Cost Benefit Analysis of Reducing Lead in Drinking Water

Final Report to the Department of the Environment, Transport and the Regions
COST BENEFIT ANALYSIS OF REDUCING LEAD IN DRINKING WATER

Final Report to the Department of the Environment, Transport and the Regions

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COST BENEFIT ANALYSIS OF REDUCING LEAD IN DRINKING WATER

EXECUTIVE SUMMARY

BACKGROUND

The European Commission has proposed a revision of the Drinking Water Directive; for lead, it is proposed to reduce the limit from 50 to 25 µg/l for an interim period, with an ultimate limit of 10 µg/l to be achieved within 15 years of the implementation of the revised Directive. These limits are to apply to “representative” water samples although the precise form of the standard (e.g. mean, maximum) has not yet been determined. Even with the phasing-in of the new limit over a period of several years, this will incur considerable costs for water undertakers (for water treatment and communication pipe replacement) and property owners (supply pipe and plumbing replacement).

Lead in drinking water has long been associated with detrimental impacts on consumers’ health status. Changes in health status which have been examined have included the impact on IQ and lead-induced hypertension effects. It is possible to attach monetary values to the improvements in health status which would result from reducing exposure to lead from drinking water. A programme to reduce lead exposure would also generate other benefits and costs such as reduced leakage from a modernised distribution system and the disruption this modernisation would cause.

OBJECTIVES

A previous project ("the Cost Study") determined the costs to water suppliers (water treatment and pipe replacement) and householders (pipe replacement) of compliance with the various possible interpretations of the interim and final standards and monitoring requirements and compared these qualitatively.

The objective of this project is to carry out a cost benefit analysis utilising the cost information provided by the Cost Study in order to assess the economic benefits likely to arise from meeting various possible interpretations of the interim and final standards.

APPROACH AND METHODOLOGY

Two policy options were considered: compliance with the revised Directive and the “do nothing scenario”. In addition the study assessed the benefits arising from meeting the proposed interim standard of 25 µg/l as a maximum and as an average and the proposed final standard of 10 µg/l as a maximum and as an average. The cost information was taken directly from the Cost Study.

Relationships were established between lead in water and associated health effects such as the effect on childhood IQ. Dose response relationships were examined in terms of the exposure-damage relationships between lead levels, the numbers of people affected and the ways in which their health and development is impacted. Finally the benefits were estimated, where possible, in quantitative terms.
RESULTS

From the scientific literature, it is possible to derive models relating relevant low levels of blood lead to IQ and blood pressure. These models were used in the cost benefit analyses. Other endpoints which have been associated with low blood lead levels, such as effects on gestational age and biochemical indices, were not taken into account. Appropriate quantitative models for these endpoints are less well developed, and the clinical significance of, for example, minor changes in biochemical indices, is unclear.

Relationships were developed between waterborne lead and blood lead levels, with adjustments to account for the fall in blood lead levels since the original models were developed. However, it is likely that orthophosphate treatment of drinking water to reduce lead solubility will complex dissolved lead ions and reduce uptake; this could have a significant impact on the relationship between water lead and blood lead concentrations.

Current exposure to water lead was derived from the results of water company compliance sampling; future exposure was estimated for the “do nothing” and interim and final standard cases interpreted as an average and a maximum. Populations affected were derived from official actuarial figures.

The benefits that would accrue through increased IQ on future earnings were monetarised, taking into account the several linkages between lead, IQ, school performance, work-force participation and wages. The economic benefits associated with a reduction in blood pressure were estimated based on the linkages between lead, hypertension and morbidity and mortality, using a quality adjusted life years (QALYs) approach and valuation of quality and length of life changes. Non-health benefits and dis-benefits were assessed: it was possible to value some of these effects; however, their monetary values were small compared to the value of the health related benefits.

Given the uncertainty associated with the estimates, the overall appraisal considers ‘best case’ and ‘worst case’ scenarios. Best case results are estimated on the basis of the highest benefits and the lowest costs derived from the separate evaluations of impact, whilst worst case results are estimated on the basis of the lowest benefits and the highest costs.

All costs and benefits are discounted at a 6% real interest rate over the period 1998 to 2034 and are presented below in 1995 prices. In addition sensitivity tests were conducted on 4% and 2% discount rates. Costs are shown as negative, and benefits as positive, values; the net benefits are calculated as the sum of the (negative) costs and benefits. These figures are for England and Wales.

1 A terminal year of 2034 is used as it is the last year for the population forecasts used in the study and sufficiently far in the future to capture all relevant costs and benefits. Sensitivity analyses have confirmed that going further than 2034 does not alter the outcome.
## Costs and benefits of proposed lead standards (£million in 1995 prices)

<table>
<thead>
<tr>
<th>Cost benefit element</th>
<th>Interim 25 µg/l</th>
<th>Final 10 µg/l total</th>
</tr>
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<tr>
<td></td>
<td>Worst case</td>
<td>Best case</td>
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<tr>
<td>(a) standard interpreted as a mean</td>
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<tr>
<td>Total costs</td>
<td>-23</td>
<td>-23</td>
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<tr>
<td>Total health benefits</td>
<td>447</td>
<td>986</td>
</tr>
<tr>
<td>Total non health effects</td>
<td>33</td>
<td>76</td>
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<tr>
<td>Net benefit</td>
<td>457</td>
<td>1039</td>
</tr>
<tr>
<td>(b) standard interpreted as a maximum</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total costs</td>
<td>-5897</td>
<td>-177</td>
</tr>
<tr>
<td>Total health benefits</td>
<td>981</td>
<td>2191</td>
</tr>
<tr>
<td>Total non health effects</td>
<td>214</td>
<td>245</td>
</tr>
<tr>
<td>Net benefit</td>
<td>-4702</td>
<td>2258</td>
</tr>
</tbody>
</table>

## CONCLUSIONS

Interpreted as a mean, the proposed interim standard of 25 µg/l is associated with net benefits regardless of the success or otherwise of water treatment or other factors contributing to the range of costs and benefits between best case and worst case scenarios.

However, the proposed final standard of 10 µg/l would only produce additional net benefits if water treatment was successful. If treatment was unsuccessful in achieving compliance then, even if benefits were at the high end of their estimated range, there would be a net overall loss.

Interpreted as a maximum the interim standard is associated with positive benefits only under best case assumptions. The final standard interpreted as a maximum is associated with net costs under both best case and worst case scenarios. If the proposed standard is interpreted as a maximum, the success or otherwise of water treatment would have a significant impact on the costs.
The following table summarises the result of a best case - worst case assessment of the likely costs and benefits for the interim and final standards, interpreted as a mean and as a maximum. Only the interim standard produces a net benefit in the best and worst case scenarios. The final standard interpreted as a maximum produces a net loss in both cases. Meeting the interim as a maximum or the final standard as a mean produces a net benefit only under best case assumptions.

<table>
<thead>
<tr>
<th>Standard</th>
<th>Mean</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>25 μg/l Interim</td>
<td>Net benefit in best and worst case scenarios.</td>
<td>Net benefit in best case scenario only.</td>
</tr>
<tr>
<td>10 μg/l Final</td>
<td>Net benefit in best case scenario only.</td>
<td>Net loss in best and worst case scenarios.</td>
</tr>
</tbody>
</table>

The balance between costs and benefits and the extent of any net benefit depend critically on the degree of success of water treatment.

The study has revealed gaps in the data in several areas; a better understanding of a number of topics would help to improve the estimates made in this report and would be of value in considering other aspects of lead in drinking water.
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1. INTRODUCTION

1.1 Background

The current Drinking Water Directive 80/778 defines the requirements for monitoring drinking water in terms of the parameters and their Maximum Admissible Concentrations (MAC) and Guide Levels (GL); groups of parameters and the frequency at which each group is to be monitored; and reference methods of analysis. The Maximum Admissible Concentration (MAC) for lead is 50 µg/l in running water.

In 1993, the World Health Organization (WHO) published a revision of its guidelines on drinking water quality. Based on an assessment of the available data on the effects of lead on human health, the guideline value for lead was reduced from 50 µg/l to 10 µg/l. This was based on the need to avoid accumulation of lead in infants but is adequate to protect all age groups. It was derived using the 1986 Joint Expert Committee on Food Additives (JECFA) provisional tolerable weekly intake (PTWI) of 25 µg/kg body weight for infants and children. As infants were considered to be the most sensitive subgroup of the population, the guideline was based on a 5 kg bottle-fed infant consuming 0.75 litres of drinking water per day.

The significantly more stringent guideline value raised the issue of a need for a revision of the MAC for lead. The European Commission has undertaken a fundamental review of the Drinking Water Directive. A proposed revision has been published which takes into account the new WHO Guidelines and incorporates several other changes. For lead, it is proposed to reduce the MAC from 50 to 25 µg/l for an interim period, with an ultimate MAC (or “Parametric Value”, PV) of 10 µg/l to be achieved within 15 years of the implementation of the revised Directive. These limits are to apply to “representative” water samples although the precise form of the standard (e.g. mean, MAC) and monitoring method have not yet been determined. Even with the phasing-in of the new standard over a period of several years, this proposal clearly has substantial consequences for water undertakers and their customers.

Whilst early agreement to a revised Directive is unlikely, it is important for the UK’s negotiating strategy to understand the extent of non-compliance and how much further water treatment and pipe replacement might be needed to achieve compliance with various possible interpretations of the standards and monitoring requirements; and the effectiveness and costs of various sampling regimes. WRc recently reported on a study of these aspects of lead in drinking water - the Cost Study (Miller et al. 1997).

The position taken by the UK on the Directive will depend on confronting the costs of meeting the Directive with the likely benefits. Lead in drinking water has long been associated with detrimental impacts on consumers’ health status. Changes in health status which have been examined have included neurotoxic effects such as the impact on IQ and
the cognitive development of children; effects on foetal development and reproduction; and lead-induced hypertension effects in adults and the impact on cardiovascular disease, strokes and heart attacks.

The benefits arising from the reduction of lead contamination of drinking water supplies will be driven by two factors: the number of individuals experiencing an improvement in health status and the value attached to that improvement.

The true benefit of reductions in lead in drinking water will be a function of the willingness to pay for health and other benefits by the affected parties. Several techniques exist for valuing changes in health status which arise from changes in exposure.

A programme to reduce lead exposure is also likely to generate other costs and benefits such as reduced materials damage through reductions in the corrosivity of water, reduced leakage from a modernised distribution system and the dis-utility from the disruption this modernisation would cause.

The US EPA examined the costs and benefits of a similar reduction in lead concentrations (from 50 µg/l to 20 µg/l) and estimated costs of US$ 230M and benefits of US$ 1113M.

1.2 Objectives

The Cost Study determined the costs to water suppliers (treatment and pipe replacement) and householders (pipe replacement) of compliance with the various possible interpretations of the interim and final standards and monitoring requirements and compared these qualitatively. The objective of this project is to carry out a cost benefit analysis utilising the cost information provided by the Cost Study to assess the benefits likely to arise from meeting the various possible interpretations of the standards.

1.3 Guide to the report

Section 2 provides a brief description of the overall approach to the study.

Section 3 is a brief introduction to lead in drinking water.

Section 4 gives a summary of the methodology and outcome of the Cost Study.

Section 5 is a review of the health effects of lead and the relationship between water lead and blood lead concentrations.

Section 6 presents the approach, methodology and results of the estimation and valuation of health effects.

Section 7 describes the non-health effects that would arise from action to reduce lead concentrations, and derives monetary values where possible.
Section 8 brings together the costs and benefits to provide an overall cost benefit appraisal.

The results are discussed in Section 9, whilst the conclusions and recommendations are given in Section 10.

This report necessarily includes several technical terms - these are explained in the Glossary (Appendix A).
2. APPROACH

Four main bodies of work were undertaken (1) literature review, (2) the identification of the exposure-damage relationships and quantification of the effects of different interpretations of the proposed Directive, (3) the identification and application of benefit measures and techniques to the resulting impacts and (4) the overall appraisal.

The cost information was taken directly from the Cost Study (Miller et al. 1997); this is summarised in Section 4.

Two policy options were considered: compliance with the revised Directive and the "do nothing scenario". In addition the study assessed the benefits arising from meeting the proposed interim standard of 25 μg/l as a maximum and as an average and the proposed final standard of 10 μg/l as a maximum and as an average.

Relationships were established between lead in water and associated health effects such as the effect on childhood IQ. Dose response relationships were examined in terms of the exposure-damage relationships between lead levels, the numbers of people affected and the ways in which their health and development could be impacted.

Next the benefits were estimated in qualitative and where possible quantitative terms.

Inevitably some effects were not amenable to monetarisation either because no monetary values exist or the estimates that do exist do not conform to the benefit transfer criteria (e.g. where the estimates are derived under very different circumstances in comparison to the present context or where low statistical confidence is attached to the estimates). Those effects which could not be monetarised were treated in a qualitative manner.
3. LEAD IN DRINKING WATER

Contamination of drinking water by lead occurs almost exclusively through contact with pipe material or fittings that contain lead. In the UK contamination of drinking water by lead through polluted raw waters is rare since lead concentrations in raw waters are generally low, and conventional water treatment will remove lead present in raw water down to trace quantities.

Although lead pipework is the main source of lead contamination there are other materials which contain lead; these include solders, brasses and galvanised pipes. Thus it is possible that even where there are no lead pipes lead concentrations may exceed 10 μg/l due to the lead in the solders and fittings. There are laboratory data to show these can leach lead but the true extent to which this is a problem in distribution has not been measured. Lead free solders and brasses are now available for drinking water systems. The lead salts used as stabilisers in some uPVCs can also leach lead, but this is only significant for a short period from newly installed pipes.

Lead is largely present in drinking water in the dissolved form. Particulate lead, where small flakes of lead become detached from lead pipes, or adsorbed onto other particulate matter, is also found in a small minority of areas of the UK.

The only method which will eliminate lead from drinking water altogether is replacement of all lead containing materials in contact with the water. Water treatment by pH adjustment and orthophosphate dosing to reduce lead solubility can reduce dissolved lead concentrations but will not eliminate the problem completely.

Since lead enters drinking water by leaching from lead pipe work which is in the near vicinity of the sampling point, i.e. the consumer’s tap, the concentration of lead in a sample is markedly affected by the way in which the sample is taken. Essentially there are five types of sample that are used to measure lead - fully flushed, composite proportional, fixed stagnation time, random daytime and overnight stagnation - these are described in Appendix A.

In the UK random daytime samples are used for compliance monitoring. A 1 litre sample is taken at a random time during the working day from a pre-selected random address.

The proposed Directive requires the sampling method for lead to be “representative of the water drawn by the consumer”. The precise form of sampling is the subject of an ongoing EU study. Nonetheless it is possible to consider and evaluate the possible sampling methods. The best representation of water drawn by a consumer is provided by the composite proportional sample. Thirty minute stagnation samples could also be a possibility since studies have shown that the average daytime stagnation period for a typical household is thirty minutes. Lead concentrations in random daytime samples are considered representative of water drawn at the time of sampling but they may not be representative of the water drawn at other times because they exhibit a wide variability.
Flushed samples are not representative of water consumed since in general householders do not run the tap for any length of time before filling the kettle, for example. Fully flushed samples would represent the lowest lead concentration likely at a property. Conversely, first draw samples would give a worst case lead concentration.
4. SUMMARY OF COST STUDY

4.1 Approach

Even though the new Directive has yet to be finalised, it is possible to make assumptions in order to produce estimates of the likely degree of compliance based on the various possible interpretations of the standards and monitoring requirements. The Cost Study estimated the degree of pipe replacement or additional water treatment that would be required in England and Wales to achieve compliance with the possible scenarios, and estimated the resultant costs.

The aim of this study was to estimate, for a number of compliance scenarios, where further water treatment would be effective and where the only method to achieve compliance would be pipe replacement. The study made estimates for three possible sample types: random daytime (RDT), 30 minute stagnation (30MS) and composite proportional (COMP).

4.1.1 Statistical scenarios

Three different numerical concentrations are or will be used as the standard for lead:

- 50 µg/l, the current numerical EC and UK standard
- 25 µg/l, the proposed interim EC standard
- 10 µg/l, the WHO guideline value and the proposed final EC standard.

The sampling procedure, the concentration limit and the interpretation of the limit determine whether a particular sample passes or fails the standard.

Various rules may be applied to determine whether there is a risk that a zone fails the current UK standard, whether the risk applies to an insignificant part of a zone and therefore whether action is needed to reduce lead concentrations. Zones classified by water undertakers as 1 and 2a according to Schedule 3 from the Water Undertakers (Information) Direction 1992 were assumed to pass the no change scenario, i.e. 50 µg/l regarded as a maximum. There is assumed to be no risk of failing the current UK standard in a zone if all survey samples contain less than 50 µg/l (Class 1, zone passes). There is a risk of failing the current standard in a zone if any of the survey samples exceed 50 µg/l (Class 2, zone fails). But if the proportion of properties failing is less than 2% of all properties on the basis of a sample survey or 5% of the population (not more than 1000 people or 400 properties), the risk may be regarded as relating to an insignificant part of the zone and treatment need not be considered (Class 2a, zone passes). The full range of zone lead risk classifications are presented in Appendix A.
The current UK standard concentration of 50 µg/l is a maximum. The proposed EC Directive would require a tightening of the standard. At 25 or 10 µg/l, any of three rules of interpretation might apply, and are considered nominally maximum, mean and percentile. (For the purposes of this study an 80 percentile is used.)

Several different scenarios were identified:

- Concentration 25 or 10 µg/l
- Sampling procedure 30MS/COMP or RDT
- Rule for zone pass/failure maximum or 80 percentile or mean

The data used to estimate the numbers of zones that would pass or fail, given each of these scenarios, were:

- Summarised results of the 1995 statutory sampling, that included the numbers of measured concentrations by compliance zone in concentration bands, and the water company classification of lead risk by zone.
- Summarised results for 1994 and 1995, that included the maximum measured lead concentrations by zone for each year.
- The proportion of properties with lead pipes, by zone or in some cases by company area or division.

The following definitions of pass and failure that were used:

- **Pass**
  Either the zone contains less than both 5% of its population or less than 1000 population (400 properties) supplied through lead pipes, or if it contains more, the results of applying the scenario to a well designed survey would be a pass.

- **Failure**
  The zone contains at least 5% of its population or more than 1000 population (400 properties) supplied through lead pipes, and the results of applying the scenario to a well designed survey would be a failure.

Application of these definitions was limited by the nature of the data, and it was necessary to make some assumptions. Details of the method used to estimate the numbers of zones that would pass or fail under each scenario are given below.

### 4.1.2 Pipe replacement assumptions

For the purposes of cost estimation it is assumed that all lead pipes would be replaced in a zone where water treatment alone would not achieve compliance. For zones passing a lead limit of 10 µg/l, no allowance is made for pipe replacement even where these zones contain lead pipes.
4.1.3 Water treatment assumptions

The following rules were derived to determine which zones would comply if further water treatment were installed. It was assumed that orthophosphate treatment would be necessary to achieve a sufficiently low solubility to meet a 25 µg/l standard. A 10 µg/l limit (as a mean) might be also achievable in some zones, similarly some zones might fail a 25 µg/l limit (as a maximum) after treatment was installed.

The equilibrium lead solubility ($Pb_E$) can be calculated from water quality data (including pH, alkalinity, orthophosphate concentration and calcium concentration); the solubility is approximated by the linear function

$$Pb_E = 0.22 \times Alk$$

where $Pb_E$ is the equilibrium solubility (µg/l Pb)
$Alk$ is the alkalinity (mg/l HCO$_3$)

This equation underestimates the solubility of lead in low alkalinity waters; to compensate for this it was assumed that the minimum equilibrium solubility achievable is 20 µg/l.

Theoretical and empirical models exist to predict the mass transfer behaviour of lead to produce theoretical stagnation curves and to calculate lead concentrations that would be found in different types of sample. These models and practical measurements together with the comparative study of current data resulted in the derivation of solubility values applicable to each scenario. These values were used to predict where water treatment could achieve a 25 µg/l limit for different samples types and compliance criteria.

Data were studied from three companies, eight zones in total, where orthophosphate dosing to control plumbosolvency has been installed for a number of years. The data show that the numbers of zones passing or failing standards of 10 µg/l as a mean and 25 µg/l as a 95 percentile (near maximum) are similar. In addition to assisting in the derivation of the solubility predictions for the 25 µg/l lead standard as described above, these data indicated that orthophosphate dosing can also achieve compliance with the 10 µg/l lead standard in some zones.

For each zone, an assessment was made of whether treatment would be a feasible (additional) option to meet each scenario on the following basis:

- Significant lead pipe population: at least 5% of population or at least 1000 population (400 properties), whichever is the smaller, supplied through lead pipe have to be present for treatment to be practicable (compared to pipe replacement).
- Treatment is not already installed: it is assumed that any optimisation of existing treatment would incur negligible cost.
- Calculated equilibrium solubility satisfies the criteria discussed above and as summarised in Table 4.1.
Table 4.1  Assessment criteria for water treatment

<table>
<thead>
<tr>
<th>Sample type</th>
<th>Compliance criterion- solubility values (PbE) μg/l</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Maximum</td>
</tr>
<tr>
<td>25 μg/l, random daytime</td>
<td>&lt;20 - 25</td>
</tr>
<tr>
<td>25 μg/l, 30 minute stagnation or composite proportional</td>
<td>&lt;20 - 25</td>
</tr>
<tr>
<td>10 μg/l, random daytime or 30 minute stagnation or composite proportional</td>
<td>&lt; 10 (1)</td>
</tr>
</tbody>
</table>

(1) i.e. treatment will not achieve compliance with 10 μg/l as a maximum
(2) i.e. similar to 25 μg/l maximum

4.2 Methodology

4.2.1 Data collection

Data were collected from four sources:

- Information Direction returns on lead for 1995, and 1994 and 1995 water quality summary compliance data were provided by DWI.

- Data from the Water Companies in England and Wales on lead pipe occurrence, special lead sampling surveys and lead rehabilitation costs.

- Data held by WRc from previous lead studies was used where appropriate and with the originators’ permission.

- Data from lead surveys undertaken in other parts of the UK and Europe were also gathered.

4.2.2 Comparing different sampling methods

One objective of the project was to compare the results obtained using different sampling methods, to see if there was a direct relationship between them. Several water companies provided data which allowed comparisons to be made. These were data from special surveys, where properties had been sampled using more than one method.
None of the companies was able to provide data on composite proportional sampling. According to Randon (1996), the ratio of 30 minute stagnation to composite proportional sampling is 0.93:1. Here it was assumed that 30 minute stagnation samples would give equivalent lead concentrations to composite proportional sampling.

Data were obtained for three zones on random daytime and 30 minute samples, which were reasonably consistent. Median ratios between the 30 minute stagnation concentration and the corresponding random daytime concentration were 0.89:1, 0.79:1 and 0.85:1.

4.2.3 Compliance with the lead standard in 1995

The DWI lead returns were analysed as follows. A matrix was set up with lead concentration horizontally and lead risk vertically. The returns were examined, and each zone was categorised according to the maximum concentration measured for that zone, and its lead risk. This built up a matrix which showed, for each concentration band and lead risk, how many zones fell into that category. The matrixes for all the different companies were aggregated into one, which is shown below as Table 4.2. The definitions of zone lead risk classes are given in Appendix A.

