinery.

Reviewing the available literature suggest the following costs for Strategy 1. Table 7-2 shows the costs suggested by Hawkes in 1971[2] which would be incurred if lead free gasoline was to be introduced in the U.K. These costs assume the maintenance of then current gasoline octane quality and the added gasoline cost represents the costs of the additional processing required to do so, which is separate from the additional crude oil requirement. What is not included is the cost of the additional crude oil and processing required to compensate for the loss of the lead-road bonus - it is estimated that a fuel currently leaded to 100 RON would have to be replaced by a lead free fuel of 103 RON standard. The Nett Present Value of these figures, assuming a 10% rate of return is £1514 x 10^6, the then current exchange rate being £1 = $2.5[3].

Table 7-2

Additional Costs of Lead Removal[2]

Year	Cost	Amount
1980	Added Gasoline Cost	$193 x 10^6 p.a.
1980	Added Refinery Investment	$555 x 10^6
1980	Added Cost of Crude Oil	$130 x 10^6 p.a.
	Predicted Gasoline Consumption for the Year 1980	215 x 10^6 barrels equivalent to 20 x 10^6 tonnes

Nett Present Value Calculation

For Above Costs; 10% Discount Rate

Amount		NPV
$193 x 10^6 p.a.		$1930 x 10^6
$555 x 10^6		$555 x 10^6
$130 x 10^6 p.a.		$1300 x 10^6
	NPV Total Cost	$3785 x 10^6

The CONCAWE[3] study published in 1973 cites figures from the U.K. Department of Trade and Industry for the costs of reducing and eliminating lead from gasoline, using a base concentration of 0.84 g Pb/l. These figures, produced in 1971, are shown in Table 7-3. It is possible to calculate the cost of eliminating lead from gasoline from a base case of 0.45 g Pb/l using Columns 3 and 5 of this Table. However, there are no figures available for the calculation of the additional production costs, and only the additional refinery investment may be found. The figures cited relate to an exchange rate of £1 = $2.5, and an assumed inflation rate of 5% per annum. Column 3 relates to the year 1974 and, correcting this for inflation to 1980, gives the following additional refinery investment cost for the elimination of lead from gasoline, from a base case of 0.45 g Pb/l:

Total Additional Refinery Investment

$433 to $523 x 10^6

As may be seen, however, the above figures are similar to the additional refinery costs suggested by Hawkes[2]. In this case no calculation can be made for the additional production cost due to the absence of a value for this cost element in Column 5 of Table 7-3. These two studies are the only ones dealing exclusively with the cost of lead free gasoline. If it is assumed that the agreement between the D.O.T.I.'s and Hawkes' figures for the additional refinery investment cost is an indicator of the accuracy of all the latter's data, then the total cost for lead free fuel in the U.K. given by Hawkes may be taken as an acceptable figure, in 1971 monetary

	Base	1	2	3	4	5
Gasoline Lead Content						
Base Case: g/litre	0.84	0.84	0.84	0.84	0.84	0.84
Reduced Level: g/litre	0.84	0.63	0.50	0.45	0.15	0
Research Octane No.						
Premium	101-98	93.0	98.0	98.0	98.0	98.0
Regular		92.0	92.0	92.0	92.0	92.0
Pool		96.2	96.2	96.2	96.2	96.2
Market Split: Prem./Reg.	70/30	70/30	70/30	70/30	70/30	70/30
Production: 10^6 t/year	14.0	14.0	14.0	14.0	14.0	14.0
Additional Refinery Investment						
Total: $ 10^6	-	12.5-25.0	25-50	50-75	189-226	500-625
$/ton Annual Capacity	-	0.9-1.8	1.8-3.6	3.6-5.4	13.4-16.0	36.0-45.0
Additional Production Cost						
$/t	-	0.8	-	3.0	7.5	-
Reference Year	1971	1971-1974		1974	1976	1980

D.O.T.I. Figures

Table 7 - 3

From Hopper, 1973

values. Also, as there is no other complete data available, if a monetary value is required as in this case it is, there is no alternative but to employ the above value. Since 1971, however, there has been a 180%[4] increase in monetary values, up to 1976, and so the NPV to 1976 of Strategy 1 is 1.06×10^{10}. Costs relating to all strategies will be expressed in 1976 dollars to facilitate their comparison from literature sources. There are a number of criticisms which may be levelled at the above estimate. The main objection is that, having been first calculated in 1971, the figures do not take into account the 1973 OPEC price increases and furthermore U.K. inflation rates were greater than the 5% per annum employed. These early figures have been converted to 1976 monetary values using information contained in the CONCAWE study[4], but it was decided to recalculate the cost of Strategy 1 from a different approach to determine if a consistent value could be obtained. Both Hawkes[2] and the DOTI[3] agreed that the Additional Investment Cost, in 1980 calculated in 1971 using a 5% inflation rate, is of the order of $\$525 \times 10^6$. From this a 1976 estimate of the above value would have been $\$432 \times 10^6$, equivalent to $\$1209 \times 10^6$ in 1976 dollars. Investment costs were much less affected by changes in oil prices than were production costs, particularly crude oil prices. Hence, a further estimate of the cost of Strategy 1 may be obtained using the original Investment Cost data, suitably corrected, and modifying the Production Costs to take account of more recent crude oil prices, 1976 figures, as follows.

The extra annual operation costs for manufacturing lead free gasoline, from Hawkes would be $\$445 \times 10^6$ in 1976. It has been estimated that to reduce the lead additive concentration in gasoline from 0.45 g Pb/l to 0.15 g Pb/l, or less, would require a 3 to 8% increase in crude oil consumption. For lead free fuel a worst-case assumption would be at least the 8% figure and hence the U.K. would have consumed an additional 7.8×10^6 tonnes of crude oil over the 97.8×10^6 tonnes actually consumed during 1976[6]. At a 1976 cost of $15 per barrel of crude oil, this would have resulted in an additional cost of $\$866 \times 10^6$, per annum in 1976. Again using a discount rate of 10%, the NPV to 1976 of Strategy 1 would be, by this latter approach, $\$1.4 \times 10^{10}$. This is a higher cost for a smaller production compared with the 1980 forecast by Hawkes, the difference being due to the increase in oil prices which occurred in 1973, after the publication of the Hawkes paper in 1971. This latter value, $\$1.4 \times 10^{10}$, is a more realistic estimate of the cost of lead free gasoline and also relates to the elimination of lead from a base case of 0.45 g Pb/l, although the investment and manufacturing costs relate to a base case of 0.53 g Pb/l. However, these latter two cost elements are much smaller than the additional crude oil cost and are much less affected by the price of crude oil, and the small difference in lead additive base level.

Strategies 2 and 3 are concerned with the reduction as opposed to the elimination of lead antiknock additives in gasoline. In both these cases it is assumed that, as with

Strategy 1, current gasoline octane quality standards are maintained and that there is a 70:30 split between 4-STAR and 2/3 STAR fuel grades. This concurs with the Institute of Petroleum data[5] presented in Table 6-2, Section 6. The recent CONCAWE study[4] suggests the following breakdown of costs for Strategy 2 in the EEC:

	1976 $/annual ton
Additional Investment Costs	1 to 4
Additional Production Costs	1.1 to 2.8

According to the Institute of Petroleum, in 1976 16.9×10^6 tonnes of gasolines were used in the U.K. Thus the total costs of Strategy 2 would be:

Additional Investment Cost	$\$16.9 - 67.6 \times 10^6$
Additional Production Cost	$\$18.5 - 47.3 \times 10^6$

Again using a 10% Discount rate the NPV of this strategy would be $\$201.9 - 540.6 \times 10^6$. For Strategy 3 the same study suggests the following breakdown of costs:

	1976 $/annual ton
Additional Investment Cost	15 to 28
Additional Production Cost	9.5 to 14

The total costs for this strategy are therefore

Additional Investment Cost	$\$253.5$ to 1142.4×10^6
Additional Production Cost	$\$160.6$ to 236.6×10^6

The NPV for the above strategy, to 1976 is $\$1859.5$ to 3508.4×10^6

A second estimate of the Cost of Strategy 3 may be obtained from Table 7-3, Columns 3 and 4. The costs of Column 3 have to be re-estimated to the year 1976 using the 5% inflation rate given in Hopper[3]. From these values the following figures are obtained:

Total Additional Investment Cost, 1976	\$132.9 to 143.3 x 10^6
Annual Additional Production Cost, 1976	\$4.2/ton

This study assume 14.0 x 10^6 tons of gasoline to be produced in 1976 giving total costs of:

	\$ x 10^6
Additional Investment Costs	132.9 to 143.3
Additional Production Costs	58.8

This gives a NPV of \$720.9 to 731.3 x 10^6 which has to be converted from 1971 monetary values to 1976 monetary values due to the 180% value change stated to have occurred. This suggests a NPV to 1976 of 1976 \$2018.5 to 2047.6 x 10^6. This figure is within the range of those figures suggested by the CONCAWE study[4]. If the 14 x 10^6 tons gasoline production cited above is replaced by the 16.9 x 10^6 tons gasoline production for 1976 reported by the Institute of Petroleum, then the estimate of the NPV becomes \$2359.6 to 2388.7 x 10^6, again within the range of the 1977 CONCAWE study.

With regard to the lead reduction strategies, it would appear that estimates calculated from recent data offer a wider spread of cost than earlier estimations. There may be a number of explanations for this:

1) It has become more difficult recently to calculate crude oil costs due to the possibility of non-market forces making unexpected alterations in the price; e.g. the OPEC decision of 1973.

