3 Lead in petrol 'makes the mind give way'

Herbert Needleman and David Gee ([1])

This chapter addresses the widespread use of lead in petrol. It focuses on the period 1925–2005, when leaded petrol was first widely marketed in the US and then spread to the rest of the world before being gradually phased out from the 1970s. In Europe, the Aarhus Protocol (UNECE, 1998) initiated the phase-out of leaded petrol in the period 1998–2005.

The neurotoxic effects of lead were recognised as far back as Roman times. And in 1925, at the 'one day trial' of leaded petrol in the US, many experts warned of the likely health impacts of adding lead to petrol. Yet, despite the availability of an equally effective alcohol additive which was assessed by experts to be cleaner, the leaded route to fuel efficiency was chosen in the US and then exported to the rest of the world.

For several decades after the introduction of leaded petrol, virtually no independent research was carried out and the main source of information was industry and industry-sponsored researchers. Not until the 1960s and 1970s did independent scientists from outside this group show, for example, that body burdens of lead arising from human activities were not 'normal', as industry claimed, but were hundreds of times higher than before the industrial revolution and were therefore likely to be harmful.

At its peak in the mid-1970s, leaded petrol released about 200 000 tonnes of lead into the atmosphere annually in both the US and Europe. Following the subsequent phase-out, blood lead levels in children (the most sensitive group exposed) quickly fell, in line with the decrease in air concentrations. The lessons nevertheless remain relevant globally today. Although nearly all countries worldwide had phased out leaded petrol by 2012, lead concentrations in soils and sediments remain high. Meanwhile, electronic wastes containing lead and other contaminants also cause elevated blood lead levels.

Supplementary panel texts focus on the events leading up to the US choice of leaded petrol as the primary fuel source in 1925 and more recent accounts of EU policymaking on lead in petrol and the road to phase-outs in Germany and the United Kingdom.

([1]) Authors would like to thank Gerald Markowitz and David Rosner for their detailed history of the leaded petrol story in *Deceit and denial: the deadly politics of industrial pollution*.

Dr Yandell Henderson, Professor of Physiology at Yale, told the conference that lead was as serious a public health menace as infectious diseases. He foresaw that:

> 'conditions will grow worse so gradually, and the development of lead poisoning will come on so insidiously ... that leaded petrol will be in nearly universal use ... before the public and the government awakens to the situation' (USPHS, 1925).

3.1 Introduction

There were 50–70 years between Henderson's prescient early warning about the long-term, low-level poisoning from leaded petrol, when it was introduced in 1925, and its phase out in the US and then Europe in the 1970s and mid-1980s. This long history is rich in lessons about the science, economics, and politics of identifying and controlling the hazards of toxic substances.

The focus of this chapter is lead in petrol and its damage to children (Box 3.1). However, the even longer histories of lead in pots and paints weave in and out of the leaded petrol saga, both complicating the search for the causes of lead poisoning in children, and creating a ubiquitous stock of lead in people and their environments that persists today in soils, sediments, plants, house dust and old paint. Such exposures affect adults too and recent research emphasises the increasing evidence linking lead with hypertension, heart disease and kidney disease (Navas-Acien et al., 2007; EFSA, 2010).

Most of Europe and North America now has lead free petrol but the lessons from this chapter are relevant for controlling most toxic chemicals. They may also be useful for the millions of people, including children, who are still exposed to leaded petrol or

Box 3.1 Children and lead: health impacts

At high levels of chronic exposure, lead attacks the brain and central nervous system, causing coma, convulsions and even death. Children who survive acute lead poisoning are typically left with grossly obvious mental retardation and behavioural disruption. At lower levels of exposure that cause no obvious symptoms and that were previously considered safe, lead is now known to produce a spectrum of harm involving diminished cognition, shortened attention span, disruptive behaviour, dyslexia, attention deficit disorder, hypertension, renal impairment, immunotoxicity and toxicity to the reproductive organs. For the most part, these effects are permanent and largely untreatable.

The major sources of children's exposure to lead are:

- lead from active industries, such as mining (especially in soils);
- lead-based paints and pigments;
- lead solder in food cans;
- ceramic glazes;
- drinking-water systems with lead solder and lead pipes;
- lead in products, such as herbal and traditional medicines, folk remedies, cosmetics and toys;
- lead released by incineration of lead-containing waste;
- lead in electronic waste (e-waste);
- lead in the food chain, via contaminated soil;
- lead contamination as a legacy of historical contamination from former mining and industrial sites.

Acute lead poisoning still occurs today and is most common among children in low-income countries and marginalised populations or in children living on lead-polluted sites of old lead factories or mines.

Recent research indicates that lead is associated with neurobehavioural damage at concentrations in the blood of 5 µg/dl and even lower. There appears to be no threshold level below which lead causes no injury to the developing human brain (see Box 3.11).

The biology of childhood lead poisoning is similar everywhere and the results of lead studies in one country are largely relevant to children in other countries.

Source: WHO, 2010.

Box 3.2 Ancient lead poisoning

Analysis of the Greenland ice core covering the period from 3 000 to 500 years ago — the Greek, Roman, Medieval and Renaissance eras — shows that from about 500 B.C. to 300 A.D. lead was present at concentrations four times greater than natural values. Greek and Roman lead mining and smelting clearly polluted the northern hemisphere long before the industrial revolution, which initiated the modern era of lead poisoning from about from 1750 onwards. Cumulative lead fallout to the Greenland ice sheet during those eight centuries was as high as 15 % of that caused by use of lead alkyl additives in petrol since the 1930s.

Source: Hong, Candelone et al., 1994.

other sources of lead, such as from old lead works, paint and toys, in many countries of the world.

3.2 Lead toxicity: some early warnings

The neurotoxic properties of lead were first noted during the first century AD by Dioscerides, a physician in Nero's army. In his book *Materia Medica* he observed that 'Lead makes the mind give way'. Exposure came from the leaded glaze on pots and from using lead in winemaking to counteracted the harsh acidity of the grapes (lead plates were dipped into the wine during fermentation and the lead acetate, which is also called 'sugar of lead', sweetened the taste). Centuries later, toddlers who chewed the leaded paint on cradles, beds and verandas found that it tasted like lemon drops.

Lead continued to be used in wine-making and epidemics of lead colic were common in Europe and the Americas. One of the earliest public health laws in the US was passed in 1723 to protect rum drinkers from what was called 'the dry gripes'. The law banned the use of leaden 'worms' (condensing coils) in the distilling process. A penalty of 100 pounds was imposed on law breakers, half being distributed to the poor and half to the person who informed the authorities about the breach of law. This was an early attempt to reward and protect 'whistleblowers' — an issue taken up in Chapter 24.