Table 4.2 Summary of 1995 lead returns for England and Wales

<table>
<thead>
<tr>
<th>Lead risk</th>
<th>0-10</th>
<th>&gt;10-20</th>
<th>&gt;20-30</th>
<th>&gt;30-40</th>
<th>&gt;40-50</th>
<th>&gt;50-100</th>
<th>&gt;100</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>881</td>
<td>137</td>
<td>57</td>
<td>32</td>
<td>23</td>
<td>16</td>
<td>8</td>
<td>1154</td>
</tr>
<tr>
<td>2a</td>
<td>77</td>
<td>29</td>
<td>14</td>
<td>11</td>
<td>11</td>
<td>50</td>
<td>29</td>
<td>221</td>
</tr>
<tr>
<td>2b</td>
<td>8</td>
<td>5</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>10</td>
<td>3</td>
<td>30</td>
</tr>
<tr>
<td>2c</td>
<td>18</td>
<td>9</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>4</td>
<td>2</td>
<td>40</td>
</tr>
<tr>
<td>2d</td>
<td>64</td>
<td>51</td>
<td>47</td>
<td>43</td>
<td>33</td>
<td>116</td>
<td>109</td>
<td>463</td>
</tr>
<tr>
<td>2e</td>
<td>27</td>
<td>14</td>
<td>10</td>
<td>6</td>
<td>5</td>
<td>17</td>
<td>11</td>
<td>90</td>
</tr>
<tr>
<td>2f</td>
<td>15</td>
<td>16</td>
<td>22</td>
<td>15</td>
<td>14</td>
<td>55</td>
<td>21</td>
<td>158</td>
</tr>
<tr>
<td>2g</td>
<td>56</td>
<td>20</td>
<td>7</td>
<td>8</td>
<td>5</td>
<td>15</td>
<td>15</td>
<td>126</td>
</tr>
<tr>
<td>2h</td>
<td>13</td>
<td>2</td>
<td>2</td>
<td>3</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>23</td>
</tr>
<tr>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>1159</td>
<td>283</td>
<td>164</td>
<td>122</td>
<td>95</td>
<td>283</td>
<td>199</td>
<td>2305</td>
</tr>
</tbody>
</table>

The procedure used to estimate the likely extent of non-compliance with the various scenarios is summarised in Table 4.3.
Table 4.3  Estimating likely non-compliance by zone for future standards

<table>
<thead>
<tr>
<th>Step</th>
<th>Procedure</th>
</tr>
</thead>
</table>
| 1    | **Initial grouping of zones**  
The 1995 results and the company lead risk classification were used to divide the zones into three groups:  
*Group I*: zones in class 1 with the maximum measured lead concentration ≤10 μg/l.  
For these zones there is no evidence of any problem. However, data from a specially designed survey or from other years might contain measured concentrations over 10 μg/l, and a proportion of the zones would therefore be expected to fail under some scenarios. This was allowed for as described in step 7.  
*Group II*: zones in classes 2b to 2h.  
Surveys in or around 1989 indicated that action was judged to be required in these zones on the basis of the 50 μg/l standard. Since the new lead standard is intended to be tighter, it is almost certain that action would have been required under any scenario. Treatment to reduce lead concentrations has now been installed in many zones, and hence this has not been included in the cost calculations.  
*Group III*: zones in class 1 with a maximum concentration over 10 μg/l, and zones in class 2a (problem restricted to an insignificant part of the zone).  
Zones in this group might pass or fail, depending on the scenario, and steps 2 to 6 were applied to this group only. |
| 2    | **Allowing for samples from properties without lead pipes**  
For each zone, a proportion of the measured lead concentrations were removed from the 1995 results, corresponding to the proportion of properties stated by the water company not to have lead pipes. In all zones the lowest measured concentrations were removed first. In most but not all cases this meant removing measured concentrations ≤10 μg/l. This does not influence the maximum concentration, but has the effect of raising the percentile and mean values. |
| 3    | **Adjusting the concentrations for different sampling procedures**  
Relationships between the concentrations measured using different sampling procedures were derived:  
| Mean | 30MS and COMP = 0.89 × RDT  
| Maximum | 30MS and COMP = 0.74 × RDT.  
A ratio was not derived for the 80 %ile values. It would lie between the values for the mean and maximum. |
| 4    | **Applying the pass/fail rules**  
For each zone, the maximum, mean and 80 percentile values were estimated. Because the data were provided in ranges, actual measured concentrations were estimated as the mid point of the range. These values were then compared to the appropriate limit - 10 or 25 μg/l. |
| 5    | **No change scenario**  
Under this scenario, 50 μg/l and a maximum rule, the original water company classifications were applied and all zones in classes 1 and 2a were assumed to pass. |
| 6    | **10 μg/l maximum scenario**  
All zones were regarded as at risk of failure. However, the data are not sufficient to indicate the proportion of zones that would fail. |
| 7    | **Zones in class 1 with the maximum measured lead concentration ≤10 μg/l**  
The maximum concentrations by zone in 1994 and 1995 were compared, in order to gain some understanding of the proportion of group I zones that might have been found to fail by a comprehensive survey. |
The questionnaire which was used to gather data asked the utilities to assess whether or not each zone would comply with several possible new scenarios for lead concentration. An audit was carried out to find out how the project estimates compared with the utilities’ estimates. For all but the 10 µg/l limit on the maximum, random daytime sampling scenario, the numbers of zones estimated to pass or fail using the project “rules” and utilities’ estimates were similar. However, a large proportion of the zones that pass the rules, i.e. in risk class 1 and for which the maximum concentration measured in the 1995 data is less than 10 µg/l, are probably at risk of failing under this scenario.

Between 2 and 10% of zones would fail a lead standard of 25 µg/l depending on the sampling scenario adopted. Of these zones between 39 and 58% would require pipe replacement to achieve compliance and in 42 to 61% compliance could be achieved with additional water treatment. At a standard of 10 µg/l, 20 to 63% of zones (including those failing at 25 µg/l) would fail depending on the sampling scenario adopted.

Thus it is concluded that the water treatment measures presently installed in England and Wales to comply with the current lead standard (50 µg/l) are adequate in all but the minority of zones to achieve compliance with a proposed interim standard (25 µg/l). But to achieve compliance with the proposed final standard (10 µg/l) as a maximum would require some considerable effort. Pipe replacement would be necessary at between 5.5 to 9.8 million properties. It should also be noted that this would not remove all the lead pipework in England and Wales since the available data suggest that a further 39% of zones that are believed by water companies to contain lead pipework would achieve compliance at 10 µg/l. If the proposed final standard (10 µg/l) is expressed as a mean the cost estimates are more uncertain. Some zones would achieve compliance from water treatment alone but others would require pipe replacement.

4.3 Cost estimation

A range of cost estimations was made. The most demanding assumes that water treatment will reduce solubility sufficiently to comply with a lead standard of 25 µg/l as a mean or 80 percentile but not with a standard of 25 µg/l as a maximum or 10 µg/l as a maximum, mean or 80 percentile. The least stringent assumes that some zones will pass a lead standard of 25 µg/l as a maximum and 10 µg/l expressed as a mean or 80 percentile solely by orthophosphate dosing for plumbosolvency control and all zones will pass 25 µg/l as a mean or 80 percentile. It was not possible to derive reliable predictions of occurrence, or cost estimates for dealing with, particulate lead.

The following assumptions were also made when estimating the cost of compliance with each scenario.
At a lead standard of 25 µg/l:

- For class 2d to 2f zones there would be no additional costs since treatment has already been installed in these zones and hence zones are assumed to pass.
- For class 2b and 2c zones treatment is not an option so there will be a pipe replacement cost where lead concentrations are above 25 µg/l.
- For class 1 and 2a zones (that exceed 25 µg/l) for some zones additional water treatment will achieve compliance for which there will be a cost. For the remaining class 1 and 2a zones (that exceed 25 µg/l) there will be pipe replacement costs.

At a lead standard of 10 µg/l:

- For class 1 zones where lead concentrations are less than 10 µg/l there will be no additional costs.
- For zones where pipe replacement was performed to meet 25 µg/l there will be no extra costs.
- For some zones water treatment will achieve compliance at 10 µg/l as a mean or 80 percentile. For class 1 and 2a zones an operational treatment cost has been included. For 2d to 2f treatment costs are not included.
- For all other zones there will be pipe replacement costs.

General:

- All estimates are made assuming zero discount rates.
- Treatment costs are estimated assuming the capital cost is incurred in year 1 plus operational costs for year 2 onwards. Costs are estimated on a per capita basis.
- When calculating the cost of pipe replacement it is assumed that the current replacement rate continues regardless of other replacement initiatives. Therefore, for replacements to meet 25 µg/l, 5 years of current replacement rates are deducted and for replacements to meet 10 µg/l, 15 years of current replacement rates are deducted from current pipe occurrences.
- The number of properties with lead communication pipes, supply pipes and plumbing requiring replacement will be similar.
- Unit cost data for pipe replacement and water treatment were supplied by the water companies and supplemented by data previously collected by WRc. These data are summarised in Appendix B.

The costs were estimated at a zonal level, then aggregated progressively to produce costs to companies, Ofwat regions, and finally total costs for England and Wales. The costs of compliance estimated for each scenario for a limit of 25 µg/l are summarised in Table 4.4,
whilst Table 4.5 shows the total costs that would be incurred in achieving a 10 µg/l limit. It can be seen that approximately 75% of the costs are associated with the removal of lead supply pipe and plumbing which are the responsibility of the property owners. The range of costs for the maximum at 25 µg/l and all 10 µg/l scenarios reflect the possible range of effectiveness of water treatment under these scenarios.

<table>
<thead>
<tr>
<th>Table 4.4</th>
<th>Summary of costs - England and Wales total - 25 µg/l limit</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Costs £million</td>
</tr>
<tr>
<td></td>
<td>Random Daytime</td>
</tr>
<tr>
<td></td>
<td>30 minute stagnation or composite proportional</td>
</tr>
<tr>
<td>Compliance criterion</td>
<td>Suppliers</td>
</tr>
<tr>
<td>Maximum</td>
<td>160 - 1900</td>
</tr>
<tr>
<td>Mean</td>
<td>30</td>
</tr>
<tr>
<td>Percentile</td>
<td>50</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 4.5</th>
<th>Summary of total costs - England and Wales total - 10 µg/l limit</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Costs £million</td>
</tr>
<tr>
<td></td>
<td>Random Daytime</td>
</tr>
<tr>
<td></td>
<td>30 minute stagnation or composite proportional</td>
</tr>
<tr>
<td>Compliance criterion</td>
<td>Suppliers</td>
</tr>
<tr>
<td>Maximum</td>
<td>1480 - 1900</td>
</tr>
<tr>
<td>Mean</td>
<td>700 - 1610</td>
</tr>
<tr>
<td>Percentile</td>
<td>770 - 1610</td>
</tr>
</tbody>
</table>
4.4 Key assumptions

- Statistically determined relationships between lead concentrations in different types of sample, together with compliance sampling data, were used to identify those zones which would pass or fail various scenarios.

- The suitability of water treatment to achieve compliance with each scenario was assessed using a simplified model of the effects of treatment on lead solubility, and various assumptions concerning the ability of treatment to achieve a 10 μg/l limit were included in the cost calculations.

- It was assumed that all lead pipes would be replaced in a zone where water treatment alone would not achieve compliance.

- Pipe replacement was not considered for zones which would pass a lead limit of 10 μg/l, even if these zones contain lead pipes.
5. HEALTH EFFECTS AND EXPOSURE TO LEAD

5.1 Health effects

5.1.1 Introduction

The toxicity of lead has been recognised for a number of years and many countries, including the UK, have initiated programmes to reduce the general population's exposure to lead. Nonetheless, lead continues to be an important public health issue despite successful governmental efforts in recent years to reduce general population exposure to this toxicant. Part of the reason for the continuing concern is that lead has been repeatedly found to induce adverse health effects at increasingly lower levels of exposure.

A literature review was undertaken to establish the health effects associated with low-level lead exposure in the long-term and these are summarised in Table 5.1. Infants, children up to 6 years of age, the foetus and pregnant women are regarded as likely to be the most susceptible groups to adverse health effects of lead (WHO 1993). The lowest exposures at which these health effects are reliably detected are measured in terms of blood lead levels, PbB, (generally expressed in µg of lead per deci-litre of blood). The most sensitive endpoints appear to be IQ deficits in children, effects on gestational age and foetal weight, effects on blood pressure of adults and biochemical effects. There is also some evidence that low blood lead levels can cause hearing impairment in children and effects on growth and stature, although these have not been well studied and their significance is uncertain. The other health effects summarised in Table 5.1 occur at levels which are greater than the UK Department of Health advisory action limit of 25 µg/dl for lead in blood (the level at which the person's environment should be investigated for sources of lead and steps taken to reduce exposure).

Once the health effects associated with lead had been established, data were sought on the base-line blood lead levels which currently occur in the general population (see Section 5.1.2). Recent surveys in the UK demonstrate that blood lead levels have significantly fallen over the past decade for the general population. Indeed, many of the toxic effects associated with lead exposure have been found only at levels which are higher than current blood lead levels.

Reference was made to elevated lead levels in menopausal women in the report, on drinking water, of the House of Lords Select Committee on the European Communities in 1996. This is not associated with high current exposure or increased absorption but with the release of lead in bone as a consequence of earlier exposures, since bone mobilisation is a common feature in menopause. The significance of these elevated blood lead concentrations is uncertain and will vary according to the individual. It should be viewed from the perspective of the significant decrease in population blood lead over the past decade.
Table 5.1  Health effects associated with low level exposure to lead

<table>
<thead>
<tr>
<th>Effect</th>
<th>PbB µg/dl</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biological effects</td>
<td></td>
<td>Enzymes critical in haem synthesis. IPCS (1995) concludes that blood lead levels for parameters which may have clinical significance are above 20 µg/dl. Some effects on enzymes are demonstrable at lower lead levels but the clinical significance is uncertain.</td>
</tr>
<tr>
<td>Biochemical effects</td>
<td></td>
<td>Effects on circulating levels of 1,25-dihydroxyvitamin D</td>
</tr>
<tr>
<td>Inhibition of 5-aminolaevulinate dehydratase (ALA-D)</td>
<td>10</td>
<td>Used in the synthesis of catecholamines</td>
</tr>
<tr>
<td>ALA in urine</td>
<td>35</td>
<td>Leads to an accumulation of pyrimidine nucleotides, associated with basophilic stippling</td>
</tr>
<tr>
<td>Inhibition of coproporphyrinogen oxidase</td>
<td>*</td>
<td>Regulates erythrocyte production</td>
</tr>
<tr>
<td>Increased coproporphyrin in urine</td>
<td>40</td>
<td>Decreased haemoglobin levels</td>
</tr>
<tr>
<td>Inhibition of ferrochelatase</td>
<td>*</td>
<td></td>
</tr>
<tr>
<td>Increased protoporphyrin in urine</td>
<td>20-30</td>
<td></td>
</tr>
<tr>
<td>Inhibition of 25-hydroxy-D-1α-hydroxylase</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>Inhibition of dihydrobiopterin reductase</td>
<td>10</td>
<td>Nerve conduction velocity</td>
</tr>
<tr>
<td>Inhibition of NAD synthetase</td>
<td>35</td>
<td>Electrocardiographic R-R interval variability</td>
</tr>
<tr>
<td>Inhibition of pyrimidine-5'-nucleotidase activity</td>
<td>10</td>
<td>May be lower if sensitive indicators are used</td>
</tr>
<tr>
<td>Erythropoietin production</td>
<td>*</td>
<td>The effects of confounding variables and limits in the precision in analytical and psychometric measurements increase the uncertainty attached to any estimate of effect.</td>
</tr>
<tr>
<td>Anaemia</td>
<td>40 (child)</td>
<td>IPCS (1995) concluded that the relationship is not necessarily causal.</td>
</tr>
<tr>
<td>Sensory motor function, CNS</td>
<td>40</td>
<td>Less well studied</td>
</tr>
<tr>
<td>Peripheral nervous system</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>Autonomic nervous system</td>
<td>35</td>
<td></td>
</tr>
<tr>
<td>Nephropathy</td>
<td>60</td>
<td></td>
</tr>
<tr>
<td>IQ decrements in children</td>
<td>10-15</td>
<td></td>
</tr>
<tr>
<td>Hearing impairment in children</td>
<td>7-18 ?</td>
<td></td>
</tr>
<tr>
<td>Blood pressure</td>
<td>7-34 ?</td>
<td></td>
</tr>
<tr>
<td>Pre-term delivery, foetal growth</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>Effects on growth and stature</td>
<td>&lt;25 ?</td>
<td></td>
</tr>
<tr>
<td>Sperm count and quality</td>
<td>40</td>
<td></td>
</tr>
</tbody>
</table>

* Lowest blood lead levels at which reliably detected.  
* No dose response defined
Given that recent studies indicate blood lead levels in the UK have been generally well below 25 µg/dl, our study has focused on those health effects which are associated with blood lead levels below this figure. These included:

- IQ decrements
- Pre-term infant delivery and reduced foetal weight
- Increased blood pressure
- Biochemical effects

5.1.2 Current blood lead levels in the UK

The blood lead levels reported in recent surveys carried out in the UK indicate that these have reduced substantially since the 1980s (Delves et al. 1996; Watt et al. 1996). This will be related to a number of programmes of lead reduction in the environment, e.g. banning of lead solder in tins, controlled use of lead based paints and unleaded petrol, as well as improvements in water treatment using orthophosphate dosing.

The most recent survey of blood lead levels in the population in England was carried out in 1995 by using a sub-sample of the 1995 Health Survey for England. This survey is the first to have monitored blood lead levels in the English population in 8 years. Blood samples were taken from 6868 subjects from eight regions throughout England (Delves et al. 1996). Most of the subjects were adults (95%) of whom 3139 were men and 3389 were women, the remaining 340 being children aged 11 to 15. The median blood lead concentrations (µg/dl) were: men 3.50; women 2.78; boys 2.27; and girls 1.65. Only 166 (5.3%) of the men and 41 (1.2%) of the women had blood lead concentrations above 10 µg/dl; none of the children were found to have levels above this value. Of the 6528 adults included in the survey only 11 had levels above 25 µg/dl.

There has also been a detailed investigated into blood lead levels in children aged 2½ in the Avon area (Avon Longitudinal Study of Pregnancy and Childhood). These results were presented at the IEH workshop on “Recent UK blood lead surveys” on 4 March 1997. Blood lead levels were obtained from 584 children; the arithmetic mean was 4.20, the median 3.3 and the geometric mean 3.44 µg/dl. The levels ranged from 0.8 to 27.6 µg/dl and showed a skewed distribution. Results showed that 94.6% of this sample had blood lead levels less than 10 µg/dl, with only 0.3% having a blood lead level ≥20 µg/dl (the remainder having levels between 10 and 20 µg/dl).

Watt et al. (1996) found that in a survey involving 342 mothers in Glasgow, none had a blood lead concentration above 25 µg/dl. In a random sample of mothers, the geometric mean maternal blood lead concentration was 3.65 µg/dl. In mothers whose tap water lead concentrations were consistently below 2 µg/l, the geometric mean blood lead level was reported as 3.16 µg/dl.
Blood lead levels in general population surveys around the world also show a major decrease after the introduction of measures to reduce lead exposure. In the US the National Health and Nutrition Examination Survey for 1988-1991 (NHANES III) showed that efforts to reduce lead in gasoline and soldered cans containing food were associated with a 78% decrease in blood lead levels in the US population compared with the 1976-1980 survey.

5.1.3 IQ

There have been numerous studies in the past 15 years that have investigated the relationship between blood lead (or tooth lead) and cognitive development in children. These studies have included cross-sectional studies and prospective studies and most have used either a full or short form of the 'Wechsler Intelligence Scale for Children - Revised', as the measure of intelligence. The relationship reported between lead exposure during early development and later deficits in intellectual and academic performance has been remarkably consistent. However, it must be emphasised that the overall evidence indicates that for low level lead exposure the effect on IQ is very small, and that the relationship may not necessarily be a causal one. This should also be viewed from the perspective that other factors such as social background have a much greater rôle and are likely to have a substantially larger impact on intellectual performance.

Cross-sectional epidemiological studies have been conducted in Europe, including Eastern Europe, USA, Australia, New Zealand and China. In these studies, samples of children are identified and their IQ and blood lead measured, to test whether there is an association between the two. In addition, a number of prospective studies have been carried out in USA (Cleveland, Boston and Cincinnati) and Australia (Port Pirie and Sydney) which identify a sample of children at or before birth and follow them as they develop. The majority of the studies indicate that there is a relationship between lead exposure and IQ deficits, although the magnitude of the lead IQ association varies between individual studies. There is also little evidence of a threshold in the studies.

Since any single study has considerable random error in estimating any relationship, a quantitative overview (or meta-analysis) can be a valuable means of defining what is the plausible magnitude of statistical association between blood lead measures and child IQ. Rather than reviewing each individual epidemiological study, we have therefore focused on those reports which have undertaken a meta-analysis of individual studies (Pocock et al. 1994; Schwartz 1994a; Schwartz 1993; Needleman and Gatsonis 1990).

Pocock et al. (1994) carried out a systematic review of 26 epidemiological studies which have been published since 1979, which included both cross-sectional and prospective studies. The review included studies which had measured blood lead and dentine lead levels. The authors demonstrated that results from the five prospective studies in relation to exposure measures taken at or around birth, did not show any statistical association with later measures of IQ (nor did measurements of maternal ante-natal blood). However, blood lead measures taken at 2 years did show fairly strong evidence of an inverse association with IQ. The estimated mean change was -1.85 points (95% confidence interval (CI) = -0.85 to -2.85 points) for a change in blood lead from 10 to 20 μg/dl. A
mean of all post-natal blood measures showed rather less convincing evidence of an association than that of the 2 year old measures. The 14 cross sectional studies using blood lead showed more convincing evidence of an association, but greater variability. The mean change in IQ based on a meta-analysis of cross-sectional studies was -2.53 (standard error 0.41) for a change in blood lead from 10 to 20 μg/dl. However, if the extreme value from the Shanghai study is excluded, the mean change in IQ becomes -1.74 (standard error 0.43). Following an overall meta-analysis, the authors concluded that an increase in blood lead from 10 to 20 μg/dl was associated with a mean deficit in full scale IQ of 1 to 2 points. However, the authors also commented that, given the observational nature of the studies, the evidence is inconclusive on the causal rôle of low level lead exposure.

A meta-analysis by Schwartz (1994a) included seven epidemiological studies relating blood lead to full scale IQ in school age children. Three prospective and four cross-sectional studies were included in the meta-analysis. An increase in blood lead from 10 to 20 μg/dl was associated with a decrease of 2.6 IQ points (standard error 0.41) in this study which is similar to the results reported by Pocock et al. (1994). Schwartz also concluded that there is unlikely to be a threshold for this effect, but the evidence did point to a causal rôle. Previously Schwartz (1993) had analysed seven epidemiological studies and reported that, overall, the data indicated an average decrease of 0.25 IQ points occurred for each 1.0 μg/dl increase in blood lead levels. This inverse relationship between IQ and blood lead levels continued below 10 μg/dl.

In the International Programme for Chemical Safety (IPCS) evaluation on inorganic lead, two separate meta-analyses dealing with prospective and cross-sectional studies were undertaken. Overall, it was considered that there was a mean decrease in full scale IQ of the order of 2 for a change in mean blood levels from 10 to 20 μg/dl (IPCS 1995). Similarly, the USEPA (1984) considered that blood lead levels of 15 to 30 μg/dl can be associated with IQ losses of 1 to 2 points, blood lead levels of 30 to 50 μg/dl can be associated with IQ losses of about 4 points and over 50 μg/dl can correlate with losses of 5 points.

The question as to whether the effects on IQ during early childhood exposure are persistent beyond childhood cannot be answered unequivocally because current data are insufficient. One of the difficulties is that there are virtually no data available concerning the effects of removing children from a "high" exposure environment to one of a "low" exposure or on reduction of body lead burden in children. However, there are recent data which suggest that exposure to environmental lead during the first seven years of life is associated with cognitive deficits that seem to persist into later childhood (Tong et al. 1996). This study measured IQ for children at ages 11 to 13 whose histories of blood lead concentrations had been followed. The children's blood lead levels increased sharply during the first two years of life and then subsequently declined gradually, with the mean concentration at 11 to 13 years being slightly lower than the level recorded at birth. The authors report that the inverse associations between blood lead and cognitive development at ages 2, 4 and 7 years persisted into later childhood, even though blood lead levels had declined. The estimated deficit in full-scale IQ at age 11 to 13 years was 3 points for a shift in lifetime average blood lead concentration from 10 to 20 μg/dl. The
Boston cohort study also gave some evidence of a persistent IQ effect and reported that blood lead measurements at 2 years of age were associated with cognitive development at ages 57 months and 10 years (Bellinger et al. 1992).