2) Costs, other than crude oil prices, vary widely throughout the EEC, covered by the CONCAWE organisation, due to a large inter-country variation in inflation rates.

3) The lower end of the cost continuum may be made possible by the large scale introduction of up to date refinery technology, before the decision to reduce the lead concentration in gasoline, thus reducing the additonal cost of this decision.

4) The problem is affected by so many variables, the cost of refinery plant, crude oil, environmental legislation, engine design, gasoline market demand, gasoline quality requirements etc., that it is not feasible to narrow the cost down any further than was done by the studies cited.

The last of the above reasons would appear to be the most likely, although the others undoubtedly have an affect on the cost estimation for the lead reduction strategies. Table 7-4 shows the NPV's, to 1976, of the lead reduction and elimination strategies examined in this study. These are the values which will be cited in Section 8 in the calculation of the Cost-Effectiveness of each strategy.

Table 7-4

Strategy		NPV $ x 10^6
1	at least	14,000
2		201.9 - 540.6
3		1859.5 - 3508.4

The second group of strategies under consideration involve the use of lead particulate traps fitted to vehicle exhaust systems which prevent the escape of particulate lead to the environment. The TRRL report[7] estimated the additional cost of lead traps to be in the region of £8 to £12 per vehicle taking into account the increased durability and lifetime of filtered exhaust systems over those then currently fitted by the manufacturers. Allowing an exchange rate of £1 = $2.3, this represents a cost of $18.4 to $27.6. In 1975 there were 13.7×10^6 private vehicles licensed in the U.K.[7a]. The EEC car population is conservatively estimated to remain static with approximately 10% being replaced each year by new vehicles. For the purposes of cost calculation, the severest application of Strategy 4 is being considered; i.e. compulsarily fitting the entire vehicle population with lead traps within one year, $\backsim 14 \times 10^6$ vehicles in all. If exhaust system fitting facilities could cope with the entire car population in one year, all of these vehicles, should legislation require it, would have to be equipped with filtered exhaust systems at the full cost of $39.1 to $115 per vehicle. This is because these vehicles were already fitted with standard exhaust systems which would have to be replaced by filtered systems, as opposed to new vehicles which could be fitted with the filtered exhaust systems at time of manufacture , at the additional costs shown above. Hence, assuming all vehicles were required by law to fit the filtered exhaust systems, there would be an initial cost in 1976 of $547.4 to 1610×10^6 with a subsequent annual cost of $25.8 to £38.6

x 10^6. The NPV of Strategy 4 on the basis of the above costs would be \$805.4 to \$1996 x 10^6 in 1974 dollars.

The Ethyl S.A. study on lead trapping exhaust systems for the EEC[8] suggested the following costs for lead traps:

	1976
Additional Costs of Traps	\$ 8 to 13
Costs of Traps	\$17 to 26

Applying these figures to an estimated U.K. car population of 14 x 10^6, there would be an initial cost of \$238 to \$364 x 10^6, and an annual cost of \$11.2 to \$18.2 x 10^6, again in 1976 prices, for the U.K.

A further study on the costs of fitting lead trap exhaust systems to U.K. cars was carried out by the Associated Octel Company Limited[9]. Although not explicitly stated, as the report was published in 1977, it has been assumed that 1976 prices have been used, with an exchange rate of £1 = \$1.8[10]. This study breaks down filter costs by engine size as shown in Table 7-5. However, the Octel Study does not cite a cost for a standard exhaust system, only the differential cost for the filter systems. From Table 7-5 it can be seen that the range of the additional costs of lead trap exhaust systems, expressed in 1976 dollars, is \$6.0 to \$11.9, similar to the additional costs suggested by the Ethyl S.A. study[8]. Taking the actual cost of the lead trap exhaust systems to be that suggested by the Ethyl S.A. study, i.e. \$17 to \$26, there would be an initial cost of \$238 to \$364 x 10^6, with an annual cost of \$8.4 to \$16.7 x 10^6. Thus the NPV of Strategy 4 suggested by

Table 7-5

Engine Capacity litres	Additional Filter Cost (£) — Filter Type[a]			
	Axial Flow		Radial Flow	
		$		$
0 - 1.2	3.31	6.0	4.49	8.1
1.2 - 1.8	3.10	5.6	5.37	9.7
1.8+	4.61	8.3	6.63	11.9

[a] For a full description of Filter Types refer to Section 6.

the Octel report would be \$322 to \$531 x 10^6.

For the purpose of estimating the cost of Strategy 4, i.e. fitting all vehicles with lead trapping exhaust systems, it was decided to discount the costs suggested by the TRRL study because, as is apparent from Section 6, it in no way reflects the state of the art regarding lead trap design, and hence contemporary filter costs. This being the case, Table 7-6 shows the costs of Strategy 4. For the calculation of the cost effectiveness of this strategy in Section 8, the NPV will be from the lowest to the highest estimate, i.e. from \$322 to \$546 x 10^6.

With respect to Strategies 5 and 6, it will be seen that they are combinations of the various lead additive reduction schedules and the fitting of lead particulate traps. A conservative estimate of their cost would be the sum of the NPVs of the techniques being combined and these figures are shown in Table 7-7, together with the costs of the various other strategies. These will be combined with the effectiveness of the strategies, calculated in Section 6, to give the cost-effective ranking in Section 8.

Table 7-6

Strategy No.	Costs, $1976 x 10^6
4	350 to 546[8]
4	322 to 531[9]

Table 7-7

Strategy No.	Cost, $ 1976 x 10^6
1	14,000
2	201.9 to 540.6
3	1859.5 to 3508.4
4	322 to 546
5	523.9 to 1086.6
6	2181.5 to 5054.4

Also see Figure 7-1.

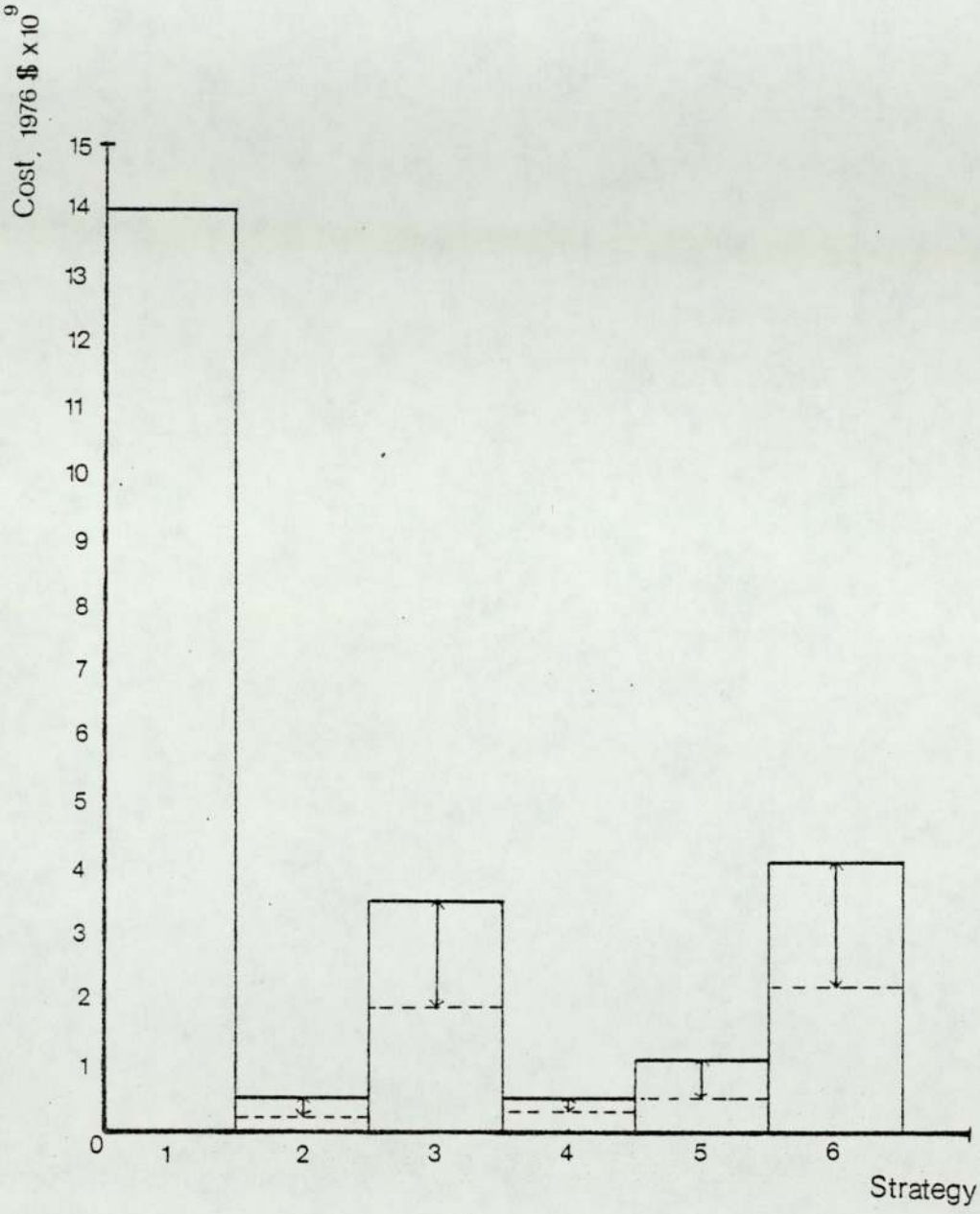


Figure 7-1

References

1) Adams W. E. and Gibson H. J., Cost-Effective Emission Control, Ethyl Corporation, Ferndale, Michegan, U.S.A.