In early-18th century England, a severe outbreak of colic was reported each autumn in Devon. The disease was strictly limited to particular areas while neighbouring shires escaped. The physician Sir George Baker identified the source of the epidemic as the leaden keys in the millstones used in pressing cider apples. His paper to the Royal College of Physicians showed that Devonshire cider contained lead (Baker, 1768). Rather than receiving praise for his incisive work, Baker was condemned by the clergy, by mill owners and by fellow physicians: cider was Devon's main export.

Baker suffered the fate of many 'early warning' scientists whose inconvenient truths are not welcomed by supporters of the status quo ([2]).

During his European travels, Benjamin Franklin had also noted how long it took for 'useful truths' about health hazards to be acted on (Box 3.3).

The UK pottery manufacturer, Josiah Wedgewood, also experienced the long delay between 'useful truths' and regulatory actions. He was sufficiently moved by accidents and lead poisoning in his factories, and concerned about the unfair competition that arose from other, less scrupulous, employers, that he asked the British government to extend the recent Factories Act of 1833 from the textiles industries to the potteries industries so that he could share a 'level playing field' with his competitors. Opposition from other pottery manufacturers meant, however, that Wedgewood had to wait some 30 years before legal controls on lead were established in the Potteries Regulations of 1867.

These early warnings about occupational and consumer lead toxicity largely went unheeded. The use of lead for pots, paints, pipes and toys greatly

([2]) Such 'shooting the messenger' is well illustrated by Ibsen's play *An enemy of the people*. The play chronicles the gradual downfall of the town physician who discovers pollution in the river caused by the local leather factory: a discovery that is initially welcomed by the mayor, the media and the public, but then rejected, as the economic implications for local industry begin to emerge. Many public health advocates in similar circumstances to Baker have taken comfort from Ibsen's play, such as Dr Hosokawa of the Chisso Company Japan, which polluted Minimata Bay with mercury. He, like Ibsen's Doctor Stockmann, was told to suppress his early discovery that sewage from Chisso caused the mercury poisoning (see Chapter 5 on the Minamata disaster).

Box 3.3 The 'useful truths' about lead poisoning in French painters, potters and plumbers

In 1818, Benjamin Franklin, while ambassador to France, described the 'dangles' of wrist drop, and the 'dry gripes' of stomach aches in painters, potters and plumbers who were widely known to suffer from lead poisoning. But he also observed two other trades that suffered similarly but seemed not to be obviously exposed to lead: stonecutters and soldiers. Pursuing this puzzle, he found that the stonecutters used lead to set metal rails in stone, and that soldiers found part time employment as painters' assistants. This provided an early example of the value of detailed job and life exposure histories in identifying occupational and environmental hazards.

Franklin concluded: 'this mischievous effect from lead is at least 60 years old; and you will observe with concern how long a useful truth may be known and exist, before it is generally received and practiced on' (Franklin, 1818).

expanded in the 19th and 20th centuries (Hunter, 1975), accompanied by widespread lead poisoning that could sometimes take years to appear:

> 'Lead poisoning develops insidiously' often years after the exposure, deranging the functions and structure of the cells, 'so that life is gradually brought to a close by the intervention of disease of organs, such as the kidney or nervous system' (Oliver, 1911).

This 'insidious' nature of lead poisoning would later be noted by some of the medical experts who assessed the likely risks from lead in petrol when it was introduced in the 1920s. Children were particularly at risk (Box 3.4).

Box 3.4 The early poisoning of children by leaded paint, and current poisoning from paint and battery plants in Asia

Children face increased risk of lead poisoning compared to adults because they can be exposed during pregnancy; they take in more food, drink and air relative to their weight; they have more hands-to-mouth activity; and they are more likely to have nutritional deficiencies that can increase lead absorption (WHO, 2010).

In 1892 an Australian doctor observed the link between the lead-based paints used on porches, verandas, and window frames, and lead poisoning in 10 children who chewed the flakes of paint and swallowed the dust and chips on the floor where they crawled and played (Gibson, 1904).

In 1914 in the US, the first childhood lead poisoning case was reported with a death from lead poisoning in a child who had chewed lead paint from his crib railings (Thomas and Blackfan, 1914).

By 1925 there was much scientific evidence in the US, Europe and elsewhere showing that infants and children were poisoned by lead in the paint that they found in their daily environments. But their eventual protection from this source of lead came via regulations to protect painters, which led to the widespread banning of lead in paint in Europe and Australia between 1909 and the 1930s.

The US, however, only banned leaded paint for interior surfaces in 1971. As a result, government authorities are still dealing with the legacy of lead poisoning in the poorer areas of the US where the housing remediation costs, and associated legal cases over who is to pay remediation, still consume much time and money.

Meanwhile, there is still widespread exposure of children to leaded paint in many regions, particularly, developing countries. A survey of 10 countries in Asia, Africa, South America and eastern Europe found much leaded paint on sale, some with lead levels ranging from 4 000 to nearly 40 000 parts per million (ppm), compared to the US recommended limit of 90 ppm (UNEP/WHO, 2010). Moreover, China has reported many serious childhood lead poisoning incidents in recent years.

3.3 Lead in petrol 1922–1925: the early warnings of hazards to the public

3.3.1 Origins of lead in petrol

Until 1925, the principal source of toxic lead for the public was household paint. This changed dramatically when General Motors, in second place behind Ford Motors in car sales, sought to compete with its new higher performance Cadillac. The new GM engine had a severe engine 'knock' that arose from the premature ignition of the petrol, meaning that GM needed to find an anti-knock additive for the petrol. Their chief chemist, Thomas Midgely, who later invented the CFC chemicals that created the hole in the ozone layer, (see EEA, 2001, Ch. 7 on halocarbons), found an old German patent for tetraethyl lead (TEL) and discovered that it could be used in petrol to control the engine knock.

Alternatives to petroleum-based fuel, such as ethanol, were available. They were likely to be much less profitable, however, especially given the family and financial links between GM, the chemical company, DuPont, and Standard Oil. Pierre Dupont was chair of the GM Board, his brother Irene ran DuPont, both had close links with Standard Oil, and in 1924 the three companies created the Ethyl Corporation of America to produce TEL.