Two other studies have also found that raised concentrations of dentine lead were associated with deficits in neuropsychological functioning that persisted into older childhood or young adulthood (Needleman et al. 1990, Fergusson and Horwood 1993). Fergusson and Horwood (1993) fitted a model to their data which was consistent with the view that early elevated lead levels up to the age of 8 were associated with a constant setback in the development of cognitive abilities persisting at least to the age of 12 years. There was no evidence to suggest that children with early elevated lead levels showed a tendency to catch-up, but equally no evidence to suggest the performance of the children tended to progressively deteriorate with the passage of time.

However, Rabinowitz (1993) reported on a small study carried out in Taiwan in which a decrease in blood lead level was associated with an improvement in IQ in a group of young children. In this study, children attending a kindergarten adjacent to a lead battery recycling factory were exposed to lead beginning near the age of 3 and lasting for 1 to 2 years, until the kindergarten was closed. Thirty two children were examined whilst still exposed and, 2½ years after the closing, 28 of them were re-examined. During this time, without medical intervention, the median blood lead level decreased from 15.6 to 8.5 µg/dl among the 28 case patients and decreased from 8 to 7 µg/dl amongst the controls. Concurrently, the unadjusted average IQ score, which was 94 among the case patients, increased to 107 while it increased from 101 to 110 in the controls. Therefore the difference in IQ, which was initially 7, decreased to 2.5.

In their recent evaluation, the IPCS commented that it is more likely that neurophysiological effects seen during school years are to some degree irreversible. This has also been observed in later follow-up studies conducted in other non-lead topics of child development research.

In conclusion, for a doubling of blood lead from 10 to 20 µg/dl, the overall evidence would suggest that this is associated with a deficit of 2 full IQ points. The form of the relationship below 10 µg/dl is unclear, although it would not appear that there is a threshold. Some form of extrapolation is therefore required to elucidate the relationship between 0 and 10 µg/dl. Although it recognised that the relationship between blood lead and IQ may be curved, given the uncertainties in the impact of low blood lead levels we are suggesting that a linear extrapolation would be the most sensible approach, i.e. for a change of 0 to 10 µg/dl blood lead there is also a 2 point deficit in full scale IQ.

It would appear that any neurophysiological effects detected in children age 7 are to some degree irreversible at least to later childhood. However, the duration, intensity and timing of exposure to lead, as well as other social and familial factors, may influence the nature and degree of reversibility.
5.1.4 Pre-term delivery and birth weight

The evidence linking low-level lead exposure during pregnancy to reproductive effects is mixed. There have been some studies which have found decreased length of gestation in women whose blood lead levels were greater than 23 \( \mu g/dl \) (Moore et al. 1982); 12 \( \mu g/dl \) (Dietrich et al. 1986) or 15 \( \mu g/dl \) (McMichael et al. 1986). However, neither Bellinger et al. (1991) nor Graziano et al. (1990) found decreases in gestational length or other parameters of pregnancy in women with elevated blood lead levels. Lacey et al. (1985) similarly found no relationship between gestational age or birth weight and mother’s blood lead.

The results from studies which have investigated the effect of blood lead level on birth weight have also been mixed. In the Cincinnati prospective study, a significant reduction in birth weight was associated with maternal blood lead levels (Bornschein et al. 1989). In the Port Pirie study of 749 pregnancies, the proportion of pregnancies resulting in low birth weight infants was more than twice as high in Port Pirie women whose blood lead concentration averaged 10.4 \( \mu g/dl \) than in women outside Port Pirie (average blood lead level of 5.5 \( \mu g/dl \)). However, multiple regression analyses showed no significant association between low birth weight and maternal blood lead level. Ward et al. (1987) reported a significant simple relationship between placental lead concentrations and reduced birth weight and head circumference.

Other studies have not shown a significant association between birth weight and lead exposure. The Kosovo prospective study failed to detect any evidence of lead-related birth weight reduction in more than 900 births (Murphy et al. 1990) and Emhart et al. (1986) found no significant effect of lead on birth weight, birth length or head circumference.

IPCS (1995) concluded that, currently, the evidence for potential adverse reproductive effects in women is qualitative but suggest that pre-term delivery may be associated with maternal blood lead levels above 15 \( \mu g/dl \). However, there are insufficient data to provide the basis for estimation of a dose-effect relationship. Andrews et al. (1994) reviewed 25 studies in terms of prenatal lead exposure in relation to gestational age and birth weight and concluded that “because consistent results have not emerged from the studies, the issues of lead’s effect on foetal development have not been resolved”.

Note that Schwartz (1994b) included effects on gestational age in his cost-benefit analysis. The costs estimated with this endpoint were about 7% of the total costs (associated with IQ and blood pressure).

5.1.5 Blood pressure

A number of epidemiological studies during the past 5 to 10 years have reported positive associations between blood pressure and blood lead concentration (e.g. Pocock et al. 1988, Schwartz 1988; Pirkle et al. 1985). However, there have been other studies which have found no positive associations (e.g. Gartside 1988; Staessen et al. 1996). Furthermore, not all of those positive associations have been statistically significant, and
there is conflicting evidence as to whether effects occur on both systolic and diastolic pressure, differences between the sexes are not always consistent, and there is still mixed opinion on whether any association is causal. In the recent IPCS (1995) evaluation on lead, the overall conclusion was that there is a weak, but statistically significant, association between blood lead level and blood pressure, although there is some doubt on whether the relationship is causal. In 1993, a National Academy of Sciences panel supported an association between lead exposure and elevations in blood pressure (NRC 1993).

As for IQ, we have relied on those reports which have undertaken a meta-analysis of the epidemiological studies which investigated the relationship between blood lead and blood pressure.

Staessen et al. (1994) undertook an extensive meta-analysis of 23 studies of which 13 recruited participants from the general population, 4 included employees with clerical jobs and 6 included blue-collar workers. In total, the studies involved 33,141 subjects. The authors found that there was a weak positive association between blood lead level and blood pressure which was similar in both sexes. In all studies combined, a two fold increase in blood lead concentration was associated with a 1 mm Hg (95% CI = 0.4 to 1.6 mm Hg) increase in systolic pressure and a 0.6 mm Hg (95% CI = 0.2 to 1 mm Hg) increase in diastolic pressure.

Schwartz (1995) also undertook a meta-analysis of 15 studies and found that an increase of blood lead from 5 to 10 μg/dl was associated with an increase of 1.25 mm Hg (95% CI = 0.87 to 1.63 mm Hg) in systolic blood pressure in men. In a survey involving 7371 middle aged men from 24 British towns, Pocock et al. (1988) estimated a similar mean increase of 1.45 mm Hg in systolic blood pressure (95% CI = 0.47 to 2.43 mm Hg) was associated with a blood lead increase of 8 to 16 μg/dl. They also estimated that diastolic blood pressure was increased by 1.25 mm Hg (95% CI = 0.65 to 1.85 mm Hg). The authors also compared data from their study to three other large studies (which included NHANES II and two Welsh studies) and concluded that all studies were compatible with an increase in systolic pressure of approximately 1 to 2 mm Hg (which was more likely to be about 1 mm Hg) in men for a doubling of blood lead concentration.

Several of the individual studies showed adjusted plots of blood pressure versus blood lead. They showed dose-dependent increases in blood pressure, with increasing blood lead concentrations. The figures from Pocock et al. (1988), Schwartz (1988) and Orsua et al. (1985) all suggest that the dose-response is curved with no evidence of a threshold. The studies of Pirkle et al. (1985) and Schwartz (1988) found no evidence for a threshold down to the lowest blood lead levels recorded in the NHANES II survey (2 μg/dl). They also examined a variety of functional forms for the relationship, and found that a linear relationship between blood pressure and the logarithmic transformation of blood lead provided the best fit.

Recently, Bost et al. (1997) reported on the relationship between blood lead and blood pressure in a representative sample of the adult population in the UK. The study considered two groups of people. Group 1 included 2564 men and 2767 women including some on anti-hypertensive treatment, whereas Group 2 excluded those on anti-
hypertensive treatment and consisted of 2255 men and 2402 women. After controlling for the effects of other variables, a significant association was found only for diastolic pressure in both sexes for Group 1. When individuals on anti-hypertensive medication were excluded, blood lead remained significant for diastolic blood pressure in men but was not significant in women. Bost et al. (1997) estimated changes in diastolic blood pressure with various decreases in blood lead (see Table 5.2).

Therefore, to conclude, for a doubling of blood lead from 5 to 10 µg/dl it has been reported that there may be an increase in systolic blood pressure of around 1 mm Hg and there is likely to be a similar but slightly lower increase of 0.6 mm Hg in diastolic blood pressure; however, based on the reported confidence intervals, there is a high degree of uncertainty in the quantitative relationship and the increase in blood pressure could potentially range from 0.4 to about 2 mm Hg. The form of the blood lead and blood pressure relationship below 5 µg/dl is uncertain, although it would appear that there is no threshold and we therefore have to undertake some form of extrapolation. Some studies have suggested that a linear relationship between blood pressure and the logarithmic transform of blood lead have provided the best fit to their data. However, the use of such a relationship as a basis for extrapolations is probably undesirable in these circumstances, because the slope approaches infinity at low blood lead levels. Although it recognised that the relationship between blood lead and blood pressure may be curved, given the uncertainties we are suggesting that a linear extrapolation would be the most sensible approach for this study.

Table 5.2  Estimated changes in diastolic blood pressure with decreases in blood lead

<table>
<thead>
<tr>
<th>Blood lead (µg/dl)</th>
<th>Change in diastolic blood pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>From</td>
</tr>
<tr>
<td>Men</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>1.45</td>
</tr>
<tr>
<td>4.7</td>
<td>3.7</td>
</tr>
<tr>
<td>3.7</td>
<td>2.7</td>
</tr>
<tr>
<td>Women</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>1.45</td>
</tr>
<tr>
<td>3.6</td>
<td>2.6</td>
</tr>
<tr>
<td>2.6</td>
<td>1.6</td>
</tr>
</tbody>
</table>
5.1.6 Biochemical effects

Lead has been shown to inhibit the activity of a number of enzymes including some involved in haem synthesis. These include inhibition of δ-aminolaevulinate dehydratase at around 10 μg/dl, although this is not considered to be of clinical significance since increased levels of δ-aminolaevulinate acid (ALA) in urine only occur at higher blood lead levels of around 35 μg/dl. Lead also inhibits coproporphyrinogen oxidase and ferrochelatase, with blood lead levels of 40 μg/dl and 20 to 30 μg/dl being associated with increased coproporphyrin and protoporphyrin in the urine. In their recent evaluation, the IPCS (1995) concluded that blood lead levels that affect biochemical parameters and which may have clinical significance are above 20 μg/dl. Although some effects on enzymes are demonstrable at lower lead levels, it was concluded that the clinical significance is uncertain.

5.2 Relationships between lead in drinking water and lead in blood

5.2.1 Introduction

There have been a number of studies which have investigated the relationship between exposure to lead and blood lead concentration. Although it is generally accepted that the relationship is curved, the precise form is not clear and there is still considerable debate over the most appropriate model (i.e. cube root, polynomial or logarithmic) to describe the relationship of waterborne lead to blood lead. The highest estimate for the contribution of water lead to blood lead comes from the cube root and logarithmic models. Much lower estimates are obtained from a linear model (IPCS 1995). Elwood (1986) considered that while a cube root relationship described the situation well in areas with very high water lead levels, the use of such a relationship as a basis for extrapolations is probably undesirable because the slope approaches infinity at low water lead levels. This may lead to an over-estimation of the importance of water relative to other sources, at low levels.

The models which have been proposed for the relationship between water lead and blood lead were all based on data generated during the late 1970s and early 1980s (Moore et al. 1977, 1979; Thomas et al. 1979; Pocock et al. 1983; Sherlock et al. 1982, 1984; Elwood et al. 1984; Raab et al. 1987; DoE/MAFF 1982; Quinn and Sherlock 1986; Quinn and Sherlock 1990). The blood lead levels on which the relationships were predicted were much higher at this time than the current levels. Consequently, it would be preferable to make some form of adjustment to the models to allow for this.

The purpose of this section is discuss the relationships between lead in drinking later and lead in blood, and to outline the reasoning and sources of information on which the proposed relationship is based.
Relationships are particularly needed that would apply to:

a) children of school age, to enable the subsequent prediction of effects on intelligence;

b) adults, to enable the subsequent prediction of effects on blood pressure.

This is not to say that the effects of blood-lead on IQ and blood pressure are not manifestations of the concentrations of blood-lead at earlier stages in life. The epidemiological information on these effects is, however, mainly cross-sectional and so, for example, the influence of childhood blood-lead on blood pressure in middle age cannot be estimated. In view of this it is of no special help to know the relationship between water-lead and blood-lead in children, for estimating the effect on blood pressure in adulthood. Similarly the relationship between water-lead and blood-lead in babies is not going to be of much help in predicting later deficits in intelligence. Although some of the studies of intelligence have had a longitudinal dimension, the revealed relationships did not have age-specificity to the extent that longitudinal information on blood-lead must be used for the purpose of prediction.

5.2.2 Difficulties of estimating relationship between lead in drinking water and lead in blood

There is only one real difficulty in estimating a relationship between blood-lead and water-lead. This is that no such general relationship exists! The only hope of a general toxicological relationship would be between blood-lead and the total rate of intake of lead from all sources. Any relationship between blood-lead and a single source (e.g. water) must be conditional on an assumed contribution (either constant or stochastic) from all the other sources. This gives rise to two consequent difficulties:

1. The contributions of several sources of environmental lead appear to have changed over the past decade, because average levels of blood-lead in the UK population have fallen.

2. The key epidemiological studies of blood lead in relation to water lead did not attempt to quantify the effects of the other sources, and they were conducted before the changes responsible for (1) took place.

For these reasons there are no studies (or almost none) from which an up-to-date relationship could be taken directly.

5.2.3 Choice of model

Any model that is going to be relevant to current circumstances must therefore be somewhat speculative, as a degree of extrapolation cannot be avoided. In these circumstances it is more important that the model is toxicologically plausible than that it has enjoyed previous empirical success.
Ideas outlined by Chamberlain (1987) and others would suggest that the model for blood lead concentration $y$ should be approximately of the form

$$y = -k + \sqrt{(k^2 + k(a_1x_1 + a_2x_2 + a_3x_3...))}$$

where

- $x_i$ is a measure of the concentration of lead in the $i$th potential source of exposure,
- $a_i$ is a coefficient such that $a_i x_i$ represents the partial contribution of available lead from the $i$th source,
- $k$ is a constant dependent on the mechanism of uptake of lead from the gastro-intestinal tract and clearance of lead via the kidney.

If the contributions of all sources other than, say, water (= source 1) are assumed to be constant the equation would become

$$y = -k + \sqrt{(k^2 + k(a_1x_1 + A_1))}$$

where $A_1$, is constant absorbing the other terms.

This equation further simplifies to

$$y = -k + \sqrt{(k^2 + B_1 + b_1x_1)} \quad (1)$$

or, approximately, to

$$y = \sqrt{(B_1 + b_1x_1)} \quad (2)$$

where $k$, $B_1$ and $b_1$ are constants whose values have to be inserted.

Equation (1) would be preferable to (2) for extrapolating to low levels of lead concentration but involves the provision of a value for $k$. In Equation (2) a separate value for $k$ is not needed. This model represented by Equation (2) is analogous to that used in the Edinburgh lead study (Raab et al. 1987) and identical to that advocated by Laxen (1983).

5.2.4 Values of constants

Only one epidemiological study, as far as we know, has analysed its data using the above model. This was the Edinburgh study of primary school children. This study also used half hour fixed stagnation time sampling to assess water lead. For constant $b_1$ we can do no better than use their value which was 1.03 when $y$ and $x_i$ were measured in $\mu g/dl$ and $\mu g/l$ respectively.

To attempt an estimate of $B_1$, we need to look to more recent studies, as this value has to reflect the levels of non-water-related sources of lead at present. One very recent study (Watt et al. 1996) suggests a value, for mothers in Glasgow, of $3.16^2 = 9.99$. 

30
Other recent work (Delves et al. 1996) suggests that this estimate would be of the right order of magnitude for adults (male or female) but that even lower values might be more appropriate for children. If we assumed that the value of $B_1$ could be estimated as the square of the median blood-lead concentration in England and Wales (effectively assuming that less than half of the population of England and Wales have lead in their tap water) we would have the values shown in Table 5.3.

**Table 5.3 Estimated values of $B_1$**

<table>
<thead>
<tr>
<th>Population class</th>
<th>Median blood-lead µg/dl</th>
<th>Suggested $B_1$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>3.5</td>
<td>12.4</td>
</tr>
<tr>
<td>Women</td>
<td>2.7</td>
<td>7.2</td>
</tr>
<tr>
<td>Boys</td>
<td>2.3</td>
<td>5.2</td>
</tr>
<tr>
<td>Girls</td>
<td>1.7</td>
<td>2.7</td>
</tr>
<tr>
<td>Adults</td>
<td>-</td>
<td>10</td>
</tr>
<tr>
<td>Children</td>
<td>-</td>
<td>4</td>
</tr>
</tbody>
</table>

Approximate estimates are 10 for adults and 4 for children. The difference between these estimates may appear to be rather large but, later, when we wish to estimate changes in blood-lead consequent on changes in water-lead, the answers will not be very sensitive to our choice of value of $B_1$.

5.2.5 The equations

Blood lead (µg/dl)

\[
= \sqrt{(10 + \text{water lead}/\mu g/l)} \quad \text{for Adults}
\]

\[
= \sqrt{(4 + \text{water lead}/\mu g/l)} \quad \text{for Children}
\]

Note that the concentrations of lead in water refer to average intake. Further adjustments would be needed if the concentrations referred to first draw or random daytime samples.

5.3 Bioavailability of lead from water and orthophosphate dosing

One aspect of relating blood lead to water lead concentration which needs to be considered is whether orthophosphate dosing may reduce the bioavailability and thereby the absorption of lead by humans. Intestinal uptake of lead is reported at the same sites as phosphate and calcium and therefore these ions could reduce lead bioavailability by directly competing for these uptake sites. Orthophosphate may also complex dissolved lead ions in water and reduce uptake (Wroath and Fawell 1995). Indeed, Heard et al.
(1983) studied the uptake of lead in human volunteers ingesting lead chloride in distilled water and reported that increasing amounts of calcium and phosphate reduced lead uptake when given simultaneously. There have also been a number of studies which have suggested that lead is not as bioavailable in hard water areas compared to soft waters (Gallacher et al. 1983; Thomas et al. 1981). Although there have been no studies that we are aware of on the potential effect of orthophosphate dosing on lead absorption, it would seem likely that orthophosphate in drinking water will complex dissolved lead ions and reduce uptake. Indeed, studies in rats have shown that low phosphate diets can cause a threefold increase in absorption and enhanced lead retention (Wroath and Fawell 1995).

5.4 Conclusions

1. Blood lead levels have fallen significantly in the past decade which is largely as a result of the use of unleaded petrol, although other initiatives to reduce lead exposure such as banning lead solder in tins, controlled use of lead based paints and well as improvements in water treatment will also have had an impact.

2. The two toxic endpoints currently associated with low blood lead levels which can be quantified are IQ and blood pressure. However, it must be emphasised that there are large uncertainties as to whether the reported association between low level lead exposure on both these endpoints is causal. This question is a key one as it will make a huge impact on any cost benefit analyses. If the underlying relationships are not causal, the monetary benefits in reducing lead exposure still further may be minimal.

3. If one assumes that the associations are causal, overall the evidence for a lead-IQ dose response relationship would suggest that for every 1 μg/dl change in blood lead there is a change in full IQ deficit of 0.2 points. This is a very small effect and should be viewed from the perspective that other factors such as differences in social background have a much greater impact on intellectual performance.

4. It would appear that there is a 1 mm Hg increase in systolic blood pressure for a two fold increase in blood lead in the range 5 to 10 μg/dl. A similar but slightly lower increase of 0.6 mm Hg in diastolic blood pressure has also been reported.

5. Other toxic endpoints which occur at low blood lead levels include effects on gestational age and biochemical effects. However, given the low clinical significance of the biochemical effects below 20 μg/dl blood lead, it is considered unnecessary to include these in any cost benefit analyses. Currently, the evidence for potential adverse reproductive effects in women is qualitative and there are insufficient data to provide the basis for estimation of a dose-effect relationship.
6. A number of models have been reported to describe the relationship between waterborne lead and blood lead levels; all of these were based on data generated during the late 1970s and early 1980s. The blood lead levels on which the relationships were predicted were much higher at this time than the current levels. Consequently, adjustments are needed to account for the fall in blood lead levels since the original models were developed.

7. Although there have been no studies to date to assess the effect of orthophosphate dosing on lead absorption, it would seem likely that orthophosphate in drinking water will complex dissolved lead ions and reduce uptake. Should such a hypothesis be proven, this could have a significant impact on the relationship between water lead and blood lead concentration.

5.5 Key assumptions

- The reported associations between low level lead exposure and effects on IQ and blood pressure are causal.
- The dose response relationships for blood lead versus effects on IQ and blood pressure are linear and have no threshold.
- Blood lead concentrations are related to the square root of average water lead concentrations; equations to describe the relationship were developed for adults and children based on published data.
6. ESTIMATION AND VALUATION OF HEALTH EFFECTS

6.1 Introduction and general approach

This section presents the results of the estimation and valuation of health effects identified in the preceding analysis.

The general approach to the estimation of health impacts follows the exposure - damage - valuation methodology. The methodology provided estimates of benefits based on existing literature valuations for the outcomes of lead exposure. An alternative approach would be to ascertain directly, through a survey, consumers' willingness to pay to reduce their exposure to lead. This would have delivered a value which encompassed avoidance of illness, worry caused by increased health risk and desire to drink water of a high standard. Fieldwork would have been necessary. The survey approach was not taken because of its costliness and the expected difficulty consumers would have in attempting to fix a willingness to pay figure to reduce lead concentrations in drinking water, as a result of a poor understanding of the health benefits which might accrue.

6.1.1 Average exposure

Average exposure estimates were derived from the results of sampling in water company areas. The frequency distribution of sample results is given in Figure 6.1.

Sample results were converted from range estimates to point estimates using the range average and then from sample concentrations into average exposure levels, using an exposure factor. The exposure factors are used to convert random daytime sample concentrations to maximum and average concentrations. The exposure level used to value benefits is, however, the average exposure level. The average exposure level represents the aggregate exposure of the population affected, although specific individuals may have higher or lower exposure levels.

Where exposure is in excess of the standard, interpreted either as a mean or a maximum, exposure with the standard imposed is reduced to ensure that exposure levels are met in 2003 for the interim and 2013 for the final standard.

6.1.2 Estimates of populations affected

Estimates of the populations affected were obtained from the Government Actuary’s Office for each year in the study, 1998 to 2034. Estimates of the health effects are restricted to two vulnerable groups: populations aged 0 to 10 for IQ effects and males aged 40 to 70 for hypertension, cardiovascular heart disease (CHD) and stroke. Although some benefits may arise to other groups, the majority of benefits will occur in the groups under investigation.

35
Figure 6.1 Frequency distribution of lead sample concentrations

6.1.3 Rate of reduction in exposure without a change in the standard

Pipe replacement is ongoing in England and Wales and hence exposure will decline in the absence of a change to the standard. The revised standard therefore has the effect of bringing forward benefits that would arise in the future anyway. Benefits have been estimated on the basis of changes in exposure due to the standards in addition to any changes that would occur in the absence of the standard. The benefits that are forecast, therefore, are those due to reductions in lead exposure after the reductions due to ongoing pipe replacement have been forecast.

To calculate the changes in exposure due to the introduction of the standard it has been necessary to calculate the natural rate of pipe replacement. This has been calculated on the basis of a weighted average of the replacement rates for supply pipes, communication pipes and plumbing for all water zones in England and Wales. The weights used in the calculation reflect both the presence of lead pipes in a zone and the relative length of pipes which is used as a proxy for their influence on exposure.