2) Hawkes A.S., Gasoline, Lead and Air Pollution, World Petroleum, Vol. 42, Port 5, 1971.

3) Hopper W. C., Supplement to CONCAWE Report No. 12/72, The Problem of Gasoline Engine Exhaust Control, CONCAWE, The Hague, Holland, 1973.

4) Camarsa M. and Spencer E. H., Automotive Emission Regulations and their Impact on Refinery Operations, (Revised 1977), CONCAWE, The Hague, Holland, 1977.

5) U.K. Petroleum Industry Statistics, Institute of Petroleum, London, 1973 - 1979.

6) Digest of United Kingdom Energy Statistics 1977, HMSO, London, 1977.

7) TRRL, The Assessment of a Lead Trap for Motor Vehicles, Transport and Road Research Laboratory. Dept of the Environment, HMSO, 1974.

7a) Department of Transport, Transport Statistics Great Britain 1965-1975, HMSO, London, 1977.

8) Gibson H. J. and Wall H. H., Costs of Reducing Lead Automotive Emissions in the European Community, Ethyl S.A., Brussells, 1976.

9) The Associated Octel Co. Ltd., The Cost of Control of Lead Emissions; Lead Reduction Versus Exhaust Gas Filters, London, 1977.

10) Central Statistical Office, Financial Statistics, HMSO, London, 1976, 1977.

SECTION 8

Conclusions - The Cost-Effective Ranking of Strategies to Reduce Environmental Lead by Controlling Lead Emissions from Motor Vehicles

Cost-Effective Analysis (CEA) may be employed to resolve the conflict between the costs and effects, the two major decision criteria, of the strategies under consideration. The policy-maker may be said to have two goals, of equal importance; to conserve resources and to achieve a significant reduction in the population's blood lead concentration. For a given option, cost and effect are usually linked such that a greater effect requires a higher cost, although this relationship need not be linear as the data regarding the reduction of the lead additive concentration in gasoline and the costs of this reduction shows. In this Section the cost-effectiveness of each of the six strategies, listed in Table 8-1, which were considered in Sections 6 and 7 will be calculated. The data used to calculate these values is shown in Table 8-2. Before proceeding further, it is necessary to explain why, for some values, a range of results has been obtained whilst for others a single value has been found. The effect of each strategy was obtained using the model developed in Section 5. This model employed the currently accepted standard man values for the various parameters governing human, male, lead transport, although various values for these parameters are cited by different authors. However, the accuracy and validity of these latter estimates varies widely and accordingly it was decided to use internationally accepted standard man values. Hence, having used fixed values for the model's parameters, a discrete reduction in the lead additive concentration in gasoline, producing a theoretically discrete reduction in airborne lead level, leads to a discrete value for the strategy's effect. The

Table 8-1

Strategy 1	:	The elimination of lead additives from gasoline.
Strategy 2	:	The reduction of the lead additive concentration in gasoline from 0.45 g Pb/l to 0.4 g Pb/l.
Strategy 3	:	The reduction of the lead additive concentration in gasoline from 0.45 g Pb/l to 0.15 g Pb/l.
Strategy 4	:	Fitting all automobiles with lead particulate traps.
Strategy 5	:	Strategy 2 coupled with Strategy 4.
Strategy 6	:	Strategy 3 coupled with Strategy 4.

Table 8-2

Strategy	Effectiveness ug Pb/100 ml blood reduction achieved	Cost 1976 $ x 10^6
1	5.89	14,000
2	0.65	201.9 to 540.6
3	3.92	1859.5 to 3508.4
4	2.9 - 4.6	322 to 546
5	3.4 - 4.8	523.9 to 1086.6
6	5.00 - 5.5	2181.5 to 4054.4

range of effectiveness found for Strategies 4, 5 and 6 is due to the fact that each of these options involves the use of lead filters fitted to vehicle exhaust systems. The efficiency of these filters varies according to the type of trap design employed as described in Section 6. Hence, lead traps may produce a reduction in airborne lead concentration which lies within the range of the trap's efficiency, leading to a range of effectiveness being found for these strategies. With regard to costs, a wide range of estimates was taken from the literature and analysed to produce the costs shown in Table 8-2. However, there was one exception to this, Strategy 1. As detailed in Section 7, literature on the costs of eliminating lead additives from gasoline was found to be extremely limited. Using the most reliable and most recent estimates possible produced the figure shown in Table 8-2, it being considered counter-productive to produce a range of values from undeniably suspect data. More thorough studies were available relating to the other strategies and these were employed to produce the remaining costs in Table 8-2.

The initial cost-effective ranking was calculated and is given in Table 8-3. The histogram presented in Figure 8-1 shows how the six strategies under consideration compare directly against one another in terms of their cost-effectiveness. If the data, in Sections 6 and 7, regarding costs and effects is compared with Figure 8-1, it is apparent that some information has been lost. This is because data has been merged; i.e. a spread of cost-effectiveness was produced from the highest cost/lowest effect to the lowest cost/highest

Table 8-3

Strategy	Cost-Effectiveness ug x 10^{-10} Pb/100 ml reduction per $ 1, 1976
1	4.2
2	32.2 to 12.0
3	21.1 to 11.2
4	142.9 to 53.1
5	91.6 to 31.3
6	25.2 to 12.3

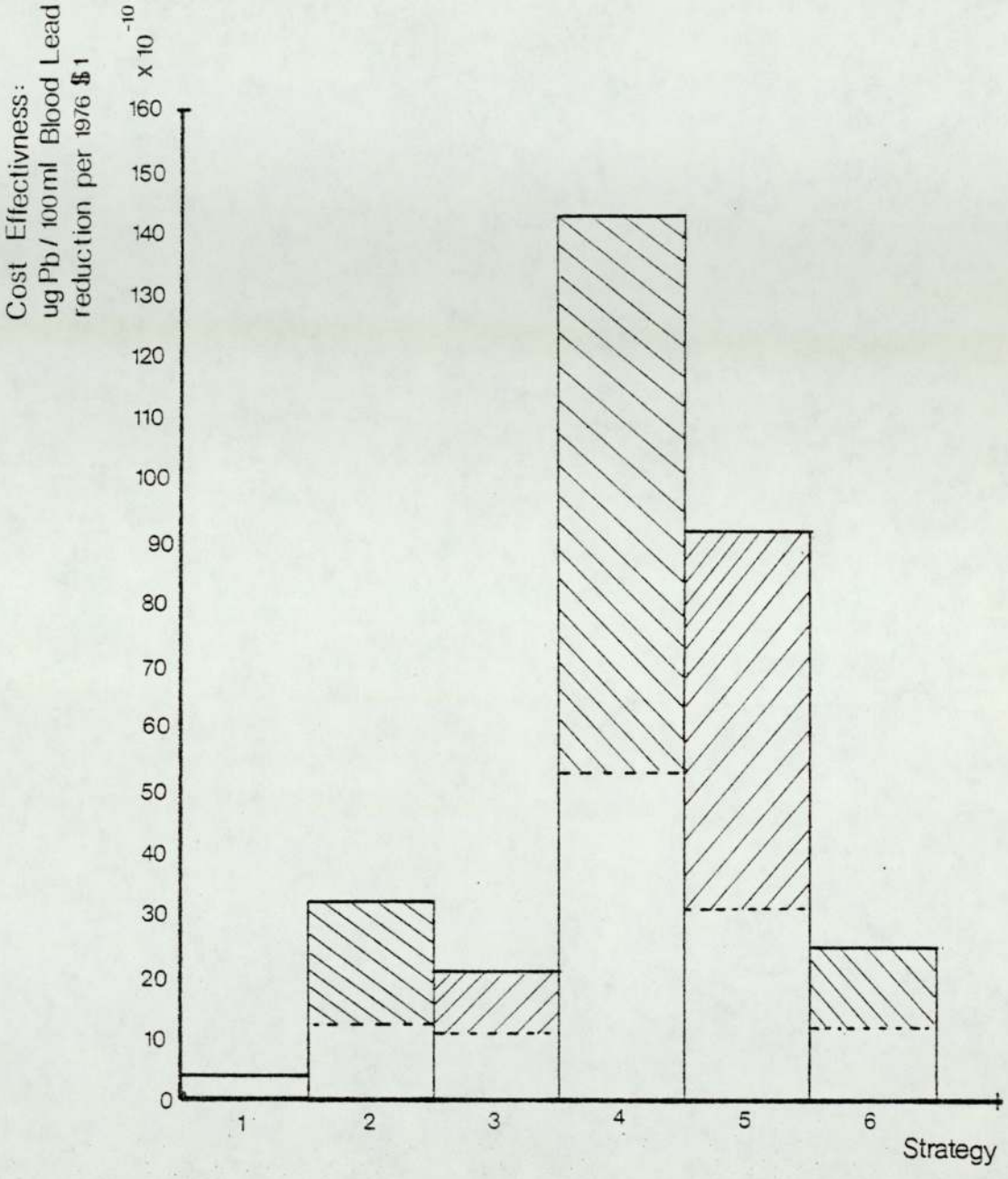
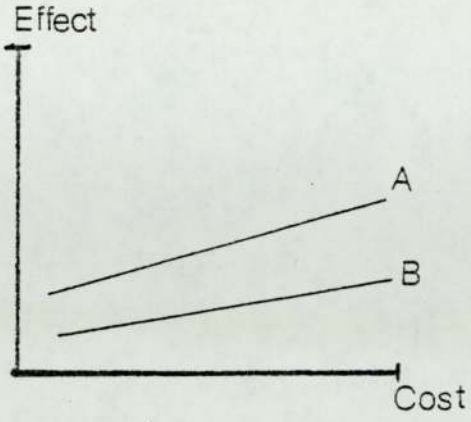