It was made clear at the outset that the word 'lead' was not to be used in the company name or sales literature: the little known term, 'ethyl' was used instead so as not to alarm the public.

This was an early example of the censoring of sensitive words from the discussion of hazards, a practice repeated in the other *Late lessons from early warnings* case studies, for example asbestos (EEA, 2001). For example, the word 'cancer' in the early studies of asbestos workers was initially replaced by the less well known terms, 'tumour' or 'malignancy', at the request of asbestos manufacturers.

3.3.2 Early warnings of risk and 'authoritative assertions' of safety

During World War I, TEL was evaluated for possible use as a battlefield weapon. Mansfield Clark, a professor of chemistry familiar with this work, warned the US Public Health Service (PHS) in 1922 about the 'serious menace to the public health' that would arise from the use of TEL in petrol because 'on busy thoroughfares it is highly probable that the lead oxide dust will remain in the lower stratum.' (Mansfield Clark, 1922)

The Surgeon General of the US Public Health Service, Huge Cummings, responded some months later by writing to Pierre Dupont, Chair of the Board of GM, asking if the public health effects of TEL had been taken into account, 'since lead poisoning in human beings is of the cumulative type resulting frequently from the daily intake of minute quantities.' (Needleman, 1997)

Thomas Midgely replied by saying that GM had given the question 'very serious consideration ... although no actual experimental data has been taken'. However, they were confident that 'the average street will probably be so free from lead that it will be impossible to detect it or its absorption.' (Midgely, 1922)

Midgely's response has parallels in other *Late lessons from early warnings* case studies, including those addressing asbestos, CFCs and BSE in Volume 1 (EEA, 2001). In response to early warnings about public health hazards, interested parties often make 'authoritative assertions' about the absence of risk despite having little or no data to support their claims. 'No evidence of harm' is thereby mischaracterised as 'evidence of no harm'. This approach to early warnings of potential harm is still common ([3]).

In order to provide some evidence to back up their assertions of safety, GM paid the US government's Bureau of Mines to conduct some animal experiments, but within tight reporting constraints imposed by the Ethyl Corporation. These conditions included the replacement of the word 'lead' by 'ethyl', even in internal correspondence, and the submission of draft reports to the Ethyl Corporation for their 'comments, criticism and approval' before publication. The chief chemist in the Bureau of Mines 'raised his concerns about this censorship but was assured by his director that … it would not be so bad if the word lead were omitted as this term is apt to prejudice somewhat against its use' (Needleman, 1997).

([3]) For example, there are no studies in children of the potential head cancer hazard of using mobile phones: the early suspicions of risk have come from studies in adults only. Yet it is widely asserted that there are no risks to children from the use of mobile phones. In 2007, 2009 and 2011 the EEA issued 'early warnings' about the potential hazard of head cancers from mobile phones, particularly in younger people. See Chapter 21 on mobile phones.

Such contractual gagging had already been condemned as unprofessional by Yandell Henderson, Professor of Physiology at Yale, who had turned down an invitation by GM to study TEL two years earlier. He was now asked by the Bureau of Mines to join their investigation but he declined, saying that it was 'extremely unfortunate' that the work was being funded by GM as there was an 'urgent need for an absolutely unbiased investigation'. He was prepared to investigate the hazards but only 'on the assumption that so terrible a poison as TEL should not be generally introduced until absolute proof was available that no danger to the public would be involved'. Soon after, his long standing contract with the Bureau of Mines was terminated, as well as his contract with Standard Oil: an early example of the harassment of 'early warning' scientists which is repeated in this and other chapters.

Henderson was later to testify against the use of TEL in petrol at its 'one day trial' in 1925 (discussed in Section 3.4.1).

By 1925 industrial production of TEL had been under way for nearly two years but within months it had caused the dramatic deaths of a dozen or so workers and mental illness in many others (Box 3.5).

The news media ran dramatic headlines about the TEL deaths: 'Mad gas claims third victim' and 'Bar Ethyl gas as fifth victim dies' appeared above pictures of workers being taken away in straitjackets.

New York State then banned the sale of leaded petrol. This put pressure on the PHS and the TEL industry to somehow demonstrate that though **workers** may be at risk, partly because of their 'carelessness', or because they 'worked too hard', as the factory management claimed, the **public** would not be at risk from TEL in their car fuel.

Standard Oil had already confidently asserted that no 'perils existed in the use of this gas in automobiles', even though no evidence had been gathered to support that view.

The day after the fifth employee at their TEL plant died, Standard Oil's assertions regarding the safety of TEL received support from the Bureau of Mines, which published a report showing that animal studies indicated no risks to the public from TEL.

On 1 November 1924, *The New York Times* (1924) ran the headline: 'No peril to public after long experiments with motor exhausts'.

However, the Bureau of Mines report was heavily criticised. It was labelled as 'inadequate' by Cecil Drinker, editor of *The Journal of Industrial Hygiene*, David Edsall, Dean of Harvard Medical School, and others, including the Surgeon General (Drinker, 1925). They considered that the number of animals used was too small and the duration of exposure was too short to draw reliable conclusions about safety. These are still features of some current toxicology that can result in underestimation of hazards (see Chapter 26 on science for precautionay decision-making).

Some public health specialists supported the Bureau of Mines and the Ethyl Corporation, but again with confident assertions rather than robust evidence.

For example, Dr Emery Hayhurst of the Ohio Department of Health provided an unsigned

Box 3.5 TEL workers die in the 'house of butterflies'

On Thursday 26 October 1924, Ernest Oelgert, a TEL worker at Standard Oil's Bayway labs in New Jersey, began hallucinating and then became extremely paranoid, running round the plant saying that 'three were coming at me'. By Saturday he had to be forcibly constrained and taken to the nearest mental hospital where he died the next day.

Over the next five days, four other TEL workers from the plant died and another 35 showed severe neurological symptoms of lead poisoning. At the other two TEL workplaces, the DuPont plant at Deepwater, New Jersey, and GM's research lab in Dayton, Ohio, at least six other workers had died.

Despite their declared difficulties in getting the facts out of the companies and the hospitals, *The New York Times* journalists uncovered more than 300 cases of lead poisoning at the Deepwater plant. Workers called that TEL plant 'the house of butterflies' as they frequently had hallucinations about insects during their bouts of lead poisoning.

editorial in *The American Journal of Public Health*, stating that 'observational evidence' and other reports from around the country have 'corroborated the statement of complete safety so far as the public health has been concerned' (Ethyl Gasoline, 1925). Few people knew that, at that time, he was also a paid consultant to the Ethyl Corporation and advisor to the Bureau of Mines (Hayhurst, 1924).