The result is a natural rate of pipe replacement for England and Wales of 0.98% per year (0.98% of existing pipes are replaced each year). For the purposes of estimating the exposure in the ‘without’ case the exposures have been forecast to fall at a rate equal to the natural rate of lead pipe replacement. Figure 6.2 gives an example of the methodology used to forecast lead exposures.
Figure 6.2  Lead exposure forecasts
The diagram shows a number of different exposure profiles for a group of the population which has a current exposure level of A. In the absence of the adoption of the standard the profiles of exposure will fall over time because of natural pipe replacement. Adoption of the Interim standard means that exposure falls faster, such that by 2003 average exposure has fallen to C. If the standard were not adopted, average exposure would have been at B. The difference between B and C (rather than A and C) is the benefit attributed to the introduction of the standard. The benefits, therefore, are net of any benefits that would arise because of ongoing pipe replacement.

Following the achievement of the Interim standard in 2003 there are two possibilities:

- The final standard is not met, in which case average exposures are assumed, once again, to fall at the natural rate of pipe replacement. In this case average exposure falls to D in 2013.

- The final standard is met, in which case average exposure falls to E in 2013. Additional benefits, represented by the difference between D and E are therefore generated. The benefits calculated are those that are incremental to the benefits generated by the interim standard.

It should be noted that a delay in meeting the standard would reduce the level of benefits generated. This is because benefits which occur further in the future are attached lower weights through discounting and the scope for benefits is reduced because average exposure levels will be lower due to ongoing pipe replacement.

6.1.4 Valuation of health benefits

Two health effects have been subject to valuation in this study, earnings benefits from avoided decrements in IQ and avoided costs of circulatory disease.

6.2 IQ benefits

6.2.1 Introduction

The purpose of this section is to quantify the economic benefits arising from the predicted increases in the level of IQ among young children in the 0-10 year age group as a result of reductions in lead levels. This is achieved by quantifying the linkages which exist between IQ, years of schooling, and ultimate labour market performance - both in terms of the probability of employment and the level of wage secured. The basic premise is that a more intelligent population will have a higher overall productivity leading to greater economic growth, and that this higher productivity can be measured by the increased earnings accruing to the individuals whose IQ has been enhanced as a result of the reduction of lead in drinking water.

It is important from the outset to lay out clearly the assumptions which underpin this chain of logic.
Increases in earnings reflect genuine increases in productivity, leading to a higher level of national output, and not merely the ability of higher IQ individuals to secure a higher share of a given level of national output.

Labour markets function sufficiently smoothly for wages to be used as an accurate indicator of an individual's productivity in employment, and of the social value of that productivity - this is the standard assumption of neo-classical economics.

The literature which quantifies the connections between IQ, years of schooling and ultimate labour market performance is sufficiently reliable and relevant in the following senses:

- the statistical analysis on which the studies are based adequately controls for the presence of other factors influencing years of schooling and ultimate labour market performance, so that the coefficients used capture the effects of IQ alone.
- the studies used which are based on US evidence, provide an adequate proxy for the likely magnitude of these relationships in the UK context.

Subject to these caveats, the analysis presented below is thought to provide a coherent examination of the linkages between IQ, years of schooling and ultimate labour market performance. Indeed, to the extent that there is any bias in the results, it is likely to be a downward one, in that the study may be under-estimating the overall impact of IQ on society by failing to take into account the wider social gains attributable to a better-educated work-force, which would not be captured by changes in individual earnings.

6.2.2 Formulating the problem

Figure 6.3 provides an illustration of the chain of linkages which exist between IQ, years of schooling, labour-force participation and working wages, which enable a relationship to be traced between lead levels and an individual's ultimate level of earnings. An important point to note is that IQ impacts on labour market performance not only indirectly, via its effect on years of schooling, but also directly, since, even controlling for years of schooling, a lower IQ individual is still likely to receive a lower level of earnings.

This section outlines each of these linkages in turn.

Arrow 1: Impact of lead on years of schooling

It is thought that increases in the level of lead in blood result in adverse behavioural effects which have a negative impact on an individual's schooling performance, as measured by ultimate years of schooling completed. However, owing to an absence of scientific evidence on this linkage for the UK, the effect has been implicitly set to zero for the purposes of this study.
Source: Adapted from Schwartz (1994a)

Figure 6.3 Flow diagram of interactions from lead to earnings

Arrow 2: Impact of lead on IQ

The direct impact of lead on IQ has been investigated in some depth for the UK in earlier sections of this report. The conclusion reached was that for every 10 µg/dl increase in blood lead, there could be expected to be a 2-point drop in IQ. These results are comparable to those obtained for the USA by Schwartz (1994a) and Salkever (1995).

Arrow 3: Impact of IQ on years of schooling

Studies indicate that a student's IQ level is one of the factors that explains the number of years of schooling successfully completed. This evidence presented below reports the increase in years of schooling for each additional IQ point.

Arrow 4: Direct impact of IQ on probability of being employed

Research suggests that there is a positive correlation between IQ and the likelihood of being employed, controlling for the years of schooling an individual has received. This evidence reports the increase in the probability of having a job for each additional IQ point.
Arrow 5: Direct impact of IQ on wages

A direct positive link between IQ and the earnings an individual can expect to obtain once employed has been found, even when controlling for years of schooling. The effect is reported as the percentage increase in the wage rate for each additional IQ point.

Arrow 6: Impact of years of schooling on wages

Labour economists have explored in detail the impact of increasing education on an individual's earnings. The answer identifies the percentage increase in earnings that arises from an additional year of schooling. However, for the question at hand, it is crucial that the studies used adequately control for IQ, to ensure that there is no double-counting between Arrows 3 and 6. There are fewer studies of this link which successfully address this issue. Individuals with different levels of education have different earnings. Part of this is due to the fact that those who stay longer at school tend to have a higher IQ and thus earn more simply due to their greater ability. It is also due to the fact that more education itself develops skills which yield higher earnings. A good study will attempt to distinguish between these two factors. For Arrow 6, only the second component is relevant.

Arrow 7: Impact of years of schooling on participation

Again, the effect of schooling on participation has been the subject of analysis by labour economists. More education leads to a higher likelihood of employment, controlling for IQ. This effect is particularly marked for women.

Based on this conceptual framework, the overall effects of IQ on wages and on the probability of being employed can be cumulated (see Figure 6.4).

\[
\frac{\Delta W}{W} = 5 + (3 \times 6)
\]

\[
\frac{\Delta P}{P} = 4 + (3 \times 7)
\]

Figure 6.4 Equations referring to arrows in Figure 6.3
The first equation expresses the effect of a change in IQ on the percentage change in working wages (W), which can be broken down into a direct effect and an indirect effect via years of schooling.

The second equation expresses the effect of a change in IQ on the percentage change in the probability of labour-force participation (P), which can once again be decomposed into a direct effect and an indirect effect via years of schooling.

6.2.3 Literature survey

The purpose of the literature survey is to review the estimates which have been produced of each of the linkages represented by Arrows 3-7 in Figure 6.3 above. The most suitable estimates will be identified from the literature on each linkage, and will then be aggregated to give the overall effect of changes in IQ on wages and the likelihood of being employed, shown in Figure 6.4 above. This final step is performed in Section 6.2.4.

For each linkage examined below, many of the studies confirmed that the expected relationship held; however, it was not always possible to translate the reported results into suitable estimates for each Arrow. The particular interests of any study determine how the results are presented; where these interests do not accord with those under examination here, it can be difficult to derive useful point estimates of the desired relationships. This is particularly an issue for the evidence relating to participation. Because of the nature of the estimation method, it is not always possible to get the particular estimates required for this exercise without access to the data set, unless the authors have chosen to report them directly.

As a result, there is an over-reliance on the Salkever (1995) study to provide the particular estimates for the benefit analysis, since it undertook the specific econometric research to study the question of the impact of lead on lifetime earnings via its IQ effects in the US. To redress this imbalance would require a UK study of this specific question. However, the other studies discussed below confirm the direction and significance of the relationships at issue here. This means that, while there is some uncertainty about the size of the particular effect, its relevance is well-established. Appendix C describes some of these technical difficulties in more detail.

In the tables below, point estimates are reported. Where a range of values arise from the different research studies, it was deemed unnecessary to overlay these with confidence intervals for particular estimates. Below each table salient features of each study are discussed; more detail on each can be found in Appendix C.

---

2 A confidence interval gives the range in which the true value of the parameter of interest lies, with 95% certainty. The uncertainty is based solely on the statistical variation of the sample.
Arrow 3: Impact of IQ on years of schooling

Table 6.1 summarises the principal studies in the literature purporting to quantify the link between IQ and ultimate educational performance, as measured by years of schooling. All four studies cited are based on evidence from the USA.

<table>
<thead>
<tr>
<th>Author</th>
<th>Men</th>
<th>Women</th>
<th>All</th>
</tr>
</thead>
<tbody>
<tr>
<td>Salkever (1995)</td>
<td>0.10</td>
<td>0.10</td>
<td></td>
</tr>
<tr>
<td>Schwartz (1994a)</td>
<td></td>
<td></td>
<td>0.13</td>
</tr>
<tr>
<td>Waller (1971)</td>
<td>0.34</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reynolds and Brown (1975)</td>
<td></td>
<td></td>
<td>0.2-0.3</td>
</tr>
</tbody>
</table>

Note: Schwartz's coefficient is equivalent to arrows \(1 + (2 \times 3)\) in Figure 6.3.

All the studies indicate a positive, significant relationship between IQ and years of schooling. Waller uses different units. Hence while it confirms the existence of a relationship, the size of this effect is not directly comparable with the other three studies, nor can it be used in this study. (This effect is discussed in more detail in Appendix C).

The estimate of Reynolds and Brown (RB) is higher than the first two, because this work does not control for important factors such as family background - parent’s income and education - age, ethnicity and regional effects, in contrast to Salkever and Schwartz. It would be expected that ignoring these features would bias the estimate upwards, since, given two people with the same IQ, the one from the wealthier or more educated background is more likely to be highly educated. Indeed, RB’s estimate is two to three times higher than the first two estimates.

Schwartz’s estimate includes a direct effect from lead on schooling (via detrimental behavioural effects), as well as an indirect effect via IQ. Given that it has been determined that the former link does not hold in the UK, this estimate is also too high for the purpose of this study. For all of these reasons, it is deemed appropriate to use Salkever’s estimates of the coefficients for Arrow 3, i.e. a value of 0.1, indicating that an increase of 10 points in IQ could be expected to lead to an extra year of schooling.
Arrow 4: Direct impact of IQ on the probability of being employed

Table 6.2 summarises the main studies in the literature which examine the link between IQ and the probability of employment. The small number of estimates reflects that this is not a question that has received a large amount of attention in the literature, except for the similar undertaking in the US. The Salkkever and Schwartz studies are based on US evidence, whereas the study by Lynn et al. (1984) was performed in Northern Ireland.

Table 6.2 Coefficients for change in probability of employment for a given change in IQ (%)

<table>
<thead>
<tr>
<th>Author</th>
<th>Men</th>
<th>Women</th>
<th>All</th>
</tr>
</thead>
<tbody>
<tr>
<td>Salkkever (1995)</td>
<td>0.16</td>
<td>0.37</td>
<td></td>
</tr>
<tr>
<td>Schwartz (1994a)</td>
<td></td>
<td></td>
<td>0.47</td>
</tr>
<tr>
<td>Lynn et al. (1984)</td>
<td>0.23</td>
<td>0.139</td>
<td></td>
</tr>
</tbody>
</table>

Note: The figure from Schwartz is equivalent to arrows \((4+3 \times 7)\) in Figure 6.3.

Again, the three studies indicate a significant, positive effect from IQ to participation in the workforce. It might be expected that women are more responsive than men to incentives to participate, since lower IQ women may choose to stay at home and raise a family, whereas lower IQ men do not have that option. The evidence, however, is mixed. Of the two studies that divided the sample according to sex, one found women more responsive, the other less.

The Lynn et al. study unfortunately is not directly applicable to the question of interest, due to reporting incompatibilities. Also, it only examines the probability of being in work one year after leaving school and is based on a Northern Ireland sample. Both these points make it less applicable to lifetime earnings for individuals in the UK as a whole.

The Schwartz study does not look at this effect in isolation, but directly aggregates the total effect of changes in IQ on likelihood of being employed. While not directly comparable at this stage, this can be used directly in Section 6.2.4. Thus the Salkkever estimate for the impact of Arrow 4 alone is used. Taking the mean of the coefficients for men and women yields a value of 0.27%. This is the increase in the probability of employment which can be expected as a result of a one-point increase in IQ.
Arrow 5: Direct impact of IQ on wages

Results from the main studies in the literature which are concerned with quantifying the link between IQ and wages are summarised in Table 6.3. All of these studies are based on US evidence.

Table 6.3  Coefficients for change in wages per point change in IQ

<table>
<thead>
<tr>
<th>Author</th>
<th>Men</th>
<th>Women</th>
<th>All</th>
</tr>
</thead>
<tbody>
<tr>
<td>Salkever (1995)</td>
<td>1.24%</td>
<td>1.40%</td>
<td></td>
</tr>
<tr>
<td>Schwartz (1994a)</td>
<td></td>
<td></td>
<td>0.5%</td>
</tr>
<tr>
<td>Barth <em>et al.</em> (1984)</td>
<td></td>
<td></td>
<td>0.2%-0.75%</td>
</tr>
<tr>
<td>Griliches (1977)</td>
<td></td>
<td></td>
<td>0.5%</td>
</tr>
<tr>
<td>Reynolds and Brown (1975)</td>
<td>$42 (whites)</td>
<td>$18 (blacks)</td>
<td></td>
</tr>
<tr>
<td>Murnane <em>et al.</em> (1995)</td>
<td>0.53%</td>
<td>0.73%</td>
<td></td>
</tr>
</tbody>
</table>

Of these, the studies deemed to be of greatest relevance were Salkever (1995) and Murnane *et al.* (1995), because they are based on comparatively recent data, collected in 1990 and 1986 respectively. This is important in the case of wage effects which may vary significantly over time, reflecting structural changes in the economy.

Although the Murnane *et al.* study uses a different measure of IQ to that adopted by the rest of the literature - the mathematics component of a test of cognitive skills - it was possible to adjust the coefficient on the basis of complementary information provided in the study. It is this adjusted coefficient which is reported in the table. As in the Salkever study, the Murnane *et al.* study is careful to control for other factors correlated with IQ - such as years of schooling, ethnicity, work experience, full- or part-time status and geographical variables - which may have a potential impact on working wages. This provides some confidence that the coefficient obtained isolates the impact of IQ alone.

As described earlier, RB does not control for other relevant factors, implying these estimates are likely to be upwardly biased. In addition, because of the manner in which the results are reported they cannot be readily compared with the rest of the literature. The remaining two papers find a relatively lower effect of IQ on wages; as mentioned above, these are based on significantly older data sets.
Taking averages of the separate coefficients for men and women reported by each of the preferred studies gives values of 1.32% from Salkever and 0.63% from Murnane et al. for the wage impact of a one-point change in IQ. Both figures will be used to define a range of values for this effect.

Arrow 6: Impact of years of schooling on wages

There are many studies which include levels of education in analyses of earnings in the economic literature; however, for the question studied here, how ability effects are controlled for is paramount. There is a long history discussing whether ability effects will bias upwards estimates of the benefits of schooling, and how to study this question. Table 6.4 presents a range of estimates from studies which have examined the issue. This includes evidence from the UK as well as the US, but it is only the US work that controls carefully for ability. One UK study examines the social returns to schooling, in contrast to the private returns. In this case there are large number of other studies that would report a comparable coefficient; the chosen set are considered to be representative, if not comprehensive.

Table 6.4 Coefficients for change in wages per extra year of schooling (%)

<table>
<thead>
<tr>
<th>Author</th>
<th>Men</th>
<th>Women</th>
<th>All</th>
</tr>
</thead>
<tbody>
<tr>
<td>Salkever (1995)</td>
<td>4.88</td>
<td>10.08</td>
<td></td>
</tr>
<tr>
<td>Schwartz (1994a)</td>
<td>6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ashenfelter and Krueger (1994)</td>
<td>12-16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angrist and Krueger (1991)</td>
<td>6.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marin and Pscharopoulou (1982)</td>
<td>6 (all)</td>
<td>3 (manual)</td>
<td></td>
</tr>
<tr>
<td>Blanchflower and Oswald (1994)</td>
<td>4</td>
<td>2.9</td>
<td></td>
</tr>
<tr>
<td>Jenkins (1995)</td>
<td></td>
<td></td>
<td>5.2, 13.8</td>
</tr>
</tbody>
</table>

Ashenfelter and Krueger (1994) and Angrist and Krueger (1991) include careful controls for the direct effect of IQ as a separate factor affecting an individual’s wages. The former is based on a study of twins, thereby controlling for genetic differences in ability, in fact they find no upward ability bias in the schooling variable. Angrist and Krueger find a smaller 6.6% return to schooling, based on a study which uses Vietnam draft information.
to control for ability. Again, no upward bias due to ability was found. Schwartz’s analysis was based on a number of older US studies, some of which attempted to control for ability.

Turning to the UK evidence, there is no study which controls for ability. However, the US evidence suggests that, against expectations, this effect may not be important. Marin and Psacharopoulos are examining the relationship between risk and earnings, but also report estimates of the effect of schooling on wages. The average estimate of this effect was around 5.8%, based on 1975 earnings data. The more recent study by Blanchflower and Oswald uses data up to 1990. Again, the focus of their research is different, but includes years of schooling to examine the effect of education on wages. They find a lower average estimate of 3.5%.

The final UK study by Jenkins, which looks at the social returns of increased education over the last 20 years, finds a range of effects between 5.2% and 13.8% for the benefits of each year of post-highschool education. This study does not look at the returns to individuals’ earnings, but the benefits to society in terms of increased GDP as the workforce increases its educational attainment. Hence it perhaps comes closer to the goal of the study here, as it includes the private and social gains of increased education. However, the larger estimates also arise because this evidence only relates to the benefits from tertiary education, rather than a broader definition of schooling.

Based on this survey, it was decided that a conservative approach would be to use the Blanchflower and Oswald average estimate of 3.5% as a lower bound for the effect of a year of schooling on wages, and the Angrist and Krueger value of 6.6% as an upper bound for the effect of a year of schooling on wages.

**Arrow 7: Impact of years of schooling on probability of employment**

In general economic literature, the bulk of the evidence on what determines the likelihood of being in work examines women, or married women. This is because, historically, most men work. Studies of the labour supply of men focus on hours worked rather than the decision to work itself. Thus there are few studies which could shed light on this linkage: the effect of schooling on the probability that an average person works. The evidence from studies of women suggests that increasing years of schooling increases the probability of being in the workforce; however, as discussed above, the way that this evidence is reported makes it unsuitable for providing particular estimates of Arrow 7.

Table 6.5 summarises the evidence from the two US studies of the links between lead, IQ and earnings, the methodological features of which have been described in some detail above.
Table 6.5  Coefficients for change in probability of employment per extra year of schooling (%)

<table>
<thead>
<tr>
<th>Author</th>
<th>Men</th>
<th>Women</th>
<th>All</th>
</tr>
</thead>
<tbody>
<tr>
<td>Salkever</td>
<td>0.35</td>
<td>2.82</td>
<td>1.58</td>
</tr>
<tr>
<td>Schwartz</td>
<td></td>
<td>0.47</td>
<td></td>
</tr>
</tbody>
</table>

Note: The figure from Schwartz is equivalent to arrows \([4 + 3 \times 7]\) in Figure 6.3

While the Schwartz figure presented earlier is not comparable to the individual estimates from Salkever's work, once the latter estimates are aggregated, there is comparability. This is done in the next section.

6.2.4 Overall impact of IQ on earnings

It is now possible to put together the different coefficient values selected above to quantify the overall impact of IQ on earnings, both directly and indirectly via the years of schooling effect. To begin with, the coefficients selected to quantify each of the arrows given in Figure 6.3 are summarised in Table 6.6.

Table 6.6  Summary of coefficients selected to quantify the various linkages between IQ and earnings

<table>
<thead>
<tr>
<th>Arrow</th>
<th>Coefficient (%)</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>3: IQ to years of schooling</td>
<td>0.1</td>
<td>Salkever (1995)</td>
</tr>
<tr>
<td>4: IQ to probability of employment</td>
<td>0.3</td>
<td>Salkever (1995)</td>
</tr>
<tr>
<td>5: IQ to working wages</td>
<td>0.6</td>
<td>Murnane et al. (1995)</td>
</tr>
<tr>
<td></td>
<td>1.3</td>
<td>Salkever (1995)</td>
</tr>
<tr>
<td>6: Schooling to working wages</td>
<td>3.5</td>
<td>Blanchflower and Oswald (1994)</td>
</tr>
<tr>
<td></td>
<td>6.6</td>
<td>Angrist and Krueger (1991)</td>
</tr>
<tr>
<td>7: Schooling to probability of employment</td>
<td>1.6</td>
<td>Salkever (1995)</td>
</tr>
</tbody>
</table>
Inserting these values into the two equations given in Figure 6.4, for percentage changes in working wages and probability of labour-force participation, arising from a one point change in IQ, yields the following results (Table 6.7).

**Table 6.7** Application of the formulae for percentage changes in wages and labour-force participation to the selected coefficients

<table>
<thead>
<tr>
<th>Formula</th>
<th>Direct effect</th>
<th>+ Indirect effect</th>
<th>= Overall effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\Delta W/W$</td>
<td>0.6% (lower bound)</td>
<td>+ [0.1 x 3.5%] (lower bound)</td>
<td>= 1.0% (lower bound)</td>
</tr>
<tr>
<td>1.3% (upper bound)</td>
<td>+ [0.1 x 6.6%] (upper bound)</td>
<td>= 2.0% (upper bound)</td>
<td></td>
</tr>
<tr>
<td>$\Delta P/P$</td>
<td>0.3%</td>
<td>+ [0.1 x 1.6%]</td>
<td>= 0.5%</td>
</tr>
</tbody>
</table>

Thus the percentage change in working wages is estimated to lie in the range 1-2%, while the percentage change in the probability of participation is estimated at 0.5%. This latter estimate is very similar to that estimated by Schwartz at 0.47%. The estimate for which there is most support for a higher value than the conservative one reported above is Arrow 6, the effect of schooling on wages. If a higher value of a 12% increase in wages for each additional year of schooling is taken, then the upper bound will be 2.5%, rather than 2%, for the first component.

Figure 6.5 below illustrates how these two figures can be summed together and multiplied by the baseline earnings levels, in order to calculate the percentage change in earnings ($E$) resulting from a one-point change in IQ. This will be described below as the IQ-earnings coefficient, and is estimated on the basis of the figures presented above to lie in the range 1.5-2.5%.

$$
\Delta E = \Delta P \cdot W + \Delta W \cdot P + \Delta P \cdot \Delta W
= \Delta P \cdot W + \Delta W \cdot P
= E \left( \frac{\Delta P}{P} + \frac{\Delta W}{W} \right)
$$

**Figure 6.5** Calculation of percentage change in expected earnings

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6.2.5 Valuation of IQ benefits from lead standards in England and Wales

Now that all these links have been established, it is possible to produce an aggregate valuation of the IQ benefits arising from a tightening of the lead standard in England and Wales. This involves incorporating estimates of:

- the change in IQ points to be expected as a result of the tightening standard;
- the baseline earnings of the population affected by the tightening standard.

The valuation is obtained by taking the product of:

- the IQ-earnings coefficient;
- the average change in IQ points in the affected population; and
- the average baseline level of earnings in the affected population (prior to the anticipated IQ change).

The calculation is fairly complex and was thus undertaken by spreadsheet. The main steps involved are summarised in Table C4.1 in Appendix C.

The results of applying this procedure are summarised in Tables 6.8 and 6.9. The 'lower' and 'upper' bounds correspond to the range of estimates for the IQ-earnings coefficient of 1.5% and 2.5% respectively, as established above.