Cost Effectivness:
ug Pb/100ml Blood Lead
reduction per 1976 $1
x 10^-10
160
150
140
130
120
110
100
90
80
70
60
50
40
30
20
10
0
1
2
3
4
5
6
Strategy

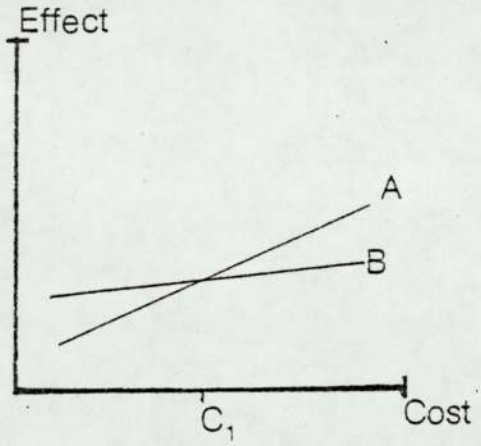
Figure 8-1

effect to produce the cost-effective ranges shown. In the light of current information regarding strategy costs and the efficiency of lead particulate traps, the cost-effectiveness of each strategy would be expected to lie somewhere within each range, although this point could only be determined accurately after the implementation of the strategy. Again the exception is Strategy 1 where paucity of data leads to a worst-case analysis producing a single value of cost-effectiveness.

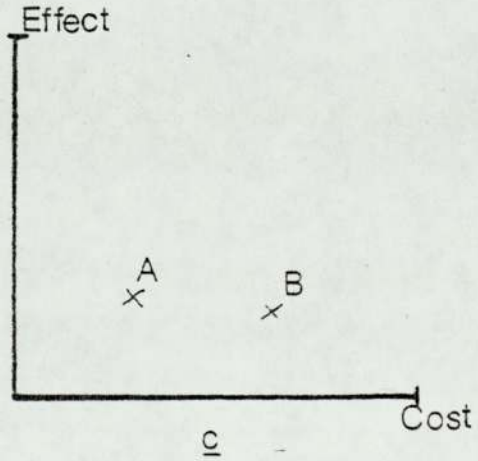
However, having produced the initial cost-effective ranking, a difficulty arises which is inevitable when employing Cost-Effective Analysis in this type of problem situation. If the relationships between the costs and effects of two strategies are as shown in Figure 8-2a, it is apparent that A is always more cost-effective than B, and that knowing that A has the greater cost-effectiveness allows the decision-maker to eliminate B. However, should the relationship between costs and effects be that shown in Figure 8-2b, then the cost-effectiveness of A is only greater than that of B at costs greater than C_1. Below C_1, the opposite is true, B is more cost-effective than A. Figure 8-2c illustrates the problem related to this study. Here, only one discrete value for the cost-effectiveness of each strategy is known, and so it is not possible to say whether the situation shown in Figure 8-2a or Figure 8-2b exists; i.e. although at the points shown, A has a greater cost-effectiveness than B, it is not possible to say which will be the more cost-effective at any other point. Furthermore, it is possible to envisage a



a



b



c

Figure 8 – 2

strategy which has a low effect for virtually zero cost. The cost-effectiveness of this strategy would appear to be relatively high although the strategy itself would be almost worthless in terms of its effectiveness in reducing the population's blood lead concentration. Thus in the situation described by Figure 8-2c it is necessary to consider not only the cost-effectiveness but also the costs and effects of each of the various strategies.

Returning to the six strategies under consideration, it must be noted that although a range of cost-effectiveness is shown for five of the options this does not represent a continuous function as shown in Figure 8-2a. All that can be said is that, were the strategy to be implemented, its cost-effectivenss would be found to be at some point within the range shown. Hence, the data is, in reality, a set of discrete points as shown in Figure 8-2c. In this situation, a difficulty arises if the strategies' ranking is analysed solely from Figure 8-1. Although this figure shows the cost-effectiveness of each strategy, it unfortunately does not give any indication of the actual costs and effects of the various options, the two prime decision criteria. This difficulty may be resolved by the use of the concept of Cost-Effective Domination. One strategy may be said to dominate another if it produces and equal, or greater, effect for an equal, or lower, cost. As is immediately apparent, this cannot be determined from Figure 8-1, although this figure does serve to illustrate the cost-effectiveness of the various strategies under consideration.

To determine which of the six strategies are dominant in cost-effective terms, the costs and effects were analysed as shown by the example in Figure 8-3. If Strategy A has a cost C_1 and an effect E_1, then its cost-effectiveness is E_1/C_1. However, it will be dominated by any strategy whose cost-effectiveness falls in the cosine quadrant of A, as illustrated in Figure 8-3; all strategies falling within this area giving a greater or equal effect for a lesser or equal cost. This eliminates problems such as strategies having apparently equal cost-effectiveness; e.g. Strategies 3 and 6, Figure 8-1. Figure 8-4 shows this latter approach applied to the six strategies discussed in this section. As can be seen, no strategy dominates Strategy 1; i.e. no other strategy falls inside the cosine quadrant of Strategy 1. However, due to its extremely high cost and the fact that it may produce an effectiveness only marginally superior to that of Strategy 6, its implementation must be regarded as improbable. Furthermore, due to the high cost of Strategy 1, an order of magnitude greater than the cost of any other strategy, the costs and effects of the remaining five strategies were replotted in Figures 8-5. From Figure 8-5 one may draw the following conclusions:

1) Strategy 4 dominates Strategy 2.

2) Strategy 4 may dominate Strategy 3.

3) Strategy 5 may dominate Strategy 3.

With respect to Strategies 5 and 6 there is an added complication due to the fact that these are combinatorial techniques.