These 'authoritative assurances' failed to quell public and scientific concern. The Surgeon General responded to requests for action from public health experts, who felt that both the public's health and the probity of the PHS were at risk from the TEL issue. He organised a high level conference of all the key actors, stating that leaded petrol 'is a public health question of extreme seriousness … if this product is actually causing slow poisoning and serious effects of a cumulative nature' (New York World, 1925).

The conference took place in Washington on 24 May 1925.

3.4 'Progress' or precaution?

3.4.1 The 'one day trial' of the 'gift of God'

Every major stakeholder was represented at the meeting. Industry opened the debate by making four main points: leaded petrol was essential to the industrial progress of America; all innovation entails risks; the deaths and disabilities caused by TEL in the manufacturing plants were due to the carelessness of the men in not taking precautions; and there was no risk to the public from the different exposure conditions in the streets, compared to the factories.

No 'innovation' other than TEL was discussed at the meeting, despite the declared intention of the Surgeon General to spend two or three days discussing alternatives to TEL. The toxicologist Robert Kehoe spoke first for industry, citing lack of evidence of risks to the public; he was followed by eight other industry representatives who took up the morning session.

The public health representatives used the afternoon to try to shift the burden of proof back to their opponents, arguing that industry needed to show that TEL was safe for the public, rather than public health scientists needing to show that it was dangerous.

Dr Yandell Henderson, Professor of Physiology at Yale, told the conference that lead was as serious a public health menace as infectious diseases. He foresaw that 'conditions will grow worse so gradually, and the development of lead poisoning will come on so insidiously ... that leaded petrol will be in nearly universal use … before the public and the government awakens to the situation' (USPHS, 1925). His claims proved to be prescient.

Dr David Edsall, Dean of Harvard Medical School, also dismissed the view of industry that 'nobody has shown any symptoms of lead poisoning'. He went on to say that: 'I cannot escape feeling that a hazard is perfectly clearly shown … here today, and that it appears to be a hazard of public moment, and that the only way it could be said it is a safe thing to continue with this hazard would be after very careful and prolonged and devoted study.' This did not happen until the 1970s.

Edsall was followed by Dr Touart, who had treated many of the workers. He too emphasised the central issue of the burden and strength of evidence: 'It seems to me that ... this ethyl gas is under suspicion and therefore should be withheld from public consumption until it is conclusively shown that it is not poisonous.'

Haven Emerson, Professor of Public Health at Columbia University, observed that industry's use of deaths as an indicator of hazard was unsound: information about functional and mental disabilities of those that did not die would also be needed.

However, industry was supported by some of the other public health scientists at the meeting. They observed that, while there was solid and direct evidence of industrial benefits from TEL, evidence on health risks to the public was not available. This asymmetry between short-term economic benefits and long-term health hazards is another continuing problem.

Dr Hayhurst of the Ohio Department of Health said that 27 months of public use of leaded petrol 'should have sufficed to bring out some mishaps and poisonings suspected to have been caused by TEL'. It had not, so he was prepared to declare that leaded petrol was safe.

His position was based on two weak assumptions: that existing statistics, collected for other purposes, provided reliable evidence of safety from a new technology; and that a short, two-year-period after first exposure would be enough to uncover any new hazards. These assumptions are still common in debates on current health hazards.

Some public health experts who supported industry at the meeting had earlier expressed their concerns about the health risks, but only in private. Hayhurst, for example, despite being a consultant for the Ethyl Corporation, had written to the PHS a week before the May meeting expressing the concerns that he shared with some of the PHS scientists, such as Dr Thompson, who had declared that 'lead has no business in the human body ... everyone agrees lead is an undesirable hazard and the only way to control it is to stop its use by the general public'.

However, Hayhurst continued his letter by noting that his scientific judgement was influenced by political and economic factors. 'Personally, I can quite agree with Dr Thompson's wholesome point of view but, still, I am afraid human progress cannot go on under such restrictions … if we are to survive among the nations. Dr Thompson's arguments might also be applied to the thousand and one other poisons and hazards which characterise our modern civilisation' (Hayhurst, 1925).

The country's foremost authority on lead, Alice Hamilton, told the May meeting that there was no way to know how to regulate leaded petrol so that it would be safe. 'You may control conditions within a factory ... but how can you control the whole country?' She later spelled out the dangers further, noting that even under the strictest factory conditions the use of lead resulted in poisoning, sooner or later (Hamilton, 1925).

The meeting seemed to be going the way of precaution and public health until Frank Howard, first President of the Ethyl Corporation, concluded the industry view: 'You have only **one** problem', he told the health scientists, 'is this a public health hazard?' Industry, he said, had other problems such as ensuring that automobiles and oil played a key role in the industrial progress of the nation. 'Our continued development of motor fuels is essential in our civilisation'. The development of TEL after a decade of research was an 'apparent gift of God'. What is our duty under these circumstances, he asked, 'should we say no: we will not use a material (that is) a certain means of saving petrol? Because some animals die and some do not die in some experiments, shall we give this thing up entirely?'

In a couple of rhetorical sentences he put the burden of proof back onto the public health scientists to prove that TEL was dangerous. He had also put them on the defensive by making them appear to be reactionaries who were retarding human progress and technological innovation on the unproven grounds that there could be public hazards.

The meeting ended after less than seven hours. The Ethyl Corporation announced that there would be a temporary ban on leaded petrol sales until a 'blue ribbon committee' of top-level scientists set up by the PHS after the meeting had studied the issue.

After the meeting Alice Hamilton thought that the direct involvement of the top scientists and decision-makers from industry and government would produce the right results, especially if there was 'a blaze of publicity turned on their deliberations'.

3.4.2 Blue ribbon committee findings

This perceived victory of objective science over short-term economic and political interests was short lived. The scientific review committee was under great time pressure to produce its report, so a very limited, seven month, study of 252 garage and filling station attendants, chauffeurs and factory workers was conducted. The committee concluded that 'at present there are no good grounds for prohibiting the use of ethyl gasoline [petrol] ... provided that its distribution and use are controlled by proper regulations'. A recommendation from committee member Winslow to continue the search for alternatives was omitted from the final committee report.