Table 6.8 Valuation of IQ benefits from drinking water average lead standards, £m in 1998 in 1995 money

<table>
<thead>
<tr>
<th>Discount rate (%)</th>
<th>Limit</th>
<th>25 µg/l standard (implemented by 2003)</th>
<th>Additional 10 µg/l standard (implemented by 2013)</th>
<th>Aggregate benefit (implemented by 2013)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>Upper</td>
<td>1500</td>
<td>1200</td>
<td>2700</td>
</tr>
<tr>
<td></td>
<td>Lower</td>
<td>890</td>
<td>700</td>
<td>1600</td>
</tr>
<tr>
<td>4</td>
<td>Upper</td>
<td>910</td>
<td>640</td>
<td>1600</td>
</tr>
<tr>
<td></td>
<td>Lower</td>
<td>540</td>
<td>390</td>
<td>930</td>
</tr>
<tr>
<td>6</td>
<td>Upper</td>
<td>570</td>
<td>360</td>
<td>940</td>
</tr>
<tr>
<td></td>
<td>Lower</td>
<td>340</td>
<td>220</td>
<td>560</td>
</tr>
</tbody>
</table>
Table 6.9  Valuation of IQ benefits from drinking water maximum lead standards, £m in 1998 in 1995 money

<table>
<thead>
<tr>
<th>Discount rate (%)</th>
<th>Limit</th>
<th>25 µg/l standard (implemented by 2003)</th>
<th>Additional 10 µg/l standard (implemented by 2013)</th>
<th>Aggregate benefit (implemented by 2013)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>Upper</td>
<td>3200</td>
<td>1100</td>
<td>4300</td>
</tr>
<tr>
<td></td>
<td>Lower</td>
<td>1900</td>
<td>640</td>
<td>2600</td>
</tr>
<tr>
<td>4</td>
<td>Upper</td>
<td>1900</td>
<td>590</td>
<td>2500</td>
</tr>
<tr>
<td></td>
<td>Lower</td>
<td>1200</td>
<td>350</td>
<td>1500</td>
</tr>
<tr>
<td>6</td>
<td>Upper</td>
<td>1200</td>
<td>330</td>
<td>1500</td>
</tr>
<tr>
<td></td>
<td>Lower</td>
<td>730</td>
<td>200</td>
<td>930</td>
</tr>
</tbody>
</table>

6.2.6 Limitations of the study

Limitations can be identified in the analysis concerning:

- the geographical origins of the data;
- scaling issues affecting the units in which the coefficients are expressed;
- lack of differentiation between population sub-groups; and
- the issue of whether changes in earnings capture the underlying economic variables of interest.

Data issues

As a general observation, the data on the impact of variation in lead concentration is subject to a high degree of uncertainty, given the difficulty in quantifying the effects of IQ on productivity and performance. The best approach under these circumstances is to ensure that, where possible, conservative estimates are made, so as to underestimate rather than over-estimate the effect of lead concentration.

However, in this particular case, there is the additional problem that many of the figures used in this report are based on US studies - raising questions as to the transferability of the coefficients. In particular, the differences in employment structure and in returns to schooling should be appreciated when analysing the US data. If one supposes that the US
labour market possesses a greater degree of flexibility than that of the UK, and is more perfectly competitive in an economic sense, then the US wage level will tend to be more closely tied to the actual worker productivity. This makes US wages potentially more responsive to the IQ and school performance-related effects of lead exposure. As a result, when the US figures are applied to the UK, they may generate an over-estimate of the effect of changes in IQ on earnings. The potentially different returns to extra years of schooling in the USA and the UK create additional uncertainty regarding the accuracy of the figures.

Differentiation

A second possible set of criticisms of the results obtained comes from the suppression of differentiation within the data in calculating the cumulative effects of IQ on wage rates and earnings. Reynolds and Brown indicate that IQ effects on earnings could differ for different occupational groups. They find that the higher the occupational grouping, the larger the effect of IQ on earnings. That is, there is more reward for ability, the higher the level of ability. This non-linearity will not be captured in the simple averaging technique used in this study. Ideally, it would be desirable to estimate separate IQ-earnings coefficients for different occupational groups and reflect the occupational composition of the population as part of the aggregation procedure. However, the absence of data on both of these issues prevented the implementation of this more refined approach.

Social benefits

The analysis has assumed that higher earnings can be taken as a proxy for the higher productivity attaching to more intelligent and more highly educated people. There are potentially two problems with this assumption.

First, it could be argued that higher earnings do not necessarily reflect higher productivity, but rather the greater ability of more highly educated workers to negotiate higher levels of remuneration. If this were so, the higher earnings reported here would simply represent an income transfer between different portions of society, and not a true economic benefit attributable to higher productivity and thus output. To the extent that this observation is true, the values presented here could be seen as over-estimates.

Second, it could also be argued that increases in productivity are not the only benefit arising from a more highly educated population, which may generate wider social benefits (or positive externalities). Such intangible social benefits are not captured through the increased money value of wages commanded by individuals with higher IQ. To the extent that such positive externalities exist, then the values presented here could be seen as under-estimates.
6.2.7 Key assumptions

- It is assumed that a tangible relationship exists between lead exposure, IQ and earnings.
- It is assumed that changes in earnings capture social benefits of higher IQ: the extent that they do not do so leads to an underestimate of benefits.
- Results of US studies can be transferred to the UK; this may lead to a slight overestimate of benefits.
- Statistical studies adequately control for other effects and isolate only the effect of IQ; this may lead to an overestimate of benefits.
- Effect of IQ on earnings is linear (i.e. independent of baseline ability level). The effect on benefits is unknown.

6.3 Circulatory disease

6.3.1 Introduction

This section deals with valuing the economic benefits of reducing hypertension. The valuation can be divided into three components:

- reduced treatment costs;
- enhanced quality of life; and
- increased life expectancy.

This section first follows the epidemiological links from blood lead to hypertensive disease and blood pressure, which is a causative factor in coronary heart disease (CHD) and cerebrovascular accident (CVA) - stroke. The study goes on to estimate the benefits of a reduction in disease incidence for men aged 40-70, because this was the only group for which a blood lead/hypertension relationship had been demonstrated. This can be compared with the US EPA (1987) study which used a sub-population of white males in the age range 40-59.

6.3.2 Formulating the problem

Figure 6.6 below identifies the main linkages between blood lead, hypertension and subsequent health events leading to morbidity and premature mortality. It has been assumed that all stages of disease result in resource costs, but that uncomplicated hypertension does not significantly reduce quality of life.
The valuation of resource benefits is straightforward, since the resource benefit is equivalent to the National Health Service treatment cost saving. Two valuation methods were used for changes in quality and length of life:

\[
\text{Valuation method 1} = \text{willingness to pay for quality and length of life (quality-adjusted life years)}
\]

\[
\text{Valuation method 2} = \text{value of lost earnings through sickness and premature death}
\]

The second method, which only considers earnings benefits provides a check on the size of the more complete assessment of economic benefit provided by Method 1. The present study takes a more advanced approach than the US EPA, adjusting for quality of life and age at death. These adjustments convert morbidity and mortality statistics into quality-adjusted life-years (QALYs), and a related measure, quality-adjusted life expectancy (QALE).

![Flow diagram of causation of health benefits](image)

**Figure 6.6**  Flow diagram of causation of health benefits

The calculation of this sum was made on a spreadsheet for each year from 1998 to 2034, allowing disaggregation by age and the application of time discount rates of 2, 4 and 6%.

### 6.3.3 Impact of blood pressure on risk of disease

Several authors have reported evidence which links circulatory disease with blood pressure, both diastolic (DBP) and systolic (SBP), see Kannel (1970). In the past, DBP has carried more support as an indicator of risk, and has been the focus of investigation,
see Schwartz (1991) and MacMahon and Peto et al. (1990). Here, the results of the meta-analysis by Peto and MacMahon are applied to changes in DBP caused by changes in drinking water lead, and the overall relationship between lead and disease risk is supported by a study by Staessen (1994).

A paper by MacMahon and Peto et al. (1990) was used to provide estimates of blood-pressure risk factors. It surveys available international data, including data from the largest survey, the Framingham Study, conducted in the USA. Peto derived functions which link DBP in mm Hg to frequency of CVA and CHD. There are several characteristics of the study which make it particularly useful.

- The risk factors are independent of the level of DBP, with no detectable threshold for the effect of hypertension on risk, and evidence that the relationship continues below 70 mm Hg DBP. This makes the statistics easier to process because it justifies the application of risk changes to the whole population, not just the sub-set of hypertensives.

- The risk factors for DBP are independent of other risk factors, e.g. genetic risk factors - so any figure for a change in DBP can be applied across the whole population.

- The risk factors can be quoted with confidence because of the large size of the samples (Peto reports small values of standard deviation).

Risk-factor changes were found to be comparable across the 6-25 year follow-up periods of the original studies: the average follow-up period in the studies examined by Peto was ten years. For the purposes of this work, it is assumed that a change in DBP immediately changes disease risk. This may lead to an overestimate of the benefit of water-lead reduction in the early years of the programme.

The functional form of Peto's model was not suitable for direct extrapolation to small changes in DBP seen with improved drinking water standards. An assumption was needed to bridge the gap, and a straight-line extrapolation was chosen. The suitability of the assumption was verified via a verbal communication with Professor Peto (Peto 1997), who provided informal age-specific estimates for changes in risk of CVA and CHD associated with a 1 mm Hg change in DBP, see Figure 6.7. These are cited in Appendix D Table D2. The age-specific figures show that disease risk declines with age: for those towards the lower end of the affected age group the extrapolation underestimates risk, and it slightly overestimates risk for those towards the upper end of the age group.

The reduction in circulatory disease incidence as a result of the 25 µg/l programme is about 0.1% on average, and that for the 10 µg/l programme is 0.25%.
6.3.4 Resource benefits from lower costs of treating circulatory disease

This section estimates the resource benefits associated with fewer hypertensives as well as fewer patients suffering from CVA or CHD.

In the case of hypertension, costs arise both from screening the general population and from providing therapy for hypertensives. Screening costs are unaffected by reductions in BP across the population, but the therapy costs would be reduced because the number of patients in the hypertensive category would fall. It was not possible to split the resource costs between screening and therapy costs, so the total resource costs associated with hypertension were used instead. This may lead to a slight overestimate of hypertension resource benefits.

In the case of CHD, CVA and hypertension, direct estimates of health service resource costs were obtained for England and Wales (see Appendix D, Table D4). To find the benefits from the lead programme, the costs were scaled down by the blood-pressure risk factors from Appendix D, Table D1 and the change in blood pressure predicted as a result of the change in water lead. Hypertension costs were factored by the number of patients likely to be removed from the hypertension register. Figure 6.8 shows an example of the resource cost savings for the 25 μg/l standard at a 6% discount rate.

![Figure 6.7 Relationship between blood pressure and risk of CHD](image-url)
6.3.5 Valuation of morbidity and mortality

The final part of the sum for circulatory diseases, the valuation of changes in quality and length of life, is explained in this section. These methods provide figures which will be added to the resource benefits to give the total circulatory disease health benefits from the lead programme. As mentioned in Section 6.3.2, two methods are used. It is assumed in both that hypertension generates no significant morbidity other than through CVA or CVD.

Method 1: Estimation of quality of life forgone owing to circulatory disease

Introduction

This method uses QALYs, which enable morbidity and mortality effects to be assessed together. The quality of life and life expectancy of disease sufferers is compared against that of the rest of the population. The public is familiar with this form of presentation for smokers, for example ‘a 40-year-old smoking 30 a day will lose 5 years of life and suffer 5 years more of illness’ compared with a non-smoker.

The valuation equation is then of the form:

Value of morbidity and mortality = QALYs forgone × £ value of a QALY

Figure 6.8 Resource cost savings, totalling £50 m for 25 μg/l standard at 6% discount rate
Estimation of QALYs forgone through disease

CHD or CVA sufferers may experience premature death, sometimes combined with periods of illness. Health statistics give proportions of the population suffering from CHD and CVA in England and Wales, and numbers of deaths. The data were available differentiated by age and sex, so that 40-70 year-old males could be studied. Their quality of life, ranging from perfect health (scoring 1) to death (scoring 0) throughout their remaining life can be summarised by an index. The 0 to 1 score, known as a Q score, is based on survey questions which ask how many years of life, at a given age and state of health, individuals would be willing to exchange for another year of life at another age and state of health. This is called ‘time trade-off’. Surveys can be tailored to give Q scores for specific disease groups or the general population, and can be differentiated by sex and age.

Example data for the general population and ‘people not in perfect health’ is presented in Table 6.10. The figures were supplied by Paul Kind, University of York. They can be interpreted as follows. A man aged 80-84 with imperfect health would exchange a year of life with a man aged 25-29 in perfect health, at a ratio of 0.584/0.941 = 0.62. Thus, they value a year of their life at 0.62 of that of the latter group.

Table 6.10  Time trade-off quality of life scores

<table>
<thead>
<tr>
<th>Age group</th>
<th>Not with perfect EuroQol score</th>
<th>All respondents</th>
</tr>
</thead>
<tbody>
<tr>
<td>under 20</td>
<td>0.796</td>
<td>0.911</td>
</tr>
<tr>
<td>20-24</td>
<td>0.751</td>
<td>0.941</td>
</tr>
<tr>
<td>25-29</td>
<td>0.718</td>
<td>0.941</td>
</tr>
<tr>
<td>30-34</td>
<td>0.709</td>
<td>0.920</td>
</tr>
<tr>
<td>35-39</td>
<td>0.721</td>
<td>0.908</td>
</tr>
<tr>
<td>40-44</td>
<td>0.730</td>
<td>0.922</td>
</tr>
<tr>
<td>45-49</td>
<td>0.633</td>
<td>0.857</td>
</tr>
<tr>
<td>50-54</td>
<td>0.588</td>
<td>0.829</td>
</tr>
<tr>
<td>55-59</td>
<td>0.594</td>
<td>0.779</td>
</tr>
<tr>
<td>60-64</td>
<td>0.624</td>
<td>0.777</td>
</tr>
<tr>
<td>65-69</td>
<td>0.608</td>
<td>0.786</td>
</tr>
<tr>
<td>70-74</td>
<td>0.628</td>
<td>0.774</td>
</tr>
<tr>
<td>75-79</td>
<td>0.628</td>
<td>0.770</td>
</tr>
<tr>
<td>80-84</td>
<td>0.584</td>
<td>0.718</td>
</tr>
<tr>
<td>85 +</td>
<td>0.634</td>
<td>0.756</td>
</tr>
</tbody>
</table>

The literature on Q scores for circulatory diseases was examined and two fundamental problems were identified.
The first problem was that most studies are concerned with valuing the health impact of medical intervention in order to measure the cost-effectiveness of treatment. As a result, they measure the improvement in Q score as a result of treatment (distance ‘y’ in Figure 6.9 below). This is not the same as the difference in Q between persons with and without the disease (distance ‘x’ in Figure 6.9). For examples, see Johansson (1991, 1993) and Malcolm (1988).

Figure 6.9  QALY assessment

The second problem was that other Q scores published in the literature - see, for example, Kind (1988) and Fryback (1993) - relate to specific sub-categories of disease, such as heart bypass, angina, myocardial infarction and congestive heart failure, rather than to the condition as a whole. It was not possible to match these scores to the health statistics for England and Wales, either because the Q scores were not age-differentiated, or because the disaggregation of health statistics did not match the definitions of sub-categories of disease used by the surveys.

It was concluded that raw data Q scores for CVA and CHD were not available. Consequently, a second-best approach was adopted. This method relied on EuroQol Q-score data, cited above. The Q data gives time-trade off figures for persons who recorded no problems under the five main categories of the EuroQol classification scheme (i.e. had perfect 11111 classification), and for those who recorded one or more problems (i.e. had a non-11111 classification). It was then assumed that persons suffering from CHD or CVA have the same Q score as those with a non-11111 EuroQol classification. The non-11111 scores represent an average for all respondents in the EuroQol study who regarded themselves as being in less than perfect health. It is important to note that this second-best approach is based on the assumption that the degree of morbidity experienced by sufferers of CHD and CVA is comparable to that faced, on average, by people in less than perfect health. In the absence of Q scores for CVA and CHD, it is not possible to establish whether this represents an over- or underestimate.

The control population is made up of those in perfect health plus those in non-perfect health who are not suffering from either CVA or CHD (Figure 6.10). The average Q score for the control population is the weighted sum of the perfect and non-perfect health Q scores weighted by the numbers of people in each category. This provides the baseline quality of life against which the quality of life of CHD and CVA sufferers can be compared.
The QALY calculations are most simply explained as word equations, shown below, some of the components of which are illustrated. They give a total for QALYs forgone from circulatory disease in England and Wales in a typical year.

\[
\text{QALYs (CHD morbidity)} = \sum \text{sum over ages 40–70} \ [Q(\text{control}) - Q(\text{CHD})] \times \text{(number of sufferers)}
\]

\[
\text{QALYs (CHD mortality)} = \sum \text{sum over ages 40–70} \ [\text{quality-adjusted life expectancy}] \times \text{(number of deaths)}
\]

Similar equations represent the calculation for CVA morbidity and mortality.

The concept of quality-adjusted life expectancy, QALE, used in the valuation of mortality, which is an element in the second equation is illustrated in Figure 6.11. The figure shows actual life expectancy for the control population compared with QALE. QALE is always below raw life expectancy; this reflects the fact that later years of life are likely to be at a lower quality as a result of higher incidence of morbidity. An example calculation of QALYs lost by the death of a 48-year old is shown in the figure.
A detailed explanation of the manipulation of this data, together with the health statistics, is given in Appendix D, Figure D1. The calculation produced a total for QALYs lost as a result of circulatory disease in each year in the population of England and Wales. In order to convert this into a monetary value for the cost-benefit analysis, the monetary value of a single QALY had to be estimated. This process is explained in the following section.

**Valuation of a QALY**

This section explains the estimation of the monetary value of a single QALY, which will enable the QALYs obtained in the previous section to be converted into financial units. A figure for the *valuation of a statistical life* of £2m in 1995 terms was taken, this being typical of results obtained from stated and revealed preference estimates. This figure represents the average QALE lost when a person dies prematurely from an accident. For a representative population of 0 to 90 year-old males in England and Wales, the average QALE lost on death was calculated to be 31.1 QALYs. This gives a value for one QALY of £2 000 000 ÷ 31.1 = £64 221 in 1995 terms.

**Estimation of the benefits of a lead programme using QALYs**

The previous two sections calculated the value of QALYs lost from all circulatory disease in England and Wales. This valuation can now be factored down by the blood-pressure risk factors from Table D1 in Appendix D and the change in blood pressure predicted as a result of the changes in water lead. The reduction in circulatory disease incidence as a result of the 25 µg/l programme is about 0.1% on average, and that for the 10 µg/l programme is 0.25%. Values were calculated for each year between 1998 and 2034 and...
the figures discounted back to 1998 terms at both 2%, 4% and 6% discount rates. Illustrative numbers are shown in Figure 6.12. The full tables of benefits can be found in Appendix D.

Method 2: Estimation of wages forgone owing to circulatory disease

This method was performed as a back-up to the QALY valuation to act as a check on the plausibility of the QALY figures. It has two drawbacks compared with the QALY approach. First, it does not capture changes in quality of life, only earnings capacity. Second, as a consequence, no value is attributed to years of life beyond retirement age. The earnings figures used were taken from the New Earnings Survey and are listed in Appendix D.

Total earnings forgone owing to circulatory disease in England and Wales

Morbidity cost was evaluated as sick leave taken owing to circulatory disease. The calculation took the total number of days of sick leave owing to circulatory diseases from the Social Security Statistics (1995), which was 102 m in 1995, and assigned the days between CHD and CVA according to the incidence of those diseases. The earnings forgone each day were assumed to be one-fifth of the average weekly wage.

Mortality cost was equated to the net present value of future earnings from the age at death up until retirement at 65. The net present values were based on actuarial earnings multiple tables (HMSO 1994).

Figure 6.12 QALY valuation of morbidity and mortality, totalling £570m for 25 μg/l standard at a 6% discount rate
Estimation of the benefits of a lead programme using lost earnings

The previous two sections described the calculation of the value of lost earnings from all circulatory disease in England and Wales. As before, this valuation can now be factored down by the blood pressure predicted as a result of changes in water lead. The reduction in circulatory disease incidence as a result of the 25 \( \mu g/l \) programme is about 0.1% on average, and that for the 10 \( \mu g/l \) programme is 0.25%. Values were calculated for each year between 1998 and 2034 and the figures discounted back to 1998 terms at both 6%, 4% and 2% discount rates.

Figure 6.13 shows illustrative numbers. The full tables of benefits can be found in Appendix D.

![Diagram showing the distribution of lost earnings]

Figure 6.13  Lost earnings valuation of morbidity and mortality, totalling £120m for 25 \( \mu g/l \) standard at a 6% discount rate

6.3.6 Limitations

The valuation problem was formulated in three stages:

- the estimation of the effect of a change in blood pressure on risk of disease;
- the impact of changes in disease incidence on quality of life and years of life; and
- the valuation of those quality and length of life changes.
At each stage assumptions were made to make the quantification problem tractable. This section reviews those assumptions and also discusses the transferability of the references used to the situation in England and Wales.

**Effect of blood pressure on disease risk**

The risk factors used were based on a meta-analysis of studies across the world, but dominated by the USA. It is possible that the risk factors for England and Wales would be significantly different from the factors in other studies owing to exogenous factors acting in one region and not the other. This was investigated by the authors of the epidemiological paper as far as possible and no dependence on exogenous factors was found. The international data can therefore be used with confidence.

The magnitude of blood-pressure changes as a result of reduction of lead in drinking water is very small and below the range for which risk estimates have been published. Although the literature suggests that a change in risk will be observed even for small changes in blood pressure, the equation typically used to relate blood pressure to risk is not defined for small changes. This study assumed a straight-line approximation for small changes which may introduce bias.

**Incidence of mortality and morbidity**

Mortality statistics were decomposed into different categories, for example, ischaemic heart disease, angina, myocardial infarction and coronary heart disease. It was sometimes necessary to group these statistics together to generate figures for coronary heart disease and stroke. Where possible, the total number of deaths were cross-referenced and standard disease codes were examined. The statistics available for mortality were banded in age groups of no less than five years. It was assumed that the risk would be constant within each band, which is likely to introduce some bias, overstating the risk for younger persons and understating it for older persons.

The morbidity statistics available were not as complete as the mortality statistics, and had to be estimated from total disease incidence less mortality. No figures for disease-specific Q scores were available, neither was an analysis of the distribution of severity of disease among sufferers. Therefore it was assumed that sufferers would record the same Q score as persons with a non-perfect EuroQol score. It is likely that a large proportion of stroke patients in particular would record low Q scores because of serious disability, so the use of the EuroQol average score would underestimate the loss in quality of life and lead to an under valuation for stroke morbidity.

**Valuation of QALYs**

The final valuation figure is dependent on the valuation of a QALY. There is no well-established figure, so a value was calculated on the assumption that the value of a statistical life is £2m. This figure is subject to debate but can be regarded as within the central range of figures put forward by experts in the past.

64
In aggregating and discounting the figures for the period 1998 to 2034, no adjustment was made for increases in wealth which might lead to increases in real QALY values over time as willingness to pay increases. Although this could be said to give an under valuation of the benefits of the lead replacement programme, any adjustment would be very contentious. In defence of the approach taken, it should be noted that counterbalancing wealth increases will be improvements in medical technology which will increase Q scores in old age and with disease.

Comparison of QALY and lost earnings valuation results

The ratio mortality valuation to morbidity valuation is about 7:1 for the QALY method, but is reversed, at about 1:3 for the lost earnings method. As a result the QALY method produces a higher valuation than lost earnings for mortality, but a lower estimate for morbidity. The following points help to explain the differences.

- Under valuation of morbidity by QALY I. The disease prevalence statistics used in the QALY calculation may be understating the full extent of morbidity by setting a high threshold compared with the effect necessary to cause sick leave, nor do they include hypertension which is covered in the labour statistics. Even the lost earnings estimate, although it is the higher of the two, will still be an underestimate, because it does not measure full welfare cost.

- Under valuation of morbidity by QALY II. The assumption that CHD and CVA patients have Q scores equal to non-healthy persons may underestimate the detriment to health from these diseases. Furthermore, the assumption that there is no morbidity cost from hypertension, if untrue, would also lead to an underestimate.

- Under valuation of mortality by lost earnings method. The lost earnings method will underestimate mortality because the average annual wage is about one-third of the value of a QALY, and because it attaches no value to the deaths of 65-70 year olds, which account for over half the deaths in the 40-70 age group.
6.3.7 Key assumptions

- There is a straight-line relationship between DBP and risk. This leads to an underestimate of benefit for the young and an overestimate for older people.