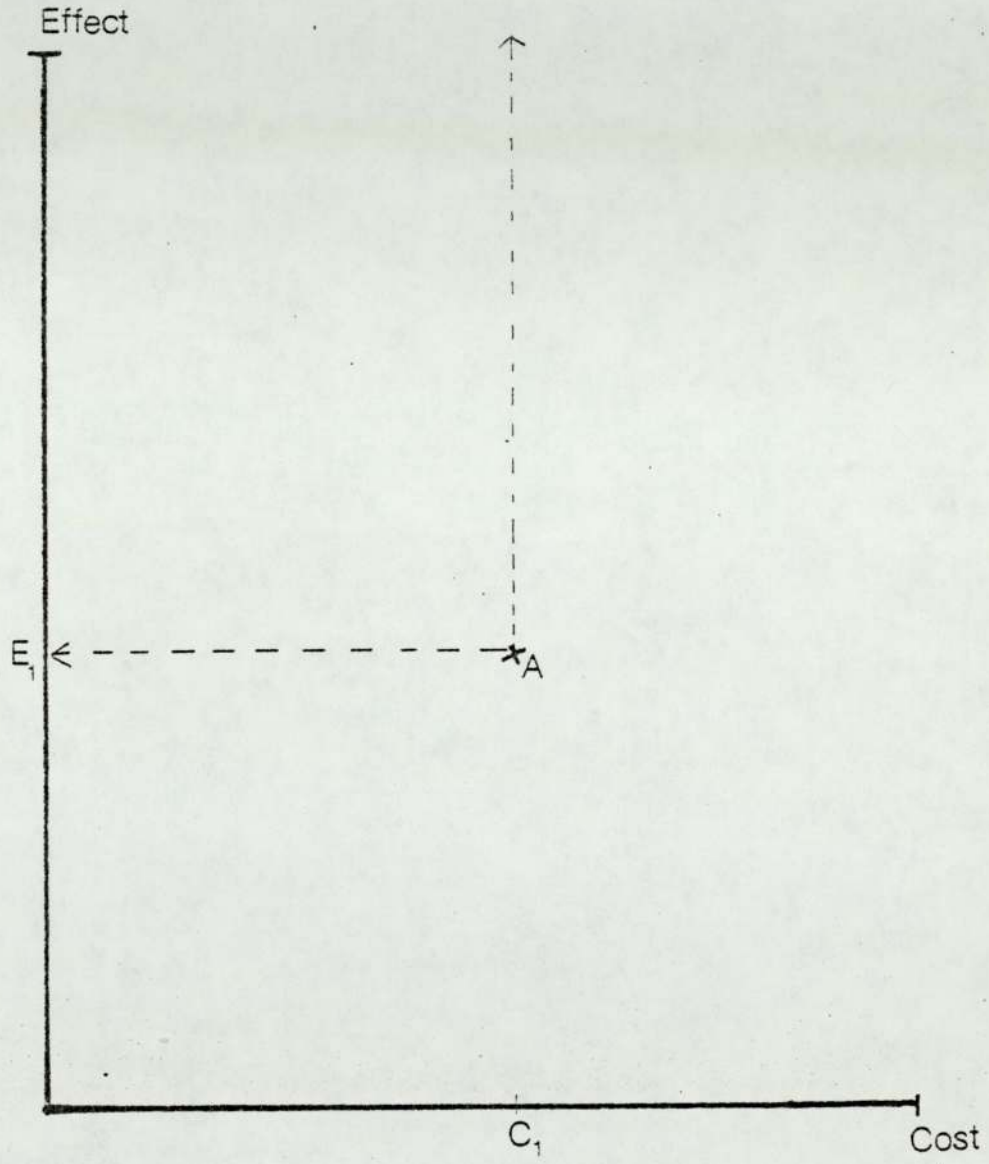


Figure 8-3

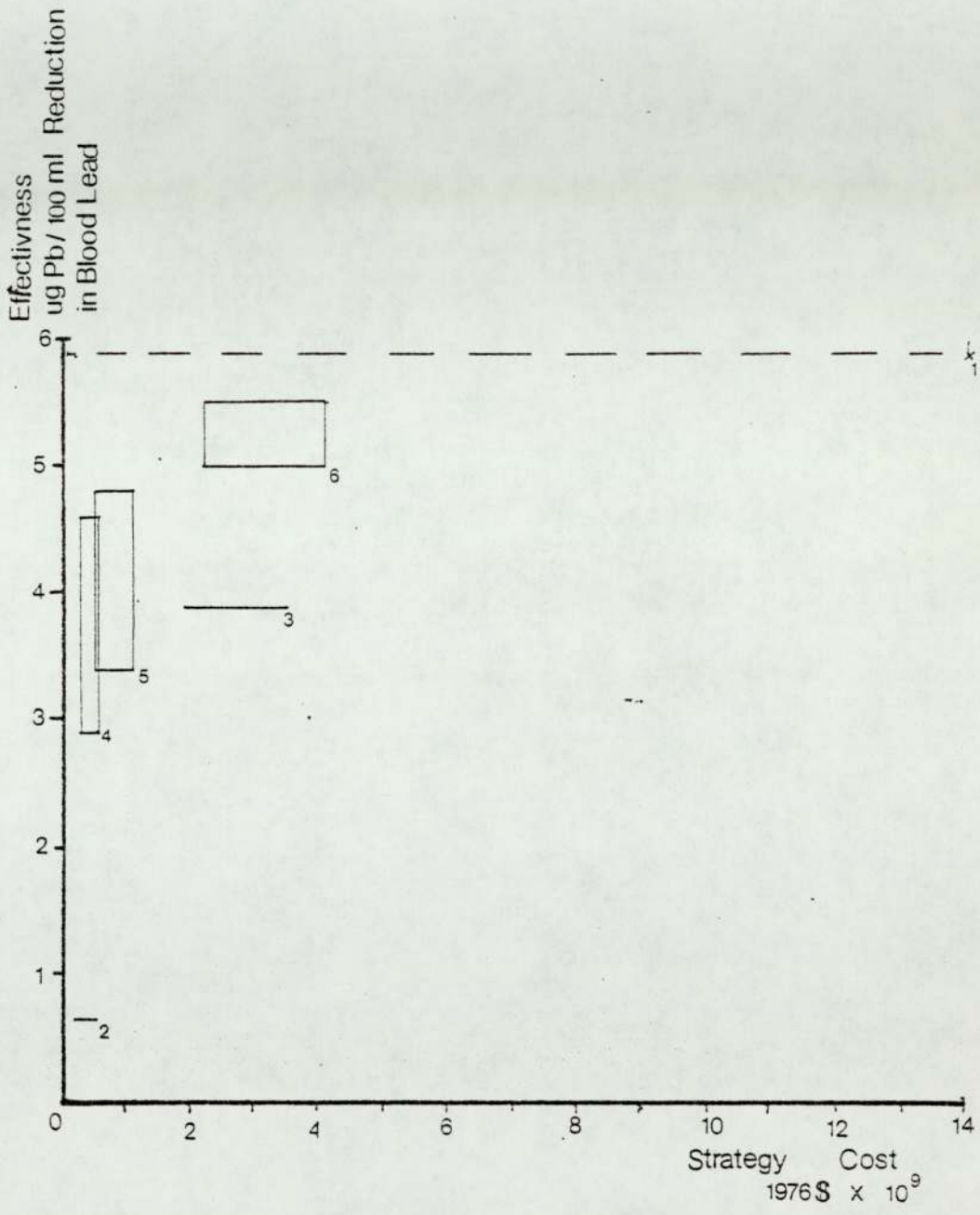

Effectivness
ug Pb/ 100 ml Reduction
in Blood Lead
6
5
4
3
2
1
0
2
4
6
8
10
12
14
Strategy Cost
1976 $ x 10^9
1
2
3
4
5
6

Figure 8-4

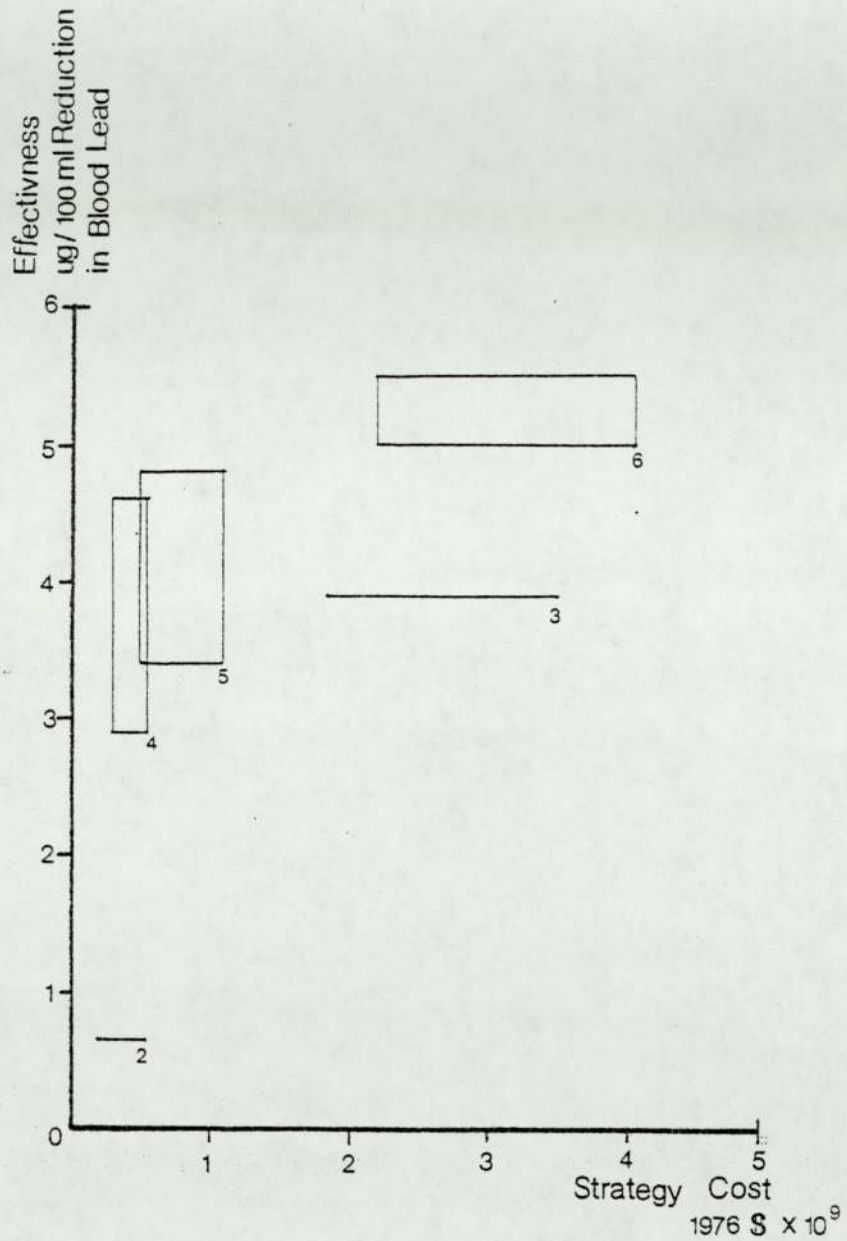

Effectivness
ug/100 ml Reduction
in Blood Lead
6
5
4
3
2
1
0
1
2
3
4
5
Strategy Cost
1976 $ X 10^9
2
3
4
5
6

Figure 8-5

This is best illustrated by an example:

From Figure 8-5 it may be seen that Strategy 6 may dominate Strategy 3. However, Strategies 3 and 6 are related by the fact that Strategy 6 is a combination of Strategy 3 and Strategy 4. Hence it is unrealistic to compare the lowest cost of Strategy 6 with the highest cost of Strategy 3 because, if the adoptions of Strategy 3 incurs its highest cost, then the extension of Strategy 3 to Strategy 6 will also incur this cost to which will be added the cost of Strategy 4; i.e. Strategy 6 must always cost more than Strategy 3, the difference being the cost of Strategy 4. A similar case holds true for Strategies 2 and 5. However, with regard to Strategies 4 and 5, an interesting situation would appear to exist where the two strategies overlap to a certain extent, although the overlap cannot, in fact, occur for similar reasons to those outlined above; i.e. Strategy 5 must always cost more than Strategy 4 because it consists of Strategy 4 coupled with Strategy 2 and likewise Strategy 5 must always be more effective than Strategy 4, due to the fact that it possesses the effect of Strategy 4 coupled with the effect of Strategy 2. Hence neither Strategy 4 nor Strategy 5 may ever dominate the other.

From the above it would appear that Strategies 2 and 3 may be discounted by the policy-maker because they are dominated by Strategies 4 and 5 respectively. Furthermore, Strategy 1 may be discounted in favour of Strategy 6 for, whilst the latter does not in fact dominate Strategy 1, its effect is virtually identical for approximately one third

the cost. Indeed it would be experimentally, extremely difficult to measure the difference in effectiveness between Strategies 1 and 6, shown in Table 8-2. Hence, CEA suggest Strategies 4, 5 and 6 as the dominant options for the basis of a control policy.