The report included clear caveats, however, stating that:

> 'Owing to the incompleteness of the data, it is not possible to say definitely whether exposure to lead dust increases in garages when tetraethyl lead is used. It is very desirable that these investigations be continued ... It remains possible that if the use of leaded petrol becomes widespread, conditions may arise very different from those studied by us which would render its use more of a hazard than would appear to be the case from this investigation. Longer exposure may show that even such slight storage of lead as was observed in these studies may lead eventually in susceptible individuals to recognizable lead poisoning or chronic degenerative disease of obvious character ... The committee feels this investigation must not be allowed to lapse.'

Panel 3.1 A road not taken: the alcohol alternative to lead in 1925

Bill Kovarik

The US Geological Service (USGS) and the US navy performed over 2 000 tests on alcohol and petrol engines in 1907 and 1908 and concluded that: 'In regard to general cleanliness, such as absence of smoke and disagreeable odors, alcohol has many advantages over gasoline or kerosene as a fuel. The exhaust from an alcohol engine is never clouded with a black or grayish smoke.'

USGS continued the comparative tests and later noted that alcohol was 'a more ideal fuel than gasoline' with better efficiency despite the high cost'. Others were also experimenting (see Box 3.6).

GM was also interested in long-term security of fuel supplies, as is apparent in an unpublished du Pont study drafted by a member of the firm's legal staff. According to the study (Wescott, 1936):

> '…An important special motive for this research [into ethyl alcohol] was General Motors' desire to fortify itself against the exhaustion or prohibitive cost of the gasoline supply, which was then believed to be impending in about twenty-five years; the thought being that the high compression motors, which should be that time have been brought into general use if knocking could be overcome, could more advantageously be switched to [ethyl] alcohol.'

The DuPont conclusion is supported by internal memos sent by Midgley. Alcohol was the 'most direct route ... for converting energy from its source, the sun, into a material that is suitable for a fuel…' he said in one internal memo.

To promote alcohol-blended fuels among automotive and chemical engineers in October 1921, Midgley drove a high compression ratio car from Dayton to an October 1921 Society of Automotive Engineers (SAE) meeting in Indianapolis using a 30 % alcohol blend in petrol. 'Alcohol has tremendous advantages and minor disadvantages,' Midgley told fellow SAE members in a discussion. Advantages included 'clean burning and freedom from any carbon deposit... [and] tremendously high compression under which alcohol will operate without knocking... Because of the possible high compression, the available horsepower is much greater with alcohol than with gasoline.'

'From our cellulose waste products on the farm such as straw, corn-stalks, corn cobs and all similar sorts of material we throw away, we can get, by present known methods, enough alcohol to run our automotive equipment in the United States,' he said. The catch was that it would cost two dollars per gallon. However, other alternatives looked even more problematic — oil shale would not work and benzene from coal would only bring in about 20 % of the total fuel need (Midgley, 1921).

Despite their enthusiastic support for alcohol as a 'fuel of the future', Midgley and his boss, Charles Kettering, categorically denied the existence of alternatives to TEL once they had begun to invest in TEL production facilities:

> 'So far as science knows at the present time, tetraethyl lead is the only material available which can bring about these [antiknock] results, which are of vital importance to the continued economic use by the general public of all automotive equipment, and unless a grave and inescapable hazard exists in the manufacture of tetraethyl lead, its abandonment cannot be justified' (*The New York Times*, 7 April 1925).

Information about alternatives could have emerged with more social and scientific force at this critical moment in the history of TEL ([4]). For example, it was widely thought that the May 1925 conference would last several days in order to discuss alternatives to TEL: the Surgeon General had declared his intention to do so at the opening of the May conference (see Section 3.4.1).

A report published but not released by the US Department of Commerce a few days before the May conference showed that alternative antiknock additives (mostly ethyl alcohol blends in petrol) were being

([4]) 'Ethyl leaded gasoline crashed through the modest defenses of the American public health system of the 1920s not only through brute force of industry's political influence over government but also due to the disorganized information resources available to public health advocates', particularly regarding the potential for alternatives to TEL (Kovarik, 2003).

Panel 3.1 A road not taken: the alcohol alternative to lead in 1925 (cont.)

used routinely in two dozen other industrial nations. And anyone familiar with the Midgley papers and statements of 1921 and 1922 would see that by 1925 he was contradicting his own published research.

Information about alternatives did not emerge from the 'one day trial' of TEL in 1925, however, except in a few statements by public health scientists and hints in the media. No record of any dissent exists, even though the industry was now flatly contradicting its own previous research and statements on the alcohol alternative.

In 1933 the US Defence Agency and US navy conducted tests on alternative fuels and found that Ethyl leaded petrol and 20 % ethyl alcohol blends in petrol were almost exactly equivalent in terms of brake horsepower and useful compression ratios. This report was never published.

Other potential substitutes for tetraethyl lead known to Kettering and the US automotive industry were based on the I.G. Farben/BASF Fischer-Tropsche and Bergius processes for making synthetic fuels from coal. This was seen as such serious competition to TEL that Standard Oil entered into a 'full marriage' agreement with Farben in which Standard agreed to stay out of the world chemical business and Farben agreed to stay out of the world fuel business — no matter how World War II progressed (Davis, 2007).

The wide variety of alternatives and substitutes known in the 1920s and 1930s were forgotten by the 1960s. Histories of the oil industry omitted any mention of alternatives.

In 1974, when Thomas Reed of MIT began his ground-breaking investigation of alcohol fuel as an alternative to petrol in the wake of the Arab oil embargo, he was unaware of any other similar work before him. It was as if, having found in TEL the one solution to the engine knock problem, no other solution — and no other history — was necessary.

Defeating the alcohol competition to leaded petrol

By the mid-1930s, Ethyl leaded petrol succeeded beyond all expectations. Public health crusaders who found this troubling still spoke out in political forums but competitors were not allowed to criticise leaded petrol in the commercial marketplace. In a restraining order forbidding such criticism, the Federal Trade Commission told competitors to stop criticising Ethyl petrol since it 'is entirely safe to the health of [motorists] and to the public in general when used as a motor fuel, and is not a narcotic in its effect, a poisonous dope, or dangerous to the life or health of a customer, purchaser, user or the general public.' (US FTC 1936)

During the 1930s, the few attempts to promote alcohol petrol were met by fierce and unfair competition from the Ethyl company, which led to an anti-trust case against Ethyl Corporation in 1937. By then Ethyl leaded petrol was used in 70 % or more of American petrol (90 % according to Ethyl's advertising) and in all but one major brand — Sunoco. Dealers who cut prices or who used alcohol or benzene in other fuels were not allowed to wholesale Ethyl's lead additive.