- It is assumed that the immediate change in risk is the same as the long-term risk factor. This may produce an overestimate of benefits.

- Inclusion of all screening costs in the resource costs of disease may overestimate the benefits.

- CHD and CVA Q scores equal to non-11111 Q score. This assumption has an unknown impact on benefits.

- Choice of value of a statistical life of £2m. This leads to no systematic error, but is crucial in determining overall level of benefits.
7. NON-HEALTH EFFECTS

7.1 Introduction

The impact of changes in average lead exposure on health states will be accompanied by non health effects. The US EPA (1987) study, which examined the reduction in the US MAC for lead in drinking water from 50 µg/l to 20µg/l, found that the non health benefits (solely from avoided corrosion damage through increased treatment) were in fact larger than the health effects that could be valued. This section identifies the non-health effects which are important and where possible expresses these quantitatively in terms of their impact on the overall costs and benefits of a change in the lead standard.

In addition to corrosion damage a number of other non health effects can be identified:

- water treatment costs savings due to lead pipe replacement;
- dis-benefits arising from the disruption to householders and businesses caused by pipe replacement;
- monetary value of the lead salvaged;
- leakage reductions; and
- co-ordinated meter installation.

These are addressed in turn in the following sections.

7.2 Corrosion damage

In zones where compliance for lead can be achieved through treatment, this treatment may generate additional benefits in terms of avoided corrosion damage because the chemicals used to suppress lead solubility may also lead to reductions in the corrosion of other materials. Benefits from avoided corrosion may occur in the water companies distribution systems and/or in the household. The benefits of marginal increments in treatment for the water companies, however, are clearly going to be less than the costs of such treatment or we would expect the company to be undertaking a higher level of treatment already. Benefits to householders of avoided corrosion damage will be additional to those experienced by the water company. Numerous studies in the US have estimated that benefits to households may be as large as or larger than the benefits to the water distribution system (EPA 1987).

In the UK water treatment, for the control of lead, involves dosing with orthophosphate. Orthophosphate dosed water could affect distribution system materials in two ways:

- corrosion rate
- deposit stability
In the UK the trend is to use materials which are suited to the water type, and not to treat the water to ‘fit the materials’. Consequently corrosion rates are usually low and the dosing of orthophosphate only has a marginal effect on the rate at which materials corrode.

In the absence of any alteration, the water and deposits in a distribution system can be considered to be in a state of equilibrium. Any change to the system, be it hydraulic or chemical, is likely to result in a change to the equilibrium. There are some reports of orthophosphates providing a stabilising effect on distribution deposits, but others report no such effect. When orthophosphate dosing is stopped a change in the deposits equilibrium is expected, but the exact nature of this change cannot be predicted. In some cases the deposits may be more likely to give rise to discoloration events.

The effect of water treatment on materials other than lead is not marked. Consequently dosing is unlikely to be continued once all lead pipes in a zone are removed. No allowance is made for the effect of orthophosphate dosing on other materials since this is expected to be very small.

In the UK, therefore, increased orthophosphate dosing as a result of the new lead standard is not expected to give rise to significant reductions in corrosion damage for either water company assets or household materials and appliances.

### 7.3 Treatment savings

The lead pipe replacement programme can be expected to reduce the need for orthophosphate dosing to control for lead concentration in those zones where pipe replacement is used to achieve compliance. Avoided treatment costs are therefore an additional benefit of the lead pipe replacement programme.

To place a value on these savings the annual costs of treatment in each of the zones where lead pipe replacement takes place have been calculated. These savings represent avoided expenditure on chemicals. No costs are assumed to arise from avoided expenditure on capital equipment required for treatment. In practice avoided treatment would also result in reduced expenditure on capital equipment where this needs to be replaced or maintained in the “do nothing” scenario. In the absence of data on the avoided capital costs the valuation of savings has been restricted to the avoided expenditure on treatment chemicals. To the extent that capital costs are saved, these savings are therefore minimum estimates.

Table 7.1 details the annual operating costs avoided under each of the scenarios.
Table 7.1  Annual avoided treatment costs, 1995 prices

<table>
<thead>
<tr>
<th>Lead standard and method</th>
<th>Annual operational cost saving £ thousand from stopping orthophosphate dosing</th>
</tr>
</thead>
<tbody>
<tr>
<td>25 µg/l 30 min stagnation mean followed by 10 µg/l 30 minute stagnation mean</td>
<td></td>
</tr>
<tr>
<td>Annual saving from year following compliance with interim standard.</td>
<td></td>
</tr>
<tr>
<td>- Scenario A (A,L)</td>
<td>962</td>
</tr>
<tr>
<td>- Scenario C (A,H)</td>
<td>964</td>
</tr>
<tr>
<td>Additional annual saving following compliance with final standard.</td>
<td></td>
</tr>
<tr>
<td>- Scenario A (A,L)</td>
<td>5319</td>
</tr>
<tr>
<td>- Scenario C (A,H)</td>
<td>2016</td>
</tr>
<tr>
<td>25 µg/l 30 min stagnation maximum followed by 10 µg/l 30 minute stagnation maximum</td>
<td></td>
</tr>
<tr>
<td>Annual saving from year following compliance with interim standard.</td>
<td></td>
</tr>
<tr>
<td>- Scenario B (M,L)</td>
<td>4746</td>
</tr>
<tr>
<td>- Scenario D (M,H)</td>
<td>2641</td>
</tr>
<tr>
<td>Additional annual saving following compliance with final standard.</td>
<td></td>
</tr>
<tr>
<td>- Scenario B (M,L)</td>
<td>zero</td>
</tr>
<tr>
<td>- Scenario D (M,H)</td>
<td>2843</td>
</tr>
<tr>
<td>Notes:</td>
<td></td>
</tr>
<tr>
<td>Scenario A (A, L)</td>
<td>the standard is regarded as a mean but treatment alone cannot achieve compliance with the final standard interpreted as an average. (average, low treatment success)</td>
</tr>
<tr>
<td>Scenario B (M, L)</td>
<td>the standard is interpreted as a maximum and treatment alone cannot achieve compliance with 25 µg/l or 10 µg/l as a maximum. (maximum, low treatment success)</td>
</tr>
<tr>
<td>Scenario C (A, H)</td>
<td>The standard is interpreted as a mean and treatment can meet the final standard interpreted as a mean. (average, high treatment success)</td>
</tr>
<tr>
<td>Scenario D (M, H)</td>
<td>the standard is regarded as a maximum and can achieve the interim but not the final standard interpreted as a maximum. (maximum, high treatment success)</td>
</tr>
</tbody>
</table>
Savings are assumed to be experienced in the years following achievement of compliance with the standards. The avoided treatment costs will be reduced over time as it becomes easier to meet the standards because of the "natural" reduction in lead concentrations as a result of ongoing pipe replacement. Hence the annual savings have been reduced over time to reflect the impact of ongoing pipe replacement on the ability of a company to meet the standards without treatment.

Table 7.2 summarises the estimated benefits, arising from avoided treatment costs between 1998 and 2034. The benefits are discounted back to 1998 on the basis of a 6% real discount rate and expressed in 1995 pounds.

Table 7.2 Summary of avoided treatment benefits 1998-2034 (£m in 1995 prices) discounted to 1998

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>25 µg/l</td>
<td>9</td>
<td>45</td>
<td>9</td>
<td>25</td>
</tr>
<tr>
<td>10 µg/l (additional)</td>
<td>24</td>
<td>-</td>
<td>9</td>
<td>13</td>
</tr>
<tr>
<td>Total</td>
<td>33</td>
<td>45</td>
<td>18</td>
<td>38</td>
</tr>
</tbody>
</table>

Overall avoided treatment benefits are highest in scenario B as would be expected as here the combination of the standard being interpreted as a maximum and the low success of treatment in achieving compliance within a zone means that lead pipe replacement will be greatest. The more lead pipes that are replaced the higher will be avoided treatment benefits. It should also be noted that under scenario B all the costs are borne in the first five years of the programme. In this scenario it is assumed that treatment alone cannot meet the interim standard interpreted as a maximum. Compliance, therefore, is achieved solely through pipe replacement.

7.4 Disruption costs

The disruption costs of a pipe replacement programme are potentially very significant. There are many ways in which external effects, such as disruption costs, might influence the overall costs and benefits of the programme, these may include:

- costs to public and businesses from travel time delays due to excavation etc.,
- "nuisance" costs from aural and visual interference during road works and excavation,
- household inconvenience including time costs from complying with replacement programme (allowing access to premises etc.), and
- losses of flora and fauna due to excavation.

These issues can be examined in turn.
7.4.1 Traffic disruption

The replacement of lead pipes to achieve compliance will inevitably lead to the imposition of additional costs on households and businesses in terms of increased vehicle operating costs and time costs from delays. A literature review identified several studies of the impact of road excavation to repair or maintain underground assets on traffic flows. Several studies have examined the direct (repair costs) and indirect (external traffic costs) of sewer collapse episodes. The general consensus of these studies is that the traffic costs are equivalent if not greater than the direct repair costs. The estimated repair costs and effects on traffic of a number of studies are summarised in Table 7.3 below.

Table 7.3 Direct and indirect costs of sewer collapse at current prices

<table>
<thead>
<tr>
<th>Study</th>
<th>Direct repair costs £</th>
<th>Indirect costs to traffic £</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sewer collapse - Preston (1980)</td>
<td>36 000</td>
<td>32 000</td>
</tr>
<tr>
<td>Sewer collapse - Stallybridge (1981)</td>
<td>38 000</td>
<td>60 000</td>
</tr>
<tr>
<td>Sewer renewal - Glasgow (1973)</td>
<td>410 000</td>
<td>200 000 to 900 000</td>
</tr>
<tr>
<td>Sewer collapse - Petersham (1979)</td>
<td>2 235 000</td>
<td>6 500 000</td>
</tr>
</tbody>
</table>

In addition, TRL have investigated a series of notional traffic disruption events in Reading. These studies confirm the estimate that the traffic disruption costs are often equal to, and sometimes substantially greater than, the direct engineering costs. These studies, however, tend to focus on unplanned events which affect major routes in built up areas (as this is where costs are likely to be most significant). As a consequence they do not provide a valid measure for use in conjunction with estimates of pipe replacement costs in the current study. The pipe replacement programme required for compliance with the final standard would involve the replacement of 2.4 to 5 million pipes in England and Wales between 1998 and 2013. The number of additional road traffic disruption events, however, will be lower than this number because:

- some pipes can be replaced as part of the ongoing pipe replacement programme,
- most pipes are away from major roads and most excavations will be of footpaths and gardens, and
- multiple pipes may be replaced simultaneously during an excavation event.

In the absence of information on these factors and without information on the disruption costs relating to pipe replacement programmes it has not been possible to value quantitatively this element of the non health effects.
7.4.2 Nuisance costs

Pipe excavation is likely to lead to additional amenity loss through noise and visual intrusion. In addition excavation can be an inconvenience to pedestrians, particularly to the elderly and families with young children. It has not been possible to provide a quantitative estimate of this impact.

7.4.3 Household inconvenience

The replacement of household plumbing will involve considerable inconvenience to householders. The perception of this inconvenience may be one of the largest obstacles to any lead pipe replacement programme. Inconvenience may arise through:

- time costs of allowing access to premises for the replacement of plumbing,
- residual dis-benefits following minimal reinstatement (the costs of replacement are restricted to minimal reinstatement of premises including gardens etc.), and
- transitional loss of water supply.

One way of valuing this inconvenience is to estimate the value of the time lost as a result of householders needing to give up their time, which would otherwise be spent working or in leisure, to allow pipes to be replaced.

The compliance model developed to assess the costs of meeting the standard assumes that where it is necessary to replace the communication pipe to a property to achieve compliance in that zone, the supply pipe and internal household plumbing will also be replaced. The number of properties affected in this way therefore will approximate the number of service pipes replaced.

In order to value this effect it is necessary to know the average duration of the plumbing replacement "event" and the value attached by householders to a unit of time lost. The duration of the plumbing replacement can be estimated from the cost of replacing the plumbing. This is estimated to be £442 in 1995 prices. An approximate time and material cost per hour would be £120. This indicates that the duration of the plumbing replacement exercise would be approximately 4 hours per household.

The overall time burden may be in excess of this estimate given that people must remain in their houses to wait for the arrival of a plumber. Often plumbers can only give approximate times and this will increase the overall level of time lost.

Values attached to a unit of lost time will vary considerably from person to person. Some individuals will undoubtedly need to take time off work, some individuals would be at home in any case. Where individuals are required to take time off work then the appropriate value of time lost would be a factor of the loss in earnings to the company affected (proxied by the total employment costs of the individual). In reality individuals would be likely to engage in mitigative behaviour (particularly the self employed) to
minimise the impact of lost time on productive output. In addition, given positive values of time, there is always pressure to offset any losses by undertaking productive activities or engaging in leisure activities that might be possible during pipe replacement. These factors suggest that a value of working time would overstate the true costs of lost time due to pipe replacement for those individuals experiencing losses in work time.

In many households work time need not be lost to allow pipe replacement to occur. Households will contain members who are not in full time employment or are retired or are “home-makers”. The appropriate value of time lost for these households will differ according to the displacement of activities they would otherwise undertake.

In the absence of data on the above effects a conservative assumption has been made by applying a standard appraisal time based on the Department of Transport (DoT) value of non working time. Using this estimate the value of time lost under each of the scenarios is summarised in Table 7.4.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>25 µg/l</td>
<td>7</td>
<td>49</td>
<td>7</td>
<td>24</td>
</tr>
<tr>
<td>10 µg/l (additional)</td>
<td>23</td>
<td>-</td>
<td>10</td>
<td>14</td>
</tr>
<tr>
<td>Total</td>
<td>29</td>
<td>49</td>
<td>18</td>
<td>37</td>
</tr>
</tbody>
</table>

The highest cost is again under scenario B as it is here that most pipes need to be replaced to achieve compliance.

7.4.4 Loss of flora and fauna

Excavation is likely to result in the loss of flora and consequently fauna from habitat disruption. Evidence of the costs imposed by such events has been displayed during debates regarding the costs of establishing cable company networks. Poor working practices tend to damage trees which tend to be highly valued especially in urban streets. Any attempts to mitigate these impacts would lead to increased operating costs for companies engaged in pipe replacement. Such costs are likely to be additional to those costs included in the compliance cost model as these relate only to minimal reinstatement.
7.5 Salvage

A further benefit arising from the lead pipe replacement programme will be the value of the lead pipes which are replaced. It should be recognised that there will be a variety of ownership rights including water companies, householders for service pipes and private and public sector property owners.

Table 7.5 summarises the number of lead pipes replaced as a result of the standards. In addition the number of pipes replaced as a result of the ongoing pipe replacement programme is included for comparison.

Table 7.5 Number of lead pipes replaced (million) 1988 to 2013

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>25 µg/l</td>
<td>0.70</td>
<td>5.00</td>
<td>0.70</td>
<td>2.40</td>
</tr>
<tr>
<td>10 µg/l (additional)</td>
<td>3.60</td>
<td>-</td>
<td>1.70</td>
<td>2.20</td>
</tr>
<tr>
<td>Total</td>
<td>4.30</td>
<td>5.00</td>
<td>2.40</td>
<td>4.60</td>
</tr>
<tr>
<td>Ongoing pipes replaced</td>
<td></td>
<td></td>
<td></td>
<td>2.12</td>
</tr>
</tbody>
</table>

The length of lead pipes used in this estimation is summarised in Table 7.6.

Table 7.6 Average length of lead pipes replaced

<table>
<thead>
<tr>
<th>Pipes</th>
<th>Length (m)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Communication</td>
<td>5.5</td>
</tr>
<tr>
<td>Supply</td>
<td>10.0</td>
</tr>
<tr>
<td>Plumbing</td>
<td>6.5</td>
</tr>
</tbody>
</table>

The value of scrap lead is estimated by the British Association of Secondary Metals to be £351 per metric tonne. This represents the pre melt price and is a six month average. It is further estimated that the weight of a metre of lead pipe is 3.5 kg (this is an average of several types of lead pipe used in water supply and plumbing). In 1995 prices this leads to an estimate of the value of a metre of lead pipe of £1.28. Given the number of lead pipes replaced over the period 1998 to 2013 the following discounted benefits are estimated (Table 7.7).
Table 7.7 Value of lead salvaged through the lead pipe replacement programme 1998 to 2013 discounted at 6% to 1998 at 1995 prices £m

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>25 µg/l</td>
<td>17</td>
<td>118</td>
<td>17</td>
<td>57</td>
</tr>
<tr>
<td>10 µg/l (additional)</td>
<td>56</td>
<td>-</td>
<td>26</td>
<td>34</td>
</tr>
<tr>
<td>Total</td>
<td>72</td>
<td>118</td>
<td>43</td>
<td>91</td>
</tr>
</tbody>
</table>

### 7.6 Leakage reductions

Benefits may arise from lead pipe replacement through:

- avoided losses of water, and
- reducing water company expenditure on leakage control programmes.

Ofwat estimate that leakage accounts for approximately 30% of water put into the distribution system, with 23% of this accounted for by water company rather than customer pipes (Ofwat 1996). A pipe replacement programme will inevitably reduce the volume of water lost through leakage. There may also be a slight reduction in the effort required by a water company in leak location. However, few zones would be affected in this way because the reduced leakage control benefits will only be significant in zones with a high proportion of lead pipes and where service pipes are normally targeted during leak location.

Leakage can be considered to comprise two components, background night flow losses and bursts. Some estimate of leakage from service pipes has been made. However, there is little detailed information available on the proportion of leaks from lead pipes. Table 7.8 details estimates of typical leakage losses on a per household basis.

To provide an estimate of the value of avoided water losses it is assumed that typical average leakage from service pipes is approximately 4.8 litres per property per hour. It is assumed that this leakage is 'unreported' and is thus only addressed by lead pipe replacement. Leakage is assumed to be evenly distributed across zone and pipe work materials.

The marginal value of water is subject to great uncertainty. Water companies have recently estimated incremental resource costs of new supply to be between 2.4 and 50 p per m³. The majority of water companies, however, estimate incremental costs at less than 20 p per m³. To incorporate uncertainty regarding the true marginal price of water values of 5 p and 20 p per m³ have been used in this analysis, Table 7.9. These are purely the private costs to the company of future supply.
Table 7.8 Typical leakage losses

<table>
<thead>
<tr>
<th>Leakage component</th>
<th>Flow rate (litres/property/hour)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Background night flow losses:</td>
<td></td>
</tr>
<tr>
<td>Communication pipes</td>
<td>3</td>
</tr>
<tr>
<td>Supply pipes</td>
<td>1</td>
</tr>
<tr>
<td>Service pipe bursts</td>
<td>0.8</td>
</tr>
<tr>
<td>(assuming 1 burst per 2000 properties)</td>
<td></td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>4.8</strong></td>
</tr>
</tbody>
</table>

Table 7.9 Annual value of avoided leakage losses (un-discounted 1995 prices)

<table>
<thead>
<tr>
<th>Lead standard and method</th>
<th>Annual cost saving £ million as a result of reduced leakage after service pipe replacement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marginal cost of water</td>
<td>5 pence per m³</td>
</tr>
<tr>
<td></td>
<td>20 pence per m³</td>
</tr>
<tr>
<td>25 µg/l 30 min stagnation mean followed by 10 µg/l 30 min stagnation mean</td>
<td></td>
</tr>
<tr>
<td>Annual saving from year 6</td>
<td></td>
</tr>
<tr>
<td>Scenario A</td>
<td>1.5</td>
</tr>
<tr>
<td>Scenario C</td>
<td>1.5</td>
</tr>
<tr>
<td>Additional annual saving from year 16</td>
<td></td>
</tr>
<tr>
<td>Scenario B</td>
<td>7.6</td>
</tr>
<tr>
<td>Scenario D</td>
<td>3.5</td>
</tr>
<tr>
<td>25 µg/l 30 min stagnation maximum followed by 10 µg/l 30 min stagnation maximum</td>
<td></td>
</tr>
<tr>
<td>Saving from year 6</td>
<td></td>
</tr>
<tr>
<td>Scenario A</td>
<td>10.6</td>
</tr>
<tr>
<td>Scenario C</td>
<td>4.9</td>
</tr>
<tr>
<td>Additional saving from year 16</td>
<td></td>
</tr>
<tr>
<td>Scenario B</td>
<td>0</td>
</tr>
<tr>
<td>Scenario D</td>
<td>4.5</td>
</tr>
</tbody>
</table>

76
These benefits will fall over time given ongoing pipe replacement. The benefits are assumed to arise following the achievement of compliance in a zone. Table 7.10 and Table 7.11 summarise the benefits due to the standard which will arise over the period 1998 to 2034.

Table 7.10  Benefits of avoided water losses, 1998 to 2034, discounted to 1998 at 1995 prices £m - marginal water price 5 pence per m³

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>25 µg/l</td>
<td>14</td>
<td>100</td>
<td>14</td>
<td>46</td>
</tr>
<tr>
<td>10 µg/l (additional)</td>
<td>35</td>
<td>-</td>
<td>16</td>
<td>20</td>
</tr>
<tr>
<td>Total</td>
<td>49</td>
<td>100</td>
<td>30</td>
<td>67</td>
</tr>
</tbody>
</table>

Table 7.11  Benefits of avoided water losses, 1998 to 2034, discounted to 1998 at 1995 prices £m - marginal water price 20 pence per m³

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>25 µg/l</td>
<td>57</td>
<td>399</td>
<td>57</td>
<td>187</td>
</tr>
<tr>
<td>10 µg/l (additional)</td>
<td>137</td>
<td>-</td>
<td>63</td>
<td>83</td>
</tr>
<tr>
<td>Total</td>
<td>195</td>
<td>399</td>
<td>121</td>
<td>269</td>
</tr>
</tbody>
</table>

External costs arise in the water industry which are not currently reflected in the prices paid for water. This would be reflected in a much higher marginal value of water. One of the most important social costs is the environmental impact of new supply creation. Any leakage saving which results in lower growth in supply capacity are likely to reduce the environmental burden of the water industry.

7.7 Co-ordinated meter installation

Meter installation which is co-ordinated with pipe replacement will be less expensive than otherwise. An estimate of the unit saving produced is summarised below.

If the cost of replacing a pipe is V, and the additional cost of putting in a boundary box when that pipe is installed is B, while the cost of installing a meter where a boundary box is present is M, then the costs of co-ordinating replacement are V+B+M. Alternatively the costs of installing a meter where no boundary box is present is N. The savings that result from co-ordination are therefore:

\[(V + B + M) - V + N\]
In the case of 32 mm 2 m short side pipe replacement this saving is estimated as £77. Hence co-ordinated replacement could save substantial sums. However, given the difficulty of predicting the number of meter installations that would take place with and without the introduction of the standard no valuation has been undertaken.

7.8 Summary

Figure 7.1 summarises the non health impacts which have been quantified in this study.

![Figure 7.1](image)

Figure 7.1 Summary of quantified non health effects

As can be seen the most important quantified non health effect is avoided water losses, if water is valued at 20p per m$^3$. If water losses are valued at 5p m$^3$ then the salvage benefits are greatest.

The estimated net non health benefits are substantially lower than those suggested in the US EPA study. This arises because the benefits of avoided corrosion damage are estimated to be much lower in the UK than in the US because of differences in the distribution system and methods of treatment. Nevertheless the non health effects which are valued are significant - particularly in terms of avoided water loss.
7.9 Key assumptions

- There is no significant impact on rates of corrosion damage in zones where increased treatment is used to achieve compliance.

- Treatment will be discontinued in zones where pipe replacement is used to achieve compliance.

- The value of time lost in households due to pipe replacement is equivalent to the DoT’s standard appraisal time.

- Lead salvaged from pipe replacement will realise the current price for scrap lead.

- The value of lost water can be approximated by the values of 5p m$^3$ and 20p m$^3$. 
8. COST BENEFIT APPRAISAL

8.1 Introduction

This section brings together the costs, health benefits and non health impacts of the change in the lead standard for the overall appraisal. The appraisal results are presented separately for meeting the standard interpreted as a mean and as a maximum and for the interim and final standards. A discount rate of 6% is used in all cases. Sensitivity results for lower discount rates (4% and 2%) are reported in Section 9.