Although it is not possible to determine at what point the policy-maker would choose to decide that a strategy's cost's outweight its effects, the relationship, described above, between Strategies 1 and 6 illustrates that one strategy may effectively dominate another even if in fact this is not the case. The policy-maker would require some special reason for choosing Strategy 1 over Strategy 6 due to the fact that the former's high cost, three times that of the latter, would be expended to achieve a difference in effect too small to measure. Hence the policy-maker's desire to conserve his resources leads to Strategy 6 effectively dominating Strategy 1, at present. However, this domination, as is the case with certain other strategies, is dependent on external factors which are determined by the time at which the policy-maker assesses the situation; e.g. OPEC either raising the price or curbing supplies of crude oil, or future research indicating that the control of automotive lead emissions will not, in fact, have any significant effect on the population's blood lead concentration. At present those strategies which involve a lower increase in crude oil consumption than others tend to dominate these latter options. This dominance would be expected to increase with the passage of time due to the inevitable rise in cost of crude oil associated with its

increasing scarcity. As this process occurs two strategies will tend to predominate over the remainder, Strategies 4 and 5. These are two highly cost-effective options, as shown in Figure 8-1, and are also dominant as shown in Figure 8-5. This is due to the fact that they employ a relatively cheap approach, the use of lead traps, to obtain a high value of effectiveness. Strategy 5 also employs a small reduction in lead additive concentration, from 0.45 g Pb/l to 0.40 g Pb/l. However, the cost of this strategy, in conjunction with Strategy 2, will be the least affected by rises in the cost of crude oil, in comparison with other strategies involving the reduction or elimination of lead additives from gasoline. As mentioned in Section 4, the reduction in lead additive concentration envisaged by Strategy 5 is due to be implemented by the EEC nations in 1980. Hence, in the light of this proposed legislation, if action to control automotive lead emissions is taken after 1980, Strategy 4 would appear to be in a dominant position as the lead reduction schedule of Strategy 5 will already have taken place.

There is a further advantage associated with the adoption of a filter based strategy as opposed to one requiring the reduction or elimination of lead additives from gasoline. This is related to the flexibility of the decision taken by the policy-maker. With strategies involving lead additive reduction or elimination large capital sums are required for investment before their implementation. This is illustrated in Section 7 with respect to the costs of Strategies 1, 3 and 6. Should it later be found necessary to reverse the original

policy decision, in the light of the results of future research or crude oil scarcity, this capital investment is irretrievably lost, being bound up in now redundant equipment. The introduction of lead particulate traps on the other hand requires very little capital investment, the traps being manufactured by the existing exhaust system industry. They also have a much lower annual cost than strategies involving lead additive reduction, and hence Strategies 4 and 5 permit major alterations in policy regarding environmental lead control, including the abandoning of the control of automotive lead emissions, at some time in the future without incurring a severe capital cost penalty. Such a future as described above is not as unrealistic as it at first might appear. Petroleum will inevitably become scarcer, and hence more expensive, and so it may be decided to reduce the population's exposure to lead by the more difficult approach of controlling lead in the diet.

As it is inevitable that any lead control policy eventually implemented will have to be government initiated, an important factor will be the emphasis placed on environmental issues by the government of the day. Although it is not possible to know what this attitude will be, it may be possible to identify some of the factors which might influence it. Groups from the environmentalist lobby, such as the Campaign Against Lead In Petrol (C.A.L.I.P.), who desire the total elimination of lead additives from gasoline, Strategy 1, would be in a position to exert considerable pressure should future research indicate that current fears regarding the neurological effects

of environmental lead exposure on children to be well founded. In this instance, Strategy 1 might well be adopted despite its poor cost-effective performance. On the contrary, should it be necessary to implement severe conservation of petroleum products, including gasoline, perhaps due to action by the OPEC organisation, a control policy for atmospheric lead would have to be based on Strategy 4, as it does not require any increase in petroleum consumption associated with its implementation.

As has been previously mentioned, an important factor influencing the adoption of any strategy will be future research into the whole field of environmental lead pollution, and in particular into the effects of lead exposure on suspected risk groups. The importance of the outcome of such research is shown by the fact that much of the pressure for current research comes from alleged effects on these suspected risk groups. A full description of these risk groups, primarily young children, together with sub-clinical effects attributed to environmental lead exposure was given in Section 1, effects ranging from the impairment of the central nervous system to hyper-activity. However, it was also stated that conclusive evidence for these effects may never be found. This lack of certainty with regard to low level effects resulting from environmental lead exposure will undoubtedly promote further research in this area as suggested above. Depending upon the outcome of this research, a decision may have to be taken on whether or not to control lead emissions from motor vehicles. Due to the fact that it is unlikely that the government of the

day will adopt a 'money-no-object' approach to the problem, some form of cost-benefit or cost-effective analysis is inevitable. This is particularly so in the light of on-going price increases, especially that of crude oil due to its increasing scarcity. The necessity of taking into account the actual costs of a particular strategy, or more properly of a control policy, leads to comparisons between the costs and effects of any control strategies under consideration. In the present state of ignorance regarding the degree, if any, of harm caused to the population as a whole by environmental levels of lead exposure, from any source, prime consideration is given to the cost of control. If at some time in the future measurable harm is shown to be directly related to environmental lead, then the emphasis may shift to the effects side of the above balance.

A less likely future would be the replacement of lead alkyl based antiknock agents by another compound, equally effective at preventing the phenomenon of knock, both high- and low-speed, which does not cause the pollution currently brought about by lead. However, since the discovery of tetra-ethyl and tetra-methyl lead over fifty years ago, only one other cost-effective gasoline antiknock compound has been formulated. That is methycyclopentadienyl manganese tricarbonyl (MMT) and the use of this compound is limited to relatively low concentrations by cost. To date it has only been employed in the U.S. to trim the RON quality of lead-free gasoline by one or two octane numbers, as opposed to the five or six octane numbers provided by lead antiknock additives.

This lead-free fuel is for use in vehicles equipped with catalytic exhuast reactors which are unaffected by MMT. Should MMT, or another gasoline antiknock agent which is found to be non-polluting, be developed to the point where its use would be competitive with that of organo lead based coumpounds, the cost-effective domination developed in this section would become obsolete due to the fact that the new additive could simply be substituted for the lead currently in use. This would result in only the more dominant strategies for the control of lead emissions being considered; i.e. the use of lead particulate traps, where their cost would be less than that of the new antiknock agent's introduction. However, at present this appears to be most improbable.

The cost-effective domination would also be altered if there were to be a major change in the passenger vehicle design concept to facilitate the use of lead-free fuel with an octane quality equivalent to that of current 2-STAR gasoline. This was discussed in Section 7, the chief advantage of this approach being that the U.K. refineries would be able to satisfy the total gasoline demand with fuel of the above octane quality without major plant or process alterations. However, it must be noted that to maintain current vehicle standards of comfort, performance and fuel economy the change in the vehicle design concept would be extremely large and hence very costly to achieve. Information, if any currently exists, possessed by the automobile industry regarding such design changes would be of a highly confidential nature due to competition within the industry. Hence, an accurate

assessment of the impact of this approach on the cost-effective ranking is impossible. The best that can be said is that the domination would change dramatically due to the major alteration of its terms of reference. The lead free option would become much more viable than at present tending to predominate all other strategies, with the proviso that the costs of designing the new generation of motor vehicles are considered separately from the costs of the strategy.

To sum up, the cost-effectiveness is seen to be markedly affected by two types of changes in its frame of reference. Firstly, changes in factors or values which directly affect the strategies' domination; e.g. OPEC increasing the price of crude oil or research suggesting that human absorption of lead through the lungs is several times greater than currently envisaged, and secondly changes in those factors indirectly affecting strategies; e.g. the introduction of new vehicle propulsion technology which did not require the use of leaded fuel such as diesel engines or low compression ratio spark ignition engines or the introduction of a new gasoline anti-knock compound eliminating the need to employ lead additives and hence the need for the control of automobile lead emissions.

The results of this study, the strategies' cost-effectiveness may be seen as complementary to the analytical research carried out into the effects of environmental lead exposure. Work in this latter field is of major importance particularly in obtaining confirmation of the sub-clinical effects of lead with respect to risk groups such as pre-school children. However, the results of such work do not of themselves suggest

a practical solution to the problem of environmental lead. This policy study on the other hand compares and contrasts prospective techniques for the reduction of lead concentrations in the environment in terms of their relative costs and effects, the only feasible measure. It is therefore apparent that action regarding environmental lead pollution will be the result of the combination of the above two aspects of research into the topic, and thus the role of the policy-maker will be to examine the results and implications of the clinical research, with respect to the public's health and welfare, and then develop a suitably cost-effective control policy in the light of this evidence. However, as previously mentioned, the actual policy which should, or would, be implemented cannot be determined within this study due to the nature of the governmental decision making process within the U.K. Hence, although the conclusions of this study do not provide a solution to the problems of environmental lead, they provide information essential to the derivation of such a solution.

A P P E N D I X I

Calculation of Derived Parameters and Model Test Procedure

1. Calculation of Lung - Gut Transfer Coefficient, F.

From Lee et al., 1971[1] , 5% of the urban lead aerosol has a mean equivalent diameter $\geqslant$ 0.5 u.

The mean retention rate for larger particles, assumed in this case to relate to all particles whose dimaeter is greater than 0.5 u is given in Danielson, 1970[22], to be 80-95%, mean 87.5%, for a mean equivalent diameter of 5 u.