'It seems clear that the Ethyl Gasoline Corporation has exercised its dominant control over the use of Ethyl fluid substantially to restrain competition by regulating the ability of jobbers to buy and sell petrol treated with ethyl fluid and by requiring jobbers and dealers to maintain certain prices and marketing policies', a 1937 Department of Justice memo said. Ethyl lost the suit at the Federal District Court level in 1938 and at the Supreme Court in 1940. The company was ordered to make the product available to any customer who met minimum technical criteria.

Many scientists, businessmen and farmers believed that making fuel from corn and cornstalks would help put people back to work and ease the severe problems of the Depression. This movement for alcohol fuels became part of a broader campaign for industrial uses for farm crops to help fight the Depression. The 'farm chemurgy' movement, as it was called, with alcohol fuel as a controversial centrepiece, had grown into an unprecedented mixture of agronomy, chemistry and prairie populism. Many felt that the time had come to compete directly with the oil industry. 'Try a tankfull — you'll be thankful,' the Agrol brochures said. The blend was sold to initial enthusiasm at 2 000 service stations. Although Agrol sold for the same price as its 'main competitor', leaded petrol, it cost wholesalers and retailers an extra penny to handle it and this cut into their profit. By 1939, the Agrol plant had closed (Hale, 1934; Kovarik, 2003 and 2005).

Box 3.6 Early London buses experiment with alcohol fuels

In 1919 the London General Omnibus Co. also compared petrol with blends of ethyl alcohol and benzene. Mileage was about the same, with petrol slightly ahead. 'In all other respects the [alcohol] fuel compared favourably with petrol, and exhibited the characteristics of other alcohol mixtures in respect of flexibility, absence of knocking and cleanliness' (although it would later emerge that benzene is a dangerous carcinogen.)

The bus experiment also showed that a large-scale switch from petroleum was technically feasible and needed in any case to increase security of fuel Contentsies. 'We are fast squandering the oil that has been stored in the fuel beds, and it seems so far as our present knowledge takes us that it is to the fuels experimented with that we must turn for our salvation,' concluded the omnibus company engineer.

The Committee recognised the limitations of its small, interim and retrospective study and strongly urged the PHS to obtain funds from Congress for long-term prospective studies that could follow the history of leaded petrol and its consequences that were 'not now foreseen'. However, the PHS never undertook such research. For the next 40 years all studies of TEL were conducted and funded by the Ethyl Corporation and GM (Markowitz and Rosner, 2002).

Shortly after the Surgeon General's committee had declared that TEL was safe for general use, in 1926, the Public Health Service recommended that the allowable concentration of TEL be set at 3 cc per gallon. Ethyl quickly agreed to comply, relieving the government of any pressure to introduce the regulations on lead in petrol that had been called for by the expert committee.

For the next 35 years lead toxicity as a public health issue virtually disappeared from sight, while at the peak of TEL production some 250 000 tonnes of lead were released into the air in the United States every year.

3.5 Lead contamination is 'normal and safe'

After the Surgeon General's report of 1926 had given the go ahead to industry, Robert Kehoe, the toxicologist from the University of Cincinnati, who had claimed the safety of TEL at the 1925 meeting, was cultivated by the TEL industry as the dominant authority on lead. C. F. Kettering established a laboratory in Cincinnati with an initial gift of USD 130 000 from Ethyl, E. I. DuPont and General Motors. He had initially asked Kehoe to study the worker deaths at the Ethyl plant in Dayton and now he asked him to direct the Kettering laboratory.

Kehoe later also became a corporate officer at GM and a consultant to DuPont.

Data on the health effects of TEL were sparse, and the only source of funding for research came from industry sources. The strong recommendation from the Surgeon General's 1926 report that there should be publicly funded research on TEL was not implemented.

Kehoe's early studies compared lead concentrations in workers in direct contact with TEL with men in the same plant but who had other jobs. He designated this second group as 'unexposed' controls. When he found lead in the excreta of his so-called unexposed group, he concluded that as lead was naturally present in all the workers it could not be very harmful to them. The mere presence of lead in workers, he argued, could not be an indicator of poisoning.

This view had been vigorously attacked by David Edsall, Yandell Henderson and others at the Surgeon General's 1925 meeting. They had argued that, as potentially all workers in the Dayton plant were exposed to TEL fumes, any comparison of workers within the plant would be of little value as exposed controls would mask the full effects of lead.

Kehoe eventually came to see the merit in his critics' assertions: clearly he had chosen the wrong control group. To answer his critics he searched for an unexposed group in a remote farming village outside Mexico City, far removed from industry or urban pollution. There he sampled food, utensils and the excreta of the residents, which he found also contained lead.

This observation of 'natural' lead levels in Mexican farmers became the nucleus of Kehoe's position

throughout his career. From this observation he concluded that lead in petrol presented no danger to the public, making the same mistake in argumentation that he had made with the Dayton workers. He assumed that general lead contamination was 'normal' and therefore 'natural' and harmless at those 'low' levels. It was not until the geologist Clair Patterson questioned this view some 30 years later that this argumentation was successfully challenged: 'normal' lead exposures in the 20th century were far from 'natural' (Patterson, 1965).

The Second World War years saw the economics and politics of TEL plumbing new depths. The Ethyl Corporation and Standard Oil had continued to develop their business links with Hitler's Germany which they had begun in the 1930s when Ethyl formed the German company Ethyl Gemeinschaft and Standard Oil linked up with the largest German company, I.G. Farben, one of the main corporate supporters of Hitler. This enabled them to provide the German war machine with the technical ability to improve the fuel efficiency of their tanks, lorries and planes by using leaded petrol (Box 3.7).

Industry control of both the economic and public agenda seemed complete by the 1950s but a new perspective was emerging from well outside the TEL community, which would soon seriously challenge industry's virtual dominance of this intellectual terrain.

Is 'normal' lead contamination really 'harmless'?

In 1965, Kehoe's monopoly on lead data was threatened by a geochemist from outside the public health debates. Clair Patterson was a research associate in geology at the California Institute of Technology. His measurements of the isotopic ratios of certain minerals convinced him that the long-held consensus of geologists that the age of the Earth was 3 billion years old was wildly wrong. Patterson's studies placed the age of the earth at 4.5 billion years, a serious challenge to the orthodox scientific view.

His findings were fiercely rejected by believers in the conventional paradigm but they were eventually confirmed, his sceptics refuted, and the geology textbooks revised.