8.2 The scenarios

Many of the estimated impacts discussed in previous chapters have been presented as ranges given the uncertainty associated with their estimation. This uncertainty is reflected in the overall appraisal through the use of best case and worst case scenarios:

- **Best Case** results are estimated on the basis of the highest benefits and the lowest costs derived from the separate evaluations of impact.

- **Worst Case** results are estimated on the basis of the lowest benefits and the highest costs.

The main drivers determining the range of costs and benefits are:

1. whether the standard is interpreted as a mean or maximum;
2. the assumptions regarding the relative success or otherwise of treatment in achieving compliance;
3. the upper and lower limits of the evaluations of IQ effects on lifetime earnings;
4. the upper and lower limits defined by the QALY and loss of earnings approaches to the valuation of resource, mortality and morbidity costs of circulatory disease; and
5. the high and low assumptions regarding the price associated with avoided water losses.

All costs and benefits are discounted at a 6% real interest rate over the period 1998 to 2034 and are presented in 1995 prices in Table 8.1 and Table 8.2. Costs are shown as negative, and benefits as positive, values; the net benefits are calculated as the sum of the (negative) costs and benefits.
Table 8.1  Net benefits of lead standard interpreted as a mean (£m in 1995 prices) discounted to 1998 from 2034

<table>
<thead>
<tr>
<th>Cost benefit element</th>
<th>Interim 25 µg/l</th>
<th>Final 10 µg/l total</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Worst case</td>
<td>Best case</td>
<td></td>
</tr>
<tr>
<td>Costs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Suppliers costs</td>
<td>-6</td>
<td>-6</td>
<td>-888</td>
</tr>
<tr>
<td>Householders costs</td>
<td>-17</td>
<td>-17</td>
<td>-2723</td>
</tr>
<tr>
<td>Total costs</td>
<td>-23</td>
<td>-23</td>
<td>-3610</td>
</tr>
<tr>
<td>Health benefits</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Avoided lost earnings from IQ</td>
<td>340</td>
<td>570</td>
<td>560</td>
</tr>
<tr>
<td>- Avoided CHD resource costs</td>
<td>12</td>
<td>12</td>
<td>21</td>
</tr>
<tr>
<td>- Avoided stroke resource costs</td>
<td>18</td>
<td>18</td>
<td>31</td>
</tr>
<tr>
<td>- Avoided hypertension resource costs</td>
<td>2</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>- Avoided CHD mortality</td>
<td>18</td>
<td>250</td>
<td>30</td>
</tr>
<tr>
<td>- Avoided stroke mortality</td>
<td>5</td>
<td>77</td>
<td>8</td>
</tr>
<tr>
<td>- Avoided hypertension mortality</td>
<td>0</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>- Avoided morbidity</td>
<td>52</td>
<td>55</td>
<td>89</td>
</tr>
<tr>
<td>Sub total health benefits</td>
<td>447</td>
<td>986</td>
<td>742</td>
</tr>
<tr>
<td>Non health effects</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Avoided treatment</td>
<td>9</td>
<td>9</td>
<td>33</td>
</tr>
<tr>
<td>- Time losses</td>
<td>-7</td>
<td>-7</td>
<td>-30</td>
</tr>
<tr>
<td>- Salvage benefits</td>
<td>17</td>
<td>17</td>
<td>72</td>
</tr>
<tr>
<td>- Avoided water loss</td>
<td>14</td>
<td>57</td>
<td>49</td>
</tr>
<tr>
<td>Sub total non health effects</td>
<td>33</td>
<td>76</td>
<td>124</td>
</tr>
<tr>
<td>Net benefit</td>
<td>457</td>
<td>1039</td>
<td>-2744</td>
</tr>
</tbody>
</table>
## Table 8.2 Net benefits of lead standard interpreted as a maximum (£m in 1995 prices) discounted to 1998 from 2034

<table>
<thead>
<tr>
<th>Cost benefit element</th>
<th>Interim 25 µg/l</th>
<th>Final 10 µg/l</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Worst case</td>
<td>Best case</td>
</tr>
<tr>
<td>Costs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Suppliers costs</td>
<td>-1601</td>
<td>-1601</td>
</tr>
<tr>
<td>Householders costs</td>
<td>-4297</td>
<td>-4308</td>
</tr>
<tr>
<td>Total costs</td>
<td>-5897</td>
<td>-5908</td>
</tr>
<tr>
<td>Health benefits</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Avoided lost earnings from IQ</td>
<td>730</td>
<td>930</td>
</tr>
<tr>
<td>Avoided CHD resource costs</td>
<td>29</td>
<td>36</td>
</tr>
<tr>
<td>Avoided stroke resource costs</td>
<td>43</td>
<td>54</td>
</tr>
<tr>
<td>Avoided hypertension resource costs</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Avoided CHD mortality</td>
<td>42</td>
<td>52</td>
</tr>
<tr>
<td>Avoided stroke mortality</td>
<td>11</td>
<td>14</td>
</tr>
<tr>
<td>Avoided hypertension mortality</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Avoided morbidity</td>
<td>122</td>
<td>156</td>
</tr>
<tr>
<td>Sub total health benefits</td>
<td>981</td>
<td>1247</td>
</tr>
<tr>
<td>Non health effects</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Avoided treatment</td>
<td>45</td>
<td>45</td>
</tr>
<tr>
<td>Time losses</td>
<td>-49</td>
<td>-49</td>
</tr>
<tr>
<td>Salvage benefits</td>
<td>118</td>
<td>118</td>
</tr>
<tr>
<td>Avoided water loss</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Sub total non health effects</td>
<td>214</td>
<td>214</td>
</tr>
<tr>
<td>Net benefit</td>
<td>-4702</td>
<td>-4447</td>
</tr>
</tbody>
</table>

Figures 8.1 and 8.2 show the interim and final costs and benefits for the standard interpreted as a mean and a maximum.

These diagrams facilitate a comparison of the relative magnitude of costs and benefits associated with the interim and final standards. Section 9 presents a discussion of the results.
Figure 8.1  Summary of interim standard costs and benefits

Figure 8.2  Summary of final standard costs and benefits
9. DISCUSSION

9.1 Results

Any interpretation of the results must take into account the underlying uncertainty regarding the impacts quantitatively valued in this study. Numerous assumptions had to be made in estimating the costs of compliance (see Miller et al. 1997); a further set of assumptions was necessary in order to estimate the health and non-health benefits and to monetarise them. The benefits were assessed using conventionally accepted economic methods.

The estimated benefits are considered to be reasonable considering the uncertainties in some of the data; as an example the mortality effects of lead in relation to blood pressure have been valued, but there is still uncertainty over whether there is a genuine causal relationship between blood lead levels and blood pressure (Section 5.1.5).

The following aspects of the economic results should be noted:

a) Where the standard is interpreted as a mean the interim standard produces a net economic benefit on the basis of both worst and best case outcomes (£457m to £1039m). The additional costs of moving to the final standard, however, are higher than the additional benefits in both best and worst case scenarios. Thus a net economic loss results for the final standard under the worst case, and overall benefits, although remaining positive, are reduced for the best case.

b) The additional costs of moving from the interim to final standards mean that the whole programme, interim and final standards, only generate a net benefit under best case assumptions.

c) Interpreted as a maximum, the benefits outweigh the costs of meeting the interim as a best case but not as a worst case. This differs from the results where the standard is interpreted as a mean in which case under both best case and worst case scenarios the net benefit of the interim standard is positive.

d) Under best case assumptions the whole programme for the standard interpreted as a maximum gives a net economic loss of £408m.

Comparisons of best and worst case costs give an indication of the potential range of outcomes given the uncertainty attached to various scenarios. However, they may not be the most appropriate form of interpretation given the large difference between best and worst case costs for a number of items valued.

It is therefore important to compare the composition of costs both in, and between, best and worst case scenarios. These comparisons can be made with reference to Figures 8.1 and 8.2. The following points should be noted:
Where the standard is interpreted as a mean the costs (£3610m) in the worse case scenario are sufficient to outweigh the best case benefits (£1815m, i.e. $1651 + 164$).

When the standard is interpreted as a maximum the worst case costs are in excess of best case benefits for both the interim and final standards.

The following table summarises the result of a best case - worst case assessment of the likely costs and benefits for the interim and final standards, interpreted as a mean and as a maximum. Only the interim standard produces a net benefit in the best and worst case scenarios. The final standard interpreted as a maximum produces a net loss in both cases. Meeting the interim as a maximum or the final standard as a mean produces a net benefit only under best case assumptions.

<table>
<thead>
<tr>
<th>Standard</th>
<th>Mean</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>25 µg/l Interim</td>
<td>Net benefit in best and worst case scenarios.</td>
<td>Net benefit in best case scenario only.</td>
</tr>
<tr>
<td>10 µg/l Final</td>
<td>Net benefit in best case scenario only.</td>
<td>Net loss in best and worst case scenarios.</td>
</tr>
</tbody>
</table>

The balance between costs and benefits and the extent of any net benefit depend critically on the degree of success of water treatment. The success or otherwise of water treatment has a critical effect on the outcome of the standards. Information obtained during the cost study showed that water treatment could achieve a 10 µg/l standard in some zones but there are insufficient data to make confident predictions of the overall success of water treatment to meet the proposed standards. Water treatment is cheaper than pipe replacement, so the more successful water treatment is the lower the cost.

9.2 Sensitivity

The appraisal results are based upon a discount rate of 6%. This is the standard treasury discount rate used in project appraisal. Sensitivities have been undertaken on two lower discount rates of 4% and 2%. Table 9.1 summarises the net benefits for the three discount rates.

The most important point to note is the change in sign as we move from higher to lower discount rates. With the standard interpreted as a mean the net additional benefits of the 10 µg/l standard change from negative under a 6% discount rate to positive for the 4% and 2% rates. However, the overall net benefits of the whole programme were already positive because of the magnitude of the difference between costs and benefits for the interim standard.
In the case of the maximum standard, the lower discount rates also produce a positive net benefit for the whole programme under best case assumptions. Under worst case assumptions, however, the net benefit remains negative.

Table 9.1  Sensitivity results

<table>
<thead>
<tr>
<th>Discount rate</th>
<th>Interim 25 μg/l</th>
<th>Final 10 μg/l additional</th>
<th>Final 10 μg/l total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Worst case</td>
<td>Best case</td>
<td>Worst case</td>
</tr>
<tr>
<td>6%</td>
<td>Mean</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6%</td>
<td>457</td>
<td>1039</td>
<td>-3201</td>
</tr>
<tr>
<td>4%</td>
<td>699</td>
<td>1546</td>
<td>-3717</td>
</tr>
<tr>
<td>2%</td>
<td>1112</td>
<td>2376</td>
<td>-4244</td>
</tr>
<tr>
<td>6%</td>
<td>Maximum</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6%</td>
<td>-4702</td>
<td>2258</td>
<td>255</td>
</tr>
<tr>
<td>4%</td>
<td>-4432</td>
<td>3344</td>
<td>427</td>
</tr>
<tr>
<td>2%</td>
<td>-3890</td>
<td>5265</td>
<td>765</td>
</tr>
</tbody>
</table>
10. CONCLUSIONS AND RECOMMENDATIONS

1. From the scientific literature, it is possible to derive models relating relevant low levels of blood lead to IQ and blood pressure. These models were used in the cost benefit analyses. Other endpoints which have been associated with low blood lead levels, such as effects on gestational age and biochemical indices, were not taken into account. Appropriate quantitative models for these endpoints are less well developed, and the clinical significance of, for example, minor changes in biochemical indices, is unclear.

2. Relationships were developed between waterborne lead and blood lead levels, with adjustments to account for the fall in blood lead levels since the original models were developed. However, it is likely that orthophosphate in drinking water will complex dissolved lead ions and reduce uptake; this could have a significant impact on the relationship between water lead and blood lead concentration.

3. Current exposure to water lead was derived from the results of water company compliance sampling; future exposure was estimated for the “do nothing” and interim and final standard cases interpreted as an average and a maximum. Populations affected were derived from official actuarial figures.

4. It was possible to monetarise the benefits that would accrue through increased IQ on future earnings, taking into account the several linkages between lead, IQ, school performance, participation and wages.

5. The economic benefits associated with a reduction in blood pressure were estimated based on the linkages between lead, hypertension and morbidity and mortality, using a quality adjusted life years (QALYs) approach and valuation of quality and length of life changes. There was a large difference between the benefits valued through the use of loss of earnings and QALYs.

6. Non-health benefits and dis-benefits were assessed: it was possible to value effects due to avoided treatment, time losses, salvage of scrap lead and avoided water loss. The monetary values of these effects were small compared to the value of the health related benefits.

The general conclusions, based solely on the monetarised benefits, are that:

7. Interpreted as a mean the interim standard gives net economic benefits regardless of the success or otherwise of water treatment.

8. However, the final standard would only produce additional net benefits if water treatment was successful. If treatment was unsuccessful in achieving compliance then even if benefits are at the high end of their estimated range, there would be a net overall loss.
9. For the standard interpreted as a maximum the uncertainty about the success of water treatment could have a significant effect on costs.

Uncertainty regarding the success or otherwise of water treatment is the main driver of conclusions 7 to 9. This study has revealed gaps in the data in several areas; a better understanding of the following topics would help to improve the estimates made in this report, and would be of value in other aspects of lead in drinking water.

- relationships between lead concentrations in different types of water sample, between water lead concentrations and blood lead levels and the impact of orthophosphate on bioavailability of lead;
- more precise (causal) relationships between blood lead levels, at low concentrations, and health effects - especially IQ and blood pressure;
- factors determining the likely success of orthophosphate dosing to reduce lead concentrations; and
- cheaper ways of meeting the proposed standards, perhaps through a more targeted programme.
REFERENCES


USEPA (1986) *Reduced lead in drinking water: a benefit analysis*.


ACKNOWLEDGEMENT

Professor Michael Jones-Lee (Centre for the Analysis of Safety Policy and Attitudes to Risk, University of Newcastle) and Professor Neil Poulter (Cardiovascular Studies Unit, Imperial College) peer reviewed a draft of this report and provided valuable comments.
APPENDIX A  GLOSSARY

A.1 DEFINITION OF TERMS

Lead risk - zone classification

In their annual returns of information on lead in drinking water supplies, water undertakers are required to provide an assessment of the risk that the lead concentration would cease to comply with the standard on leaving the undertaker’s pipes. The risk codes used are as follows:

<table>
<thead>
<tr>
<th>Risk</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>No risk</td>
</tr>
<tr>
<td>2a</td>
<td>The risk relates to an insignificant part of the zone</td>
</tr>
<tr>
<td>2b</td>
<td>treatment is not reasonably practicable</td>
</tr>
<tr>
<td>2c</td>
<td>treatment will not achieve a significant reduction in lead concentration</td>
</tr>
<tr>
<td>2d</td>
<td>treatment to be installed or optimised</td>
</tr>
<tr>
<td>2e</td>
<td>the significance of the risk and/or the effectiveness of the treatment has yet to be assessed</td>
</tr>
<tr>
<td>2f</td>
<td>treatment installed and/or optimised; effectiveness not yet evaluated</td>
</tr>
<tr>
<td>2g</td>
<td>treatment installed and/or optimised, but risk not eliminated. Further treatment not reasonably practicable</td>
</tr>
<tr>
<td>2h</td>
<td>treatment installed and/or optimised risk eliminated</td>
</tr>
<tr>
<td>3</td>
<td>assessment of risk still being made</td>
</tr>
</tbody>
</table>

Types of sample

**Random daytime sample:** A sampler visits the property at a random time during the working day (the choice of property may also be randomised). A single sample (typically 1 litre) is taken from a drinking water tap without flushing any water from the tap beforehand. The stagnation time of water in the pipes before sampling is unknown and depends on when the water was last used.

**First draw sample:** A sample is taken (normally by the consumer) from the drinking water tap first thing in the morning before water has been used anywhere in the house and without flushing the tap beforehand. The stagnation time of these samples although much longer than daytime samples, is also usually unknown.
**Fully flushed sample:** A sample is taken after prolonged flushing of the tap (at least 5 service pipe volumes) at around 4 litres/min.

**Fixed stagnation time sample:** After prolonged flushing of the tap, water is allowed to stand in the pipework for a defined period (often 30 minutes, although other times have been used) after which a sample is taken without flushing the pipe beforehand.

**Composite proportional sample:** A consumer-operated device is fitted to the drinking water tap which splits off a small constant proportion (5%) of every volume of water drawn for drinking purposes. The samples are pooled for analysis.

### Compliance sampling

Sampling used to establish whether or not the lead concentrations from a property or water supply zone conform to the prescribed limit.

**Statutory sampling:** Compliance sampling, according to the present Regulations, as described below.

**Zone (water supply zone):** Supply systems are divided into Zones supplying a population no greater than 50,000 for the purposes of statutory monitoring. The zones are based on defined water quality characteristics supplied from one or more specified sources.

**Sampling frequency:** The standard sampling frequency for lead is 4 per year which has to be increased to 12 (zone population up to 35,000) or 24 (zone population 35,001-50,000) per year, if the lead concentration in any sample exceeds the standard (50 μg/l). If all sample results for 3 years are <25 μg/l the sampling frequency may be reduced to 1 per year.

**Statutory sample type:** Statutory sampling for lead is based on one litre random daytime samples, collected without prior flushing, from consumers' taps (usually the kitchen tap) in randomly selected properties (with or without lead pipes) at random times during the working day.

### Compliance scenarios

In the present study, three statistical scenarios for compliance were considered:

**Maximum:** interpretation of the standard as an absolute maximum; a zone would fail if any one sample from the zone exceeded the standard.

**Mean:** (or average) interpretation of the standard as applying to the mean results from a zone; a zone would comply provided the mean of the lead concentrations in all samples taken from the zone was within the standard.
Percentile: interpretation of the standard as applying to the 80 percentile (80 %ile) of results from a zone; a zone would comply provided the 80 %ile of the lead concentrations in all samples taken from the zone was within the standard. The 80 %ile is the lead concentration below which 80 % of the results lie.

Pipework definitions and responsibilities

Service pipe: The pipe connecting the mains to a property, consisting of:

- Communication pipe from the main to the curtilage of the property, the responsibility of the water undertaker, and
- Supply pipe from the curtilage of the property to the stopcock inside the property, the responsibility of the property owner.

Plumbing

Internal pipework, the responsibility of the property owner.

Toxicology Terms

Absorption: the process of taking into the body.

Ante-natal: pertaining to the period before birth.

Anti-hypertensive treatment: treatment which reduces high blood pressure.

Base-line blood lead levels: background levels of lead present in the blood in the general population.

Bioavailability: the rate and extent of absorption.

Biochemical effects: effects on the chemistry of living things (largely dealing with metabolism).

Body lead burden: the total amount of lead present in the body at a particular time.

Bone mobilisation: the release from bone.

Cross-sectional studies: the study of a population with a particular characteristic (e.g. exposure to a chemical) at a single point in time.

Dentine: the substance of which teeth are mainly composed.

Diastolic: the relaxation period of the heart beat.
Gestational length: duration of pregnancy.

Haem synthesis: the production of the pigment-carrying portion of haemoglobin.

Meta-analysis: a statistical method which can be applied to provide an overall quantitative assessment of a number of individual studies.

Post-natal blood measures: blood samples taken after delivery.

Prospective studies: studies in which populations are identified and characteristics observed over time.

Systolic: the contracting phase of the heart beat.

Toxicant: a substance which causes adverse health effects.

Economic Terms

Actuarial earnings multiple: net present value of future earnings divided by present earnings.

Cox model: statistical model with an exponential functional form.

Dependent variable: key variable of interest. Analysis centres on explaining its behaviour.

Discount factor: factor to compensate for growth in wealth over time.

Disease risk factor: expected proportion of population suffering from disease in a random sample.

EuroQol: system for assessing Q scores.

EuroQol 1111: perfect health assessment across all five major categories of well-being.

GDP: gross domestic product.

HSB: High School and Beyond data set of US students completing high-school in 1980.

Independent variable: factor, itself determined exogenously, which is correlated with movements in the dependent variable.

Instrumental variable estimation: econometric technique which uses one variable as a proxy for another.

IQ: intelligence quotient-standardised intelligence score.

Morbidity: non-life-threatening symptoms of disease.
Mortality: death caused by disease.

Net present value: monetary values across time discounted to a common date.


Ordinary least squares: regression technique which selects the relationships among a set of data points that minimises the squared errors.

Participation: probability of being employed.

Probit: statistical technique for modelling discrete (non-continuous) variables.

Q score: standardised rate of exchange between years of sickness and death.

Quality-adjusted life expectancy: expected number of standardised healthy years (QALYs) left to live.

Quality-adjusted life year: standardised year of full health.

Schooling (years of): number of years spent at school.

Standard deviation: measure of spread in a distribution of data points.

Total factor productivity: measure of productive efficiency, i.e. quantity of output per unit of input.

TTO: time trade-off.

Value of a statistical life: assessment of individuals' trade-offs between exposure to life-threatening risk and avertive expenditure/compensation.

Willingness to pay: individuals' valuation of a state of health or sickness - the amount they are prepared to pay to avoid that state.

Miscellaneous terms

Stagnation curve: the pattern of build-up of lead concentration in water inside a lead pipe over time.

Dissolved lead: or "soluble" lead - lead in water that is in true solution.

Particulate lead: lead in water in the form of particles of lead compounds or lead adsorbed on other particles.
## A.2 Abbreviations for Sample Types

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Meaning</th>
</tr>
</thead>
<tbody>
<tr>
<td>RDT</td>
<td>Random daytime sample</td>
</tr>
<tr>
<td>30MS</td>
<td>30 minute stagnation sample</td>
</tr>
<tr>
<td>COMP</td>
<td>Composite proportional sample</td>
</tr>
<tr>
<td>Flush</td>
<td>Flushed sample</td>
</tr>
<tr>
<td>SD</td>
<td>Standard deviation</td>
</tr>
</tbody>
</table>
## APPENDIX B  UNIT COSTS

<table>
<thead>
<tr>
<th>Pipe</th>
<th>Mean unit cost</th>
<th>Maximum</th>
<th>Minimum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Communication</td>
<td>£393</td>
<td>£754</td>
<td>£203</td>
</tr>
<tr>
<td>Supply</td>
<td>£483</td>
<td>Not appropriate due to small data set</td>
<td></td>
</tr>
<tr>
<td>Plumbing</td>
<td>£442</td>
<td>Not appropriate due to small data set</td>
<td></td>
</tr>
</tbody>
</table>

### Water Treatment

- **Capital cost**: £45 000 per zone
- **Operating cost**: £2.50 per M litre
  - Assuming 500 litre per property per day
APPENDIX C  IQ

This Appendix covers three areas. First is a synopsis of the main papers referred to in the chapter. Second, the detailed procedure by which the tightening lead standards are translated into earnings benefits is shown. Third, the technical difficulties in using coefficient estimates from different studies are discussed.

C.1  SUMMARIES OF THE RESEARCH PAPERS

Angrist and Krueger (1991)

This paper uses earnings and schooling data for men eligible to be drafted for Vietnam to investigate the returns to schooling. The Vietnam draft lottery number of each individual is used as an instrument for educational attainment to control for ability bias. The draft lottery was conducted in such a way that individuals with low numbers had an incentive to enrol in further education to avoid conscription, as the lower your draft number, the more likely you were to be called up. This effect is borne out by enrolment data which shows there was a peak in 1968-1974 for men. Thus the draft lottery number forms a perfect instrument for extra schooling. It is highly correlated with it, but has no correlation with the dependent variable, earnings.

The instrumental variable (IV) estimate of the return to schooling was a 6.6% increase in weekly earnings. This was 10% higher than the ordinary least squares (OLS) estimate. Since any ability bias was expected to be downwards, the OLS estimate should have been higher than the IV one, indicating that any problems in identifying the relationship between schooling and earnings is more likely to be due to measurement error than ability bias.

Ashenfelter and Krueger (1994)

This paper uses a new survey of identical twins. Returns to schooling are estimated by contrasting the wage rates of identical twins with different educational levels. The use of twins controls for both genetic ability and the benefits which accrue from family background. It also uses each twin’s reporting of the other’s schooling level to provide a measurement error control.