Assuming that all particles of 0.5 u diameter, and above, which are retained in the lung are subsequently transferred to the G-I Tract, then:

F = % age of aerosol $\geqslant$ 0.5 u x mean retention factor for larger particles

F = 0.05 x 0.875

= 0.04375

= 0.044

F = 0.044

2. Calculation of λ

λ is equal to $\frac{0.693}{T_{\frac{1}{2}}}$ where $T_{\frac{1}{2}}$ is the half-life of lead in blood, from Chamberlain et al., 1978[2] , or it is equal to $\frac{1}{T}$ where T is the mean life of lead in blood, from Rabinowitz et al., 1973[5] and Rowe, 1976[3].

2. Cont Reported values for the half-life of lead in blood, $T_{\frac{1}{2}}$.

Days	
16	Chamberlain et al., 1975 [4]
18	Chamberlain et al., 1978 [2]
26)	Griffin et al., 1975 (From
)	
28)	Chamberlain et al., 1978 [2])

Reported values for the mean life of lead in blood, T

Days	
27	Rabinowitz et al., 1973 [5], 1974 [6]
36	Rabinowitz et al., 1976 [7], 1977 [8]

From the cited values of $T_{\frac{1}{2}}$:

Days $T_{\frac{1}{2}}$	Days $\overline{T_{\frac{1}{2}}}$	λ
16		0.043
18	22	0.039
26		0.027
28		0.025

From the cited values of T:

Days T	Days $\overline{T}$	λ
27	31.5	0.037
36		0.028

These results give a mean value for λ:

$$\bar{\lambda} = 0.033$$

This results gives a value of T = 30 days, and $T_{\frac{1}{2}}$ = 21 days, which agree well with the means of the measured values of T and $T_{\frac{1}{2}}$.

Model Validation

From Tsuchiya et al., 1975[9]

	Measured		Modelled
	Airborne lead ug/m^3	Blood lead ug/100 ml	Blood lead ug/100 ml
1	0.024	17.0	16.1
2	0.198	16.8	16.7
3	0.413	16.7	17.4
4	0.507	15.9	17.7
5	0.746	18.8	18.5
6	1.302	20.6	20.3
7	1.011	20.3	19.3
8	0.998	17.5	19.3
9	0.791	18.3	18.6

The oral lead intake for Japanese adults was estimated by Horiuchi (1965[10]) to be between 230 ug and 320 ug per day, giving a mean lead intake for PbG of 275 ug/day.

This data gives a value to t of

$t = 0.545$ with 8 degrees of freedom,

$P < 0.05$

This indicates there is no significant difference between the output of the model and the measured data.

From Goldsmith and Hexter, 1967[11]

	Measured		Modelled
	Airborne lead ug/m^3	Blood lead ug/100 ml	Blood Lead ug/100 ml
1	0.12	12	17.9
1*	0.12	20	17.9
2	0.5	16	19.1
3	1.0	13	20.8
4	1.0	21	20.8
5	1.9	19	23.7
6	2.2	19	24.7
7	2.4	24	25.3
8	2.1	25	24.4
9	3.8	30	29.9
10	4.2	31	31.2
11	5.2	21	34.5
12	5.5	31	35.5
13	6.3	30	38.1

1* represents the data cited by the Ethyl Corporation, , and the t-test was performed for both data sets.

Dietary exposure to lead was presumed to be 300 ug/day as assumed by Goldsmith and Hexter. (Regression analysis of their data according to their model suggests a 315 ug Pb/day dietary exposure).

Using Goldsmith and Hexter's original data a value of:

t = 3.603 with 12 degrees of freedom is obtained.

Using the modified first point, 1*, as proposed by the Ethyl Corporation gives a value of:

t = 2.861 with 12 degrees of freedom.

Both these results indicate that there is a significant difference between the model's output and the measured blood lead concentrations; i.e. the model does not fit this data, under the given constraints.

However, if the dietary exposure is reduced to 250 ug/day, a more reasonable value in the light of recent research[18], a value of:

t = 1.06, with 12 degrees of freedom is found.

This result indicates there is no significant difference between the model's output and the measured blood leads (i.e. under the new dietary constraint, the model fits the data) at the $p < 0.05$ level.

From the Seven Cities Study, Tepper et al., 1972[12].

	Measured		Modelled
	Airborne lead ug/m^3	Blood lead ug/100 ml	Blood lead ug/100 ml
Location			
Los Alamos	0.17	14.9	6.38
Okeana	0.32	15.7	6.87
Houston	0.85	12.5	8.61
Port Washington	1.13	15.3	9.53
Ardmore	1.15	18.0	9.59
Lombard	1.18	13.9	9.69
Washington D.C.	1.19	19.2	9.72
Rittenhouse	1.67	20.5	11.29
Bridgeport	1.76	17.6	11.59
Greenwich Village	2.08	16.6	12.63
Pasadena	3.39	17.5	16.92

Tepper and Levin calculated that dietary exposure was of the order of 100 ug Pb/day[12].

These results give a value of t of:

$t = 7.207$ with 10 degrees of freedom

This result indicates there is a significant difference between the measured blood lead values and the model's output. However, there are a number of factors which account for this variation. Firstly the TPU model is calibrated for a standard man and all the subjects in the Seven Cities Study[12] were female. Secondly, the study reports a male-female blood lead differential for a smaller study group - male blood leads were consistently higher than female blood leads, measured at the same location. This latter point suggests that the metabolism

of lead may vary from the male to the female, but Tepper and Levin can offer no explanation for the phenomenon. Taking these two points into account, it is not therefore surprising to find that the model does not provide a satisfactory fit to this data.

From Azar et al., 1975[13]

Measured		Modelled
Airborne lead ug/m^3	Blood lead ug/100 ml	Blood lead ug/100 ml
0.81	16.4	15.5
1.01	13.8	16.1
2.62	22.4	21.4
3.06	19.9	22.8
6.10	24.6	32.8

The above data was derived from air and blood lead measurements on 130 male subjects at five locations. The air lead values were obtained using personal air samplers. Analysis of this data indicates a dietary lead exposure of 220 ug Pb/day, and this value was substituted for PbG. This data gives a value of:

$t = 1.371$ with 4 degrees of freedom

This result indicates that there is no significant difference between the measured blood leads and the model's output at the $p < 0.05$ level.

A P P E N D I X II

The assumption implicit in all statistical models is that the form of the model, e.g. linear or semi-log, will fit all data available. The models reviewed in this section are shown plotted against their original data and are tested against other available results from epidemiological studies. These results are shown in the following tables, Table II-8 showing the correlation coefficients for the various models against the epidemiological studies cited in this section. The linear relationship was suggested by the Ethyl Corporation[14], but only in the context of highlighting possible errors in the Goldsmith-Hexter model. It is included in Table II-8 for reference purposes only. The Knelson model[15] is not tested here as there is no suitable data available for that purpose. Figures II-1 and II-2 show the two statistical models for which orginal data is available, the Goldsmith-Hexter model[11] and the NAS model[16], plotted against this data.