Patterson uncovered the errors in the conventional geological view by employing extraordinary measures to avoid contamination while collecting and analysing his specimens. As a result his measurements were much more accurate than those of earlier workers.

Many scientists would have treated the contamination of his reagents as a technical annoyance to be overcome and then forgotten. To Patterson it was not a nuisance but a clear indication of lead contamination from human activities, which needed to be further investigated. From the depths of the Pacific Ocean he brought tuna to the surface with extreme care to avoid contamination. He studied pre-iron age mummies that had been buried in sandy soil and he sampled cores from the Greenland ice pack. By slicing the ice cores he was able to date the specimen precisely and show the time course of lead in the atmosphere.

Patterson and his colleagues showed that technological activity had raised modern human body lead burdens to levels that were some 600 times higher than that of our pre-industrial ancestors.

In 1965, in response to an invitation by the editor of the *Archives of Environmental Health*, he submitted a long article entitled 'Contaminated and natural lead

Box 3.7 TEL and 'treason' in Germany

In March 1942, Thurman Arnold, a US Assistant Attorney General, told a Senate Committee investigating war profiteering that without the leaded petrol from Ethyl, the Nazis could not have flown their planes or fuelled their land vehicles so efficiently. The Chairman of the Committee, Harry Truman, called the alliance between some American companies and I.G. Farben 'treason'.

A German memo found after the war supported his view: 'It need not be especially mentioned that without TEL the present methods of warfare would be impossible. The fact that since the beginning of the war we could produce TEL is entirely due to the circumstances that shortly before the war the Americans had provided us with the production plans complete with their know-how. It was moreover the first time that the Americans had decided to give a licence on this process to a foreign country ... and this only on our urgent request to Standard Oil to fulfil our wish' (Davis, 2007).

environments of man'. Kehoe was asked to review the manuscript and to decide whether it should be published. Kehoe argued for the paper's publication so that Patterson could be offered up for demolition. 'I should let the man, with his obvious faults, speak in such a way as to display these faults.'

He went on: 'The inferences as to the natural human body burden of lead are, I think, remarkably naïve. It is an example of how wrong one can be in his biological postulates and conclusions, when he steps into this field, of which he is woefully ignorant, and so lacking in any concept of the depth of his ignorance that he is not even cautious in drawing sweeping conclusions. This bespeaks the brash young man, or perhaps the not so young [Patterson was 43 at the time] passionate supporter of a cause. In either case, hardly the mark of the critical investigator. It must be faced and demolished, and therefore, I welcome its 'public appearance'.' (Kehoe, 1965).

Patterson's *Archives of Environmental Health* paper fundamentally altered the vocabulary of the debate over the health effects of lead. He recognised that because a certain level of lead was commonplace it did not mean it was without harm. He argued that the term 'normal' should be replaced by 'typical'. 'Natural' should be reserved for those concentrations of lead that existed in the body or environment before contamination by human activities.

It also showed that the so-called 'unexposed' subjects in Kehoe's studies of the Dayton plant workers, or his Mexican farmers, were contaminated by lead, and this lack of a truly unexposed part of the study population would dilute or hide risks of exposure. This dilution of risks by background contamination is now a much more common problem for public health, given the widespread exposures of most people to low levels of chemicals and radiations.

The *Archives of Environmental Health* paper released a fusillade of angry responses from orthodox toxicologists. Their fury focused on Patterson for his hubris in stepping outside his field to talk about people instead of rocks, but they also attacked the editor of the *Archives* journal. This is another example of 'shooting the messenger' which pervades this and most other 'Late Lessons' stories, from John Snow and cholera in 1864 (EEA, 2001) to those current scientists who publish warnings of hazards about climate change, genetically modified organisms and electromagnetic fields.

The controversy over Patterson's paper crystallised the opposing views held by him and Kehoe. Those who adhered to Kehoe believed that lead poisoning occurred only at high doses with obvious signs of severe illness. Patterson clearly spelled out the other position: elevated levels of lead found in all humans were associated with sometimes silent disturbances in body chemistry. Perhaps, Patterson argued, everyone was poisoned to some extent.

For the TEL industry, however, much more than professional reputation were at stake. A group from Ethyl Corporation visited Patterson and tried, in his words, to 'buy me out through research support that would yield results favourable to their cause.' He refused to cooperate (Patterson, 1992).

Following the meeting with Ethyl, his longstanding contract with the Public Health Service was not renewed, and his substantial contract with the American Petroleum Institute was terminated. Members of the Board of Trustees at California Institute of Technology visited the chairman of his department asking that he be fired. Patterson responded with a lecture in which he predicted that future scientists would show that Ethyl's activities were poisoning both the environment and people, and that their operations would eventually be shut down.

Publicly vilified and professionally threatened, Patterson would eventually be recognised by the scientific establishment for his extraordinary contributions to science. He would win the Goldschmidt Medal, the equivalent of the Nobel Prize in geochemistry, be elected to the National Academy of Sciences, and have both a mountain peak in Antarctica and a large asteroid named after him. He also provided the main character for a Saul Bellow novel (Box 3.8).

3.6 1966: US Congress asks awkward questions

In 1966, Senator Edward Muskie, Chairman of the Senate Subcommittee on Air and Water Pollution, presided over hearings on the future Clean Air Act of 1970. He gave considerable attention to the status of lead in the air and in petrol. The Surgeon General, William Stewart, one of the first to testify, gave testimony that revealed the government's concern, perhaps for the first time, about the effects of lead at low doses, particularly in children and pregnant women:

> 'Existing evidence suggests that certain groups in the population may be particularly susceptible to lead injury. Children and pregnant women constitute two of the most

Box 3.8 Saul Bellow and 'zones of incomprehension' in scientists

In *The Dean's December*, Saul Bellow described Professor Sam Beech, a character easily recognisable as Clair Patterson, his friend. 'These scientists were diapered babies when they went public with a cause. But Beech somehow inspired respect … He had authoritatively dated the age of the earth, had analysed the rocks brought back from the moon.'

Bellow describes Beech's theories about the relationship between lead and social disorder and the chilly reception they received from the orthodoxy. 'Here science, which itself was designed for deeper realisation, experienced a singular failure. The genius of these evils was their ability to create zones of incomprehension. It was because they were so fully apparent that you couldn't see them' (Bellow, 1982).

> important of such groups. Some studies have suggested an association between lead exposure and the occurrence of mental retardation among children' (Needleman, 2000).