A very high return to schooling is discovered: each additional year of schooling generates 12-16% higher annual earnings. These arise from regressions of intrapair earnings differentials on intrapair educational differences, and the same where the educational level used is that reported by the sibling. Similarly to the previous paper, unobserved ability does not seem to bias an OLS estimate upwards. In fact the OLS estimate was substantially lower, around 8%. Investigation suggested that measurement error was causing the lower estimate.
Blanchflower and Oswald (1994)

This analysis seeks to estimate a wage curve, linking the level of pay to the rate of local unemployment. They use data from the General Household Survey between 1973 and 1990 on 175,000 workers. Weekly earnings data is regressed (pooled generalised least squares) on a wide range of variables including personal characteristics, one of which is educational achievement, as measured by years of schooling. There is no control for ability, that is, no IQ measure. Men and women are differentiated and for each additional year of schooling, men’s weekly earnings increase by 3.96%, women’s by 2.92%.

Brown and Reynolds (1975)

This analysis looks at the relationships between IQ, occupation and earnings. No further controls are used, such as family background or educational achievements. The IQ measure is the Armed Forces Qualifying Test. The sample is men only and is based on earnings, occupation and years of schooling in 1962-1964.

The results suggest that an extra IQ point is correlated with 0.2-0.3 extra years of schooling; however, this does not control for other effects such as family background, which is likely to bias this effect upwards. Hence this research suggest an upper bound for this linkage.

More interesting is some of the other findings in this study, about the stratification of effects. The authors find that the effect of IQ on earnings increases with the level of education and with the level of work experience. This suggests that ability is rewarded more when associated with training. The implications of this are that an average value for the effect of IQ on earnings or probability of employment may mask the true effects. This is particularly relevant for the lead study if the vulnerable group is likely to be of low or high ability, since the average would then over- or under-estimate the benefit of reducing lead.

Jenkins (1995)

This research investigates the role of education in production in the UK economy, between 1973 and 1992. An aggregate total factor productivity measure is regressed on the proportions of the workforce that hold tertiary qualifications, intermediate qualifications and no qualifications. Control variables for the business cycle and capital measurement difficulties are included.

The results suggest that workers with tertiary qualifications are 1.5 to 2 times more productive than those with no qualifications. Since the dependent variable is output (GDP), rather than earnings, this analysis shows the social, rather than private, gain to extra education. This effect is translated into an implied annual increase in output for each additional year of schooling, comparable to the results reported in standard earnings
equations. A person with an extra year of tertiary training produces between 5.2% and 13.8% extra output, depending on the weighting given to capital in the productivity measure.

Lynn, Hampson and Magee (1984)

This paper looks at the problem of youth unemployment in Northern Ireland. The dependent variable was whether an individual had a job one year after completing school. The explanatory factors used were: family background, IQ, personality characteristics, school type and educational attainment. The IQ measure was the Abstract Reasoning scale of the Differential Aptitude Test. The personality traits were determined from a battery of psychological tests. All these tests were conducted by the authors.

The results show that 26% of the variance in unemployment experience for men, and 14% for women was explained by the chosen factors. Educational attainment is the strongest single predictor. The IQ variable influences employment outcome through the type of school attended, grades achieved at school as well as directly. The total effect of these was 0.23 for men and 0.14 for women, in standardised coefficients. The direct effect alone was 0.02 for men and 0.01 for women.

Marin and Psacharopoulos (1982)

This paper sets out to identify whether risky jobs are more highly paid than others. A standard earnings function is estimated by OLS. The dependent variable is the logarithm of annual earnings in 1975. The explanatory factors are: years of schooling, years of experience in the work force, weeks worked in the survey year, measures of job risk (matched to occupation), a union dummy and occupational effects. There is no ability control.

Looking at the results for the whole sample, rather than breaking it down by occupational groups, the earnings effect of an additional year of schooling is an increase of between 5.72% and 5.93%. When the sample is changed to include only the top third riskiest jobs, the earnings effect falls to 4.1% for the preferred risk measure. However, this is final group is not a representative sample of the economy as a whole.

Murnane, Willett and Levy (1995)

This paper seeks to explore the increasing private returns to skills seen in the US job market. The authors wish to investigate which skills are being more highly rewarded. The data is drawn from two databases with information on the labour market performance of students who graduated from high-school in the last two decades: The National Longitudinal Study of the High School Class of 1972 (NLS72) and High School and Beyond (HSB) based on interviews on a group of people who were seniors in 1980. The sample used those men and women who had completed their formal education and had
been in paid work for six years. The key question asked is: how does the mathematics skills of graduating high school seniors affect their wage at age 24? Has this changed between the two groups?

The dependent variable is the natural logarithm of hourly wages at age 24. The IQ measure was the Item-Response Theory-scaled mathematics score, which measures mastery of elementary mathematical concepts, but not knowledge of more advanced mathematics. Other controls included were: years of completed schooling, race, ethnicity, region, full-time and part-time experience and family background.

The results indicated that the return to IQ had increased between the two groups of individuals. For men, the NLS72 coefficient was 0.4%, the HSB value rose to 1.1%. For women, the NLS72 effect was estimated as 0.9%, the HSB at 1.7%. The HSB values are used in this analysis, as they are the most recent. Since the IQ measure is different to that used for the lead study, the coefficient values must be translated to be comparable.

While this paper also reports the impact of years of schooling on earnings, these are not used in this analysis. This is because the sample can only look at the benefits for those who have completed high-school, and only for earnings up to age 24. This will bias downwards the overall effect of schooling on earnings.

Salkever (1995)

This study builds on the evidence from Schwartz (1994) by updating the estimates of the earnings benefits arising from increased IQ. Salkever develops different estimates of the effects of IQ changes on earnings, but scales them up with the same estimate of baseline earnings. Recent data from the National Longitudinal Survey of Youth in 1990 is analysed. The proxy for IQ is the Armed Forces Qualifying Test, a score based on the responses to questions that test arithmetic, verbal and mathematical skills.

Three different relationships were estimated separately for men and women:

- a least squares regression of highest grade on the IQ proxy;
- a multiple probit regression of a 0-1 indicator of positive earnings in 1990 (that is, employed) on highest grade and the IQ proxy;
- a least squares regression, for those with positive earnings, of the logarithm of earnings on highest grade and the IQ proxy.

The methodology is commendable in that a wide range of effects are controlled for in the regression analysis: family background - parents’ education and income - as well as age, ethnicity and geographical variables. This gives greater confidence that, in each case, the coefficient estimated isolates the effect of IQ alone.
Schwartz (1994)

This paper addresses the same issue as this analysis: valuing all the benefits from reducing the concentration of lead in drinking water, except that it is for the US. Thus part of this work covers the links between IQ and lifetime earnings. His methodology is similar to that undertaken here: to survey the literature and use relevant estimates, rather than undertake his own analysis.

Much of his analysis of the links between lead and school performance was based on a study by Needleman et al. in which a follow-up study of an elementary school cohort investigated the effect of lead on likelihood of dropping out of school, grades achieved and IQ. It is only the final of these effects that is considered important in the UK context.

The IQ-earnings link is based on a number of older empirical surveys: Schwartz determines 0.5% as the median effect of a 1 point increase in IQ on earnings. He estimates the schooling-earnings link to be 6%, based on empirical evidence from the 1960s and 1970s. A 1 point increase in IQ is estimated to increase the probability of being in work by 0.47%. This includes the direct and indirect (via education) effects.

Overall, Schwartz suggests that a reduction in blood lead concentrations of 1 µg/dl would produce a net benefit of $1300 per child for the cohort turning 6 years of age each year, leading to a total benefit of $5.06 billion per year. A discount rate of 5% and lifetime earnings of $301 000 are assumed.

Waller (1971)

This study investigates the links between social mobility and intelligence, educational achievement and family background. It is based on a sample of Minnesotan fathers and sons in 1965. The IQ test was administered when the individuals were in their teens; no details are given on the particular tests used.

Educational attainment (years of schooling) was regressed on IQ score, family size and father’s education and occupation. This indicated a significant positive effect from IQ to educational achievement, with a value of 0.343, standardised by standard deviation.
C.2 PROCEDURE FOR VALUING THE IQ BENEFITS OF TIGHTER WATER LEAD STANDARDS

Table C2.1 Summary of the main steps involved in valuing the economic benefit of raising IQs as a result of tightening water lead standards

<table>
<thead>
<tr>
<th>Step</th>
<th>Procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Calculate the change in water lead resulting from the introduction of the new standard for groups of individuals facing initial water leads in seven categories ranging from over 89 µg/l to under 4.45 µg/l. (Those in the highest category, 89 µg/l, face the largest improvements in water lead exposure as a result of the new standards, whereas those in the lowest category will receive no benefit from the new standards.)</td>
</tr>
<tr>
<td>2</td>
<td>Calculate the average change in water lead for the preceding ten years for each year from 1998-2034 for each group. (This assumes that the life-time change in IQ experienced by ten-year olds in each group, every year, will depend on the water lead exposure over their childhood from the age of zero to ten.)</td>
</tr>
<tr>
<td>3</td>
<td>Calculate the change in IQ resulting from the change in water lead for each group, by applying the lead-IQ coefficients obtained from the review of the epidemiological literature above.</td>
</tr>
<tr>
<td>4</td>
<td>Calculate the percentage change in future earnings, by multiplying the change in IQ by the IQ-earnings coefficients estimated from the review of the economic literature above.</td>
</tr>
<tr>
<td>5</td>
<td>Calculate the change in the present value of future earnings by multiplying the percentage change in future earnings (from Step 4) by the present value of future earnings (from Step 5). This gives the average change in earnings per person affected by the tightening standard.</td>
</tr>
<tr>
<td>7</td>
<td>Calculate the total value of IQ-related benefits by aggregating the average change in earnings per person by the total number of ten-year olds in each group for each year.</td>
</tr>
</tbody>
</table>
C.3 TECHNICAL DIFFICULTIES IN COMPARING ESTIMATES FROM DIFFERENT PAPERS

The range of analyses performed was reflected in the synopses in Section C.1. It is unsurprising that it was sometimes difficult to translate the reported estimates into a usable form. There were three main difficulties: scaling of IQ scores, use of standardised coefficients and reporting of unadjusted probit analyses.

C3.1 Scaling

Each piece of research uses its own proxy for IQ. In order to ensure comparability, these need to be scaled to ensure that a one-point change in IQ has the same meaning within each of the different studies used. This can be done by using the standard deviation for the IQ of each of the corresponding samples, which is typically reported in the published versions of the study. An important exception is the study by Pocock et al. (1994) from which the lead-IQ link has been taken in the epidemiological literature review. In order to make use of this value, it was necessary to assume that the Pocock et al. sample had a mean IQ of 100 and standard deviation of 15, which is the population average. To the extent that these assumptions do not correspond to the survey sample used in this study, some small inaccuracies will be introduced into the calculation.

C3.2 Standardised coefficients

As already noted above during the review of the economic literature, a further difficulty arises in that many of the studies report their coefficients in standardised form. A coefficient of 0.2 in 'standardised' form, implies that a 1 standard deviation change in the independent variable, will result in a 0.2 standard deviation change in the dependent variable. Thus in order to convert this into an absolute change, it is necessary to know the standard deviations for the corresponding dependent and independent variables.

Since the requisite standard deviations for variables, such as wages and participation probabilities, were not available, it was not possible to make use of the estimates reported in those studies. However, the fact that such studies confirm the existence of a significant relationship adds weight to the evidence of a benefit from reducing blood lead concentrations.

C3.3 Probit analyses

Much of the evidence on likelihood of being in work uses a regression technique known as probit. Because the dependent variable is dichotomous (1 for working, 0 for not), standard linear techniques such as OLS are not appropriate. However, this also affects the interpretation of the coefficients.

A linear (log-linear) estimation technique implies that a unit (1%) change in the independent variable will lead to a \( \beta \) (\( \beta\% \)) change in the dependent variable, where \( \beta \) is
the estimated coefficient. In a probit analysis, the size of the effect of an explanatory factor can not be interpreted directly from the reported coefficient-only the direction of the effect and its significance. In order to determine the effect on the probability of being in work of an increase of one year of schooling, the full data set is required. Thus, only if the authors have the same interests as the current study and have reported that particular effect will such studies yield a useful estimate.
APPENDIX D   CIRCULATORY DISEASE

D.1 INTRODUCTION

This technical appendix provides detail and background to some of the data and methods used in the main text.

D.2 IMPACT OF BLOOD PRESSURE ON RISK OF DISEASE

Schwartz (1991), in an analysis of the Framingham study, reports that reductions in male blood pressure of about 2 mm Hg and 1 mm Hg for females lead to 24,000 fewer myocardial infarctions a year and 100,000 fewer cases of cardiovascular disease in the USA.

Staessen (1994) reports, in his meta-analysis, that a 37% drop in blood lead accompanied a 5% fall in fatal and non-fatal myocardial infarction, and a 7% fall in the rate of fatal and non-fatal strokes, resulting in a 5-6% fall in mortality. Here the equivalent estimates are approximately a 3% fall in the risk of CHD and a 5% fall in the risk of stroke.

Peto’s work relating DBP to risk of CHD and stroke used a non-parametric method, Cox regression, producing the coefficients shown in Table D2.1 with negligible confidence limits.

Table D2.1  Relationship between incidence of stroke, CHD and mm Hg, studies for 6-25 year follow-up period (change in risk, %)

<table>
<thead>
<tr>
<th>Change in mm Hg</th>
<th>Stroke</th>
<th>CHD</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>56</td>
<td>37</td>
</tr>
<tr>
<td>7.5</td>
<td>46</td>
<td>29</td>
</tr>
<tr>
<td>5</td>
<td>34</td>
<td>21</td>
</tr>
<tr>
<td>1</td>
<td>6.8</td>
<td>4.2</td>
</tr>
</tbody>
</table>

Straight-line extrapolation
The Cox proportional hazards model cannot be used to extrapolate close to zero: in this model, such an extrapolation would imply that zero change in DBP leads to a 21% change in risk factor (NHS 1995). To circumvent this problem, it was assumed that a straight-line relationship links risk and DBP for small changes in DBP. Table D2.1 shows that this implies changes in CHD and stroke risks for a 1 mm Hg change in DBP of 4.2% and 6.8% respectively.

The suitability of the extrapolation was verified via a verbal communication with the author of the study (Peto 1997), who provided the estimates in Table D2.2. The figures illustrate that the disease risks are declining with age, and that the estimates obtained by linear extrapolation of the published results lie towards the middle of these age-specific ranges.

Table D2.2  Percentage change in risk on a 1 mm Hg change in DBP across the population, Peto (1997)

<table>
<thead>
<tr>
<th>Age group</th>
<th>Stroke</th>
<th>CHD</th>
</tr>
</thead>
<tbody>
<tr>
<td>40-59</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>60-69</td>
<td>7</td>
<td>3.5</td>
</tr>
<tr>
<td>70-79</td>
<td>5</td>
<td>2.5</td>
</tr>
<tr>
<td>80+</td>
<td>not estimated</td>
<td></td>
</tr>
</tbody>
</table>

D.3  RESOURCE BENEFITS FROM LOWER COSTS OF TREATING CIRCULATORY DISEASE

Estimates of resource costs were taken from NHS Executive (1996) and the Office of Health Economics (1997), see Table D3.1. Where discrepancies exist, the average of the two figures was used. These figures were subsequently averaged across the total populations affected by each of the circulatory diseases to produce unit values.

The unit values were then aggregated across the populations benefiting from reduced incidence of circulatory disease as a result of the tightening in water lead standards. This gave the resource benefits shown in Tables D3.2 and D3.3. The benefits are shown for the Interim standard and for the final standard in aggregate. The incremental benefits of the final standard over the interim can be found by subtraction.
### Table D3.1  Total resource costs (£m) for disease by cause, 1994 estimates, England and Wales

<table>
<thead>
<tr>
<th>Disease</th>
<th>OHE</th>
<th>Burdens of Disease</th>
<th>Working estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>294</td>
<td>466</td>
<td>380</td>
</tr>
<tr>
<td>CHD</td>
<td>1245</td>
<td>1517</td>
<td>1381</td>
</tr>
<tr>
<td>CVA</td>
<td>1209</td>
<td>1357</td>
<td>1283</td>
</tr>
<tr>
<td>Total circulatory diseases</td>
<td>3980</td>
<td>4189</td>
<td>4085</td>
</tr>
</tbody>
</table>

### Table D3.2  Resource benefits (£m), average standards

<table>
<thead>
<tr>
<th></th>
<th>6%</th>
<th>4%</th>
<th>2%</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>25 µg/l</td>
<td>10 µg/l</td>
<td>25 µg/l</td>
</tr>
<tr>
<td>CHD</td>
<td>12</td>
<td>21</td>
<td>16</td>
</tr>
<tr>
<td>CVA</td>
<td>18</td>
<td>31</td>
<td>24</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1.6</td>
<td>2.7</td>
<td>2.1</td>
</tr>
</tbody>
</table>

### Table D3.3  Resource benefits (£m), maximum standards

<table>
<thead>
<tr>
<th></th>
<th>6%</th>
<th>4%</th>
<th>2%</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>25 µg/l</td>
<td>10 µg/l</td>
<td>25 µg/l</td>
</tr>
<tr>
<td>CHD</td>
<td>29</td>
<td>36</td>
<td>38</td>
</tr>
<tr>
<td>CVA</td>
<td>43</td>
<td>54</td>
<td>57</td>
</tr>
<tr>
<td>Hypertension</td>
<td>3.8</td>
<td>4.7</td>
<td>5.0</td>
</tr>
</tbody>
</table>
D.4 VALUATION OF MORBIDITY AND MORTALITY

A4.1 Method 1

The detailed working for Method 1 is explained in Figure D4.1.

All the following steps were repeated for males of ages 40 to 70 years.


Step 2: calculate the risk of mortality from circulatory diseases from the population death by cause statistics, Office for National Statistics (1997).

Step 3: subtract risk of mortality from circulatory diseases from the general mortality risk to obtain the risk of mortality for persons without circulatory disease, i.e. the control population.

Step 4: calculate the remaining life expectancy of persons in the control population.

Step 5: calculate the incidence of circulatory disease from the total disease incidence, British Heart Foundation (1996), Wolfe (1996), less mortality from each disease.

Step 6: calculate the TTO Q score for the control population from the TTO Q scores for the whole population, the TTO Q score for EuroQol non-11111 respondents, and the proportion of CHD and CVA sufferers from Step 5.

\[
\frac{Q(\text{control population})}{(\text{Proportion in control population})} = \frac{Q(\text{all respondents})}{(\text{Proportion of circulatory disease sufferers})} - \frac{Q(\text{non-11111})}{(\text{Proportion of circulatory disease sufferers})}
\]

or, rearranging:

\[
Q(\text{control population}) = Q(\text{all respondents}) - Q(\text{non-11111}) \cdot \frac{(\text{Proportion of circulatory disease sufferers})}{(\text{Proportion in control population})}
\]

Step 7: to find the QALYs lost from a year of circulatory disease morbidity, subtract the Q score for circulatory disease from the Q score for the control population.

Step 8: to find the total QALYs lost from morbidity, multiply the QALYs lost from a single year of disease by the incidence of CHD, hypertension and stroke morbidity - this gives the final result for morbidity.

Step 9: apply the TTO Q scores for the control population to the expected remaining years of life to give QALE for the control population.

Step 10: multiply the QALE by the number of deaths by cause to find the total QALYs lost from mortality - this gives the final result for mortality.

Step 11: multiply the morbidity and mortality results by the risk factors related to lead exposure and forecast changes in lead exposure for the years 1998 to 2034, discounting at 6, 4, and 2 percent.

Figure D4.1 Calculation of QALYs lost through circulatory disease
Method 2 gave the results listed in Tables D4.1 and D4.2.

**Table D4.1** QALY valuation of the benefits of a change in water lead standards £m: average standards

<table>
<thead>
<tr>
<th></th>
<th>CHD</th>
<th>CVA</th>
<th>Hypertension</th>
<th>CHD</th>
<th>CVA</th>
<th>Hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>6%</td>
<td>250</td>
<td>77</td>
<td>2.0</td>
<td>600</td>
<td>149</td>
<td>3.7</td>
</tr>
<tr>
<td>4%</td>
<td>430</td>
<td>130</td>
<td>2.7</td>
<td>600</td>
<td>180</td>
<td>4.8</td>
</tr>
<tr>
<td>2%</td>
<td>340</td>
<td>100</td>
<td>3.5</td>
<td>460</td>
<td>149</td>
<td>3.7</td>
</tr>
</tbody>
</table>

**Table D4.2** QALY valuation of the benefits of a change in water lead standards, maximum standards, £m

<table>
<thead>
<tr>
<th></th>
<th>CHD</th>
<th>CVA</th>
<th>Hypertension</th>
<th>CHD</th>
<th>CVA</th>
<th>Hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>6%</td>
<td>600</td>
<td>180</td>
<td>4.8</td>
<td>110</td>
<td>310</td>
<td>8.1</td>
</tr>
<tr>
<td>4%</td>
<td>750</td>
<td>230</td>
<td>6.3</td>
<td>110</td>
<td>330</td>
<td>8.7</td>
</tr>
<tr>
<td>2%</td>
<td>790</td>
<td>240</td>
<td>6.0</td>
<td>1000</td>
<td>330</td>
<td>8.7</td>
</tr>
</tbody>
</table>

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A4.2 Method 2: Estimation of wages foregone due to circulatory disease

The calculation estimated the number of days taken as sick leave by cause in any given year from the Social Security Statistics (1995), taking the total number of days from circulatory diseases, 102 m in 1995, and assigning the days between CHD and CVA according to the incidence of those diseases.

Lost earnings were valued at the weekly wage rate reported in the New Earnings Survey, broken down by age, giving £455.8/week for 40-49 year old males, £420.50/week for 50-59 year old males, and £341.30/week for 60-64 year old males in April 1996 terms.

Cases of early death were valued using actuarial earnings multiple tables to estimate the value of lost future earnings (HMSO 1994).

The results are shown in Tables D4.3 and D4.4, and can be compared with those obtained by the QALY method in Table D4.1 and D4.2.

Table D4.3 Earnings benefits from tighter lead standards, £m: average standard

<table>
<thead>
<tr>
<th>Mortality</th>
<th>CHD</th>
<th>6%</th>
<th>4%</th>
<th>2%</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>25 µg/l</td>
<td>10 µg/l</td>
<td>25 µg/l</td>
<td>10 µg/l</td>
</tr>
<tr>
<td>CHD</td>
<td>18</td>
<td>30</td>
<td>23</td>
<td>41</td>
</tr>
<tr>
<td>CVA</td>
<td>4.8</td>
<td>8.2</td>
<td>6.3</td>
<td>11</td>
</tr>
<tr>
<td>Hypertension</td>
<td>0.1</td>
<td>0.2</td>
<td>0.2</td>
<td>0.3</td>
</tr>
<tr>
<td>Morbidity</td>
<td>all circulatory disease</td>
<td>55</td>
<td>94</td>
<td>73</td>
</tr>
</tbody>
</table>
Table D4.4  Earnings benefits from tighter lead standards, maximum standards, £m

<table>
<thead>
<tr>
<th></th>
<th>6%</th>
<th>4%</th>
<th>2%</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>25 µg/l</td>
<td>10 µg/l</td>
<td>25 µg/l</td>
</tr>
<tr>
<td>Mortality</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHD</td>
<td>42</td>
<td>52</td>
<td>55</td>
</tr>
<tr>
<td>CVA</td>
<td>11</td>
<td>14</td>
<td>15</td>
</tr>
<tr>
<td>Hypertension</td>
<td>0.3</td>
<td>0.4</td>
<td>0.4</td>
</tr>
<tr>
<td>Morbidity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All circulatory disease</td>
<td>130</td>
<td>160</td>
<td>170</td>
</tr>
</tbody>
</table>

The lost earnings calculation only includes males up to the age of 65, and excludes elements of the true economic cost of disease, for example, profit which is part of economic productivity, loss in welfare through distress and otherwise reduced quality of life.