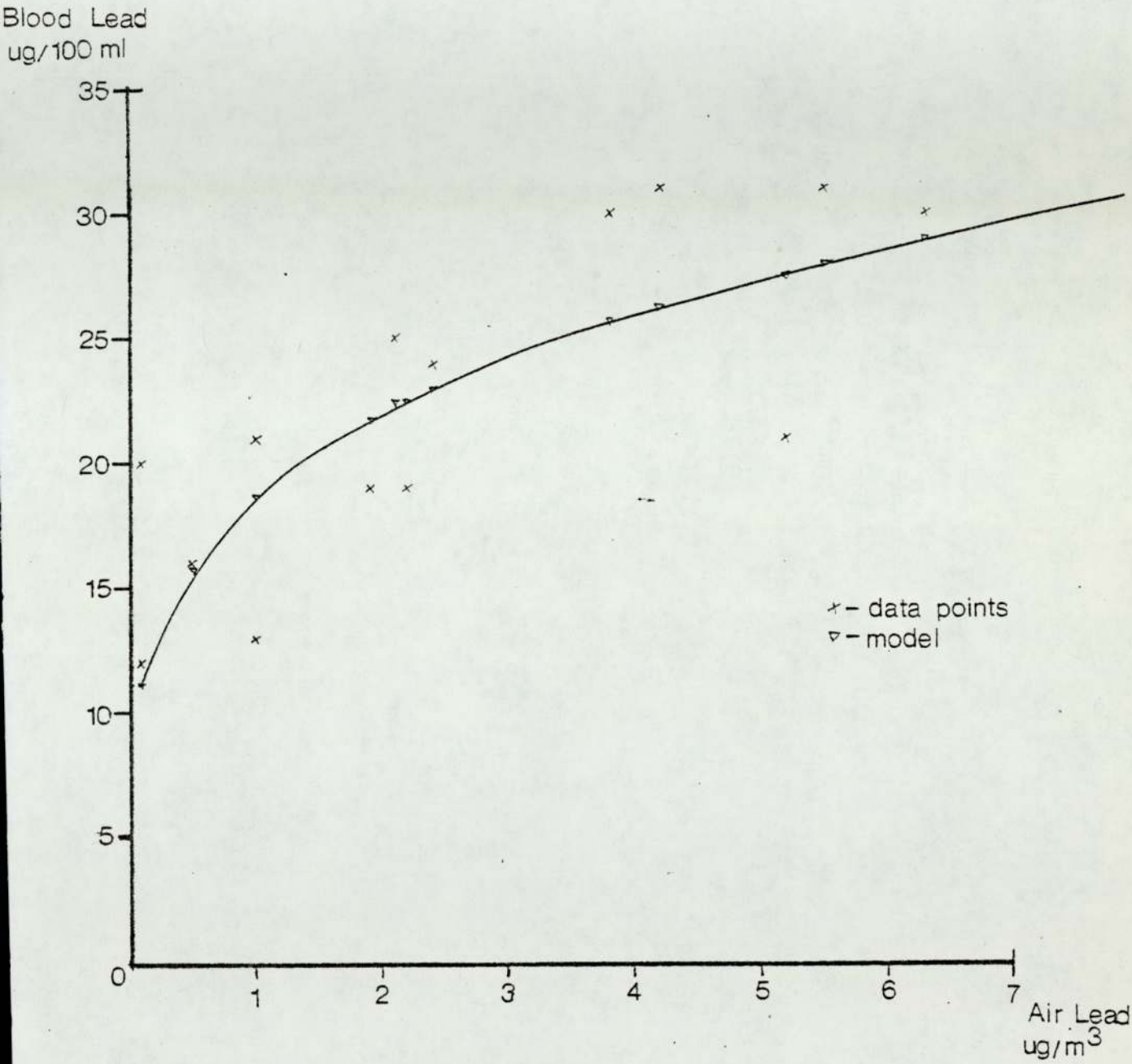
Table II-1

Goldsmith-Hexter Data

Type of Population	Estimated Exposure ug/m^3			Mean blood lead ug/100 ml	
	Occupational	Ambient	Ave.	Male	Female
Populations without known occupational exposure					
Remote California		*0.12		20	-
Mountain Residents		0.12		12	9
Composite rural U.S.		0.5		16	10
Suburban Philadelphia		1.0		13	13
Composite urban U.S.		1.0		21	16
L.A. aircraft workers		1.9		19	17
Pasadena City employers		2.2		19	12
Downtown Philadelphia		2.4		24	18
Populations with known occupational exposures					
Cincinnati policemen (all)	4.7	1.4	2.1	25	
Cincinnati traffic policemen	12.8	1.4	3.8	30	
Cincinnati auto test lane inspectors	14.8	1.4	4.2	31	
L.A. traffic policemen	16.5	2.2	5.2	21	
Cincinnati garage workers	21.1	1.4	5.5	31	
Boston Summer Tunnel workers	44.5	1.1	6.3	30	

* Alternative result suggested by the Ethyl Corporation

Figure II-1



Goldsmith – Hexter Model and Data

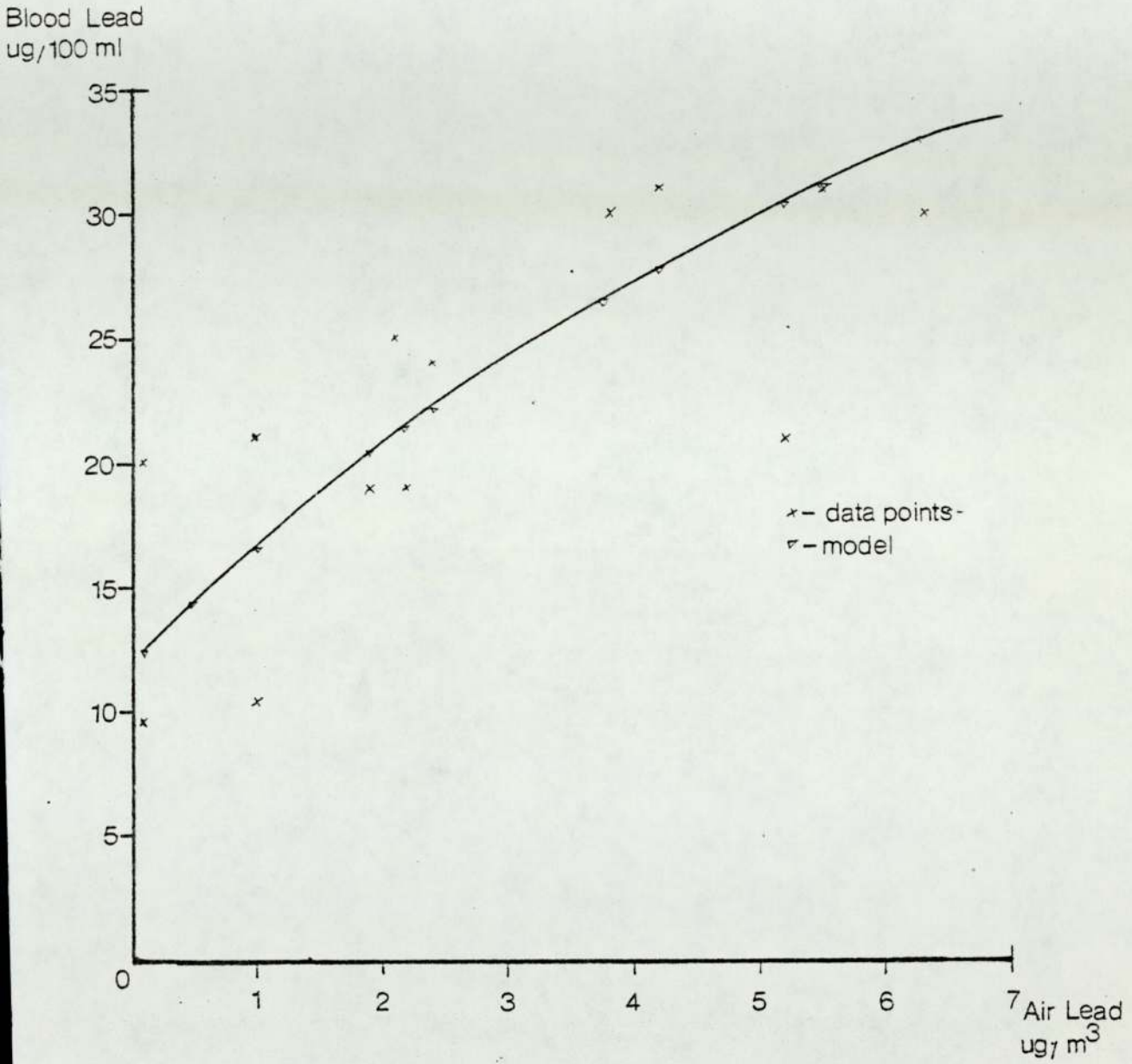
Table II-2

Supplementary points added to
Goldsmith-Hexter data by

National Academy of Sciences

Airborne lead ug/m^3	Dietary lead ug/day	Blood lead ug/100 ml
2.0	1350	48
2.0	2075	63

Figure II-2



NAS Model and Data

Table II-3

Seven-Cities Data

Area	Airborne lead ug/m^3	Blood lead ug/100 ml
Los Alamos	0.17	14.9
Okeana	0.32	15.7
Houston	0.85	12.5
Port Washington	1.13	15.3
Ardmore	1.15	18.0
Lombard	1.18	13.9
Washington D. C.	1.19	19.2
Rittenhouse	1.67	20.5
Bridgeport	1.76	17.6
Greenwich Village	2.08	16.6
Pasadena	3.39	17.5

It should be noted that all the blood lead values were obtained from female subjects.

Table II-4

Data from Tsuchiya et al.

Airborne lead ug/m^3	Blood lead ug/100 ml
0.024	17.0
0.198	16.8
0.413	16.7
0.507	15.9
0.746	18.8
1.302	20.6
1.011	20.3
0.998	17.5
0.791	18.3

This data was derived from a study on approximately 3000 Japanese policemen.

Table II-5

Data from Azar et al.

Area	Air lead ug/m^3	Blood lead ug/100 ml
Philadelphia Cab Drivers	2.62	22.4
Starke	0.81	16.4
Barksdale	1.01	13.8
Los Angeles Cab Drivers	6.10	24.6
Los Angeles Office Workers	3.06	19.9

This data was derived from a study on 150 male subjects.

Table II-6

Output from Goldsmith-Hexter model

Values from Three City Study		Model Output
Air lead ug/m^3	Blood lead ug/100 ml	Blood lead ug/100 ml
0.12	20*	11.19
0.12	12	11.19
0.50	16	15.77
1.00	13	18.62
1.00	21	18.62
1.90	19	21.72
2.20	19	22.50
2.40	24	22.97
2.10	25	22.25
3.80	30	25.65
4.20	31	26.28
5.20	21	27.66
5.50	31	28.03
6.30	30	28.96

* Alternative value suggested by Ethyl Corporation

Table II-7

Output from NAS Model

Values from NAS Report		Model Output
Air lead ug/m^3	Blood lead ug/100 ml	Blood lead ug/100 ml
0.12	12	12.32
0.50	16	14.27
1.00	13	16.60
1.00	21	16.60
1.90	19	20.30
2.20	19	21.41
2.40	24	22.13
2.10	25	21.05
3.80	30	26.61
4.20	31	27.75
5.20	21	30.38
5.50	31	31.12
6.30	30	32.98
*2.00	48	49.76
*2.00	63	59.20

* These are results from subjects with a very high dietary lead input, shown in Table II-2. This is taken into account when calculating the models' output, although they are not included in Figure II-2.

Table II-8

Data	Linear	Goldsmith-Hexter	NAS
Three Cities Study	0.806 0.755*	0.847 0.617*	0.832 0.761*
NAS Report	0.240	0.578	0.960
Seven Cities Study	0.411	0.419	0.432
Tsuchiya et al.	0.780	0.523	0.767
Azar et al.	0.879	0.901	0.904

This table shows how well the statistical models reviewed correlate (r) against the various epidemiological studies cited.

(* Modified point supplied by Ethyl Corporation inserted)

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