Once again Kehoe was the industry's principal witness. Two years earlier he had said that enough was known about TEL toxicity to allow the amount of TEL to be increased without risk, noting: 'that no other hygienic problem in the field of air pollution has been investigated so intensively, over such a prolonged period of time, and with such positive results'. When Muskie pointed out that the Public Health Service and others disagreed with Kehoe and that many felt that there were unanswered questions and a need for more research, Kehoe responded:

> 'I would simply say that in developing information on this subject, I have had a greater responsibility than any other persons in this country. The evidence at the present time is better than it has been at any time that this is not a present hazard' (Needleman, 1998).

However, Muskie pressed on: 'would it be desirable if a substitute for lead in petrol could be found?' Kehoe replied: 'There is no evidence that this (TEL) has introduced a danger in the field of public health … I may say the work of the Kettering Laboratory … has established that ... lead is an inevitable element in the surface of the earth, in its vegetation, in its animal life, and that there is no way in which man has ever been able to escape the absorption of lead while living on this planet'.

One week later Clair Patterson testified. He began by attacking the belief that natural lead cycling and human activity contributed about the same amount of lead to the environment. About 10 thousand tonnes of lead were naturally recycled each year, he said, while millions of tonnes were emitted due to industrial and transport emissions. Large numbers of people are sickened, he believed, as a result of this unnatural load, and the brain is the most significant target. Patterson attacked the PHS for relying on industry data:

> 'It is not just a mistake for public health agencies to cooperate and collaborate with industries in investigating and deciding whether public health is endangered; it is a direct abrogation and violation of the duties and responsibilities of those public health organisations. In the past, these bodies have acted as though their own activities and those of the lead industries in health matters were science, and they could be considered objectively in that sense. Whether the best interests of public health have been served by having public health agencies work jointly with representatives of the lead alkyl industries in evaluating the hazards of lead alkyl to public health is a question to be asked and answered.'

Industry had traditionally measured the prevalence of lead toxicity by counting deaths, or at least severe damage to the brain. Muskie raised the question of a larger pool of unrecognised toxic illness, an issue that had been first raised at the one day trial of leaded petrol in 1925 (Needleman, 2000): 'Is it conceivable that there is something different in the deleterious effects on health from low-level exposure than from more concentrated exposure leading to classical lead poisoning?'

Patterson replied: 'when you expose an organism to a toxic substance it responds in a continuum, to continuously changing levels of exposure to this toxic substance. There is no abrupt change between a response and no response. Classical poisoning is just one extreme of a whole continuum of responses of an organism, human organism, to this toxic metal.'

Muskie's inquiry marked the government's shift away from complacency about the hazards of lead. His Senate hearings established a new premise: that lead poisoning was not only a disease of workers; it could be an insidious, silent danger to the public. The notion that lead poisoning was an all-or-nothing phenomenon was discredited and replaced by degrees of disease spanning across a biological continuum of 'effects' to 'adverse effects'. The issue still dominates current discussions about chemicals, radiation and other public health hazards, where early 'effects' are often dismissed as having no biological or ecological significance.

3.7 Lead in petrol poisons catalytic converters — so it's got to go

In 1962 GM sold Ethyl and in 1970 GM announced that it would begin installing catalytic converters in its new models in order to comply with the Clean Air Act of 1970. As a result, GM stated, it would be necessary to phase out lead in petrol as it was poisonous for the platinum in the catalytic converter. Apparently, poisoning a technology was more important than poisoning people.

To Ethyl's management this was a betrayal and they resolved to fight the growing environmental movement in the United States. They argued that it was fully justified to speak out for this additive, which had saved billions of dollars for the American economy and helped make the modern automobile possible. To combat lead regulation, it formed a defence team and called it, with unconscious irony, the 'Ethyl Air Conservation Group'. The Group was staffed with Ethyl officials and members of the Hunton and Williams law firm. Lawrence Blanchard, a partner in Hunton and Williams and board member of Ethyl, headed the group.

EPA medical officers continued to push for a separate health standard, fearing that if a substitute for platinum were discovered sometime in the future, lead would return to fuel. In 1973, aware that 200 000 tonnes of lead were emitted from the exhausts of American cars each year, the EPA promulgated a regulation phasing down lead content in all petrol. Its target was to reduce lead in petrol to 0.5 g/gal within five years (Schoenbrod, 1980).

The TEL industry responded by skilfully exploiting the growing national anxiety about fuel supplies caused by the spike in the price of oil, which had reached unprecedented levels by 1973. The EPA estimated that the oil penalty from phasing out lead was 30 000 barrels per day. Industry's calculations were different: on 2 December 1973 a full page advertisement appeared in *The New York Times* showing an oil barrel bearing an American flag pouring oil down a manhole. Its headline proclaimed that removing lead from petrol would have the effect of dumping one million barrels of oil a day.

On 6 December 1973, however, the EPA released the final regulations requiring a phased reduction of lead in petrol to protect health. Ethyl Corporation and DuPont sued in court, arguing that removing lead would cost an enormous amount of money and crude oil resources; that no one had been poisoned by lead in air; and that any effects in humans reported at low doses of lead were not **adverse** health effects. The court agreed with industry, setting aside the regulations as 'arbitrary and capricious'.

On appeal, the earlier judgement was overturned and the EPA regulations upheld. The court stated that 'the regulatory action under this precautionary statute [the Clean Air Act] should precede, and hopefully prevent, the perceived harm.' Furthermore, 'in making his policy judgment by assessing risks the Administrator is not required to limit his consideration to the danger presented by lead additives 'in and of themselves'. He may consider the cumulative impact of lead additives with other sources of human exposure to lead' (Ethyl Corp. v Environmental Protection Agency, 1976).

Ethyl, PPG Industries, DuPont, NALCO Chemical and the National Petroleum Refiners Association then appealed to the Supreme Court, where they lost.

3.8 Public funding to study lead poisoning in children

In 1970, the US Surgeon General had called for early identification of children with 'undue' lead exposure. His statement avoided the loaded term 'poisoning' but indicated that this was probably more lead than a child should have. For the first time since 1925 significant research funds were allocated from Federal sources to study the health impacts of lead on children. The industrial monopoly on scientific data was drawing to an end.

Professor Herb Needleman was one of the public health scientists who used the recently released public funds to research the low-dose effects of lead on children's IQ. His seminal paper on the subject (Needleman et al., 1979) showed that the higher the lead content, the greater the negative impact on IQ. His work shifted another paradigm by focusing not on the **flow** of blood through the body but on the

Box 3.9 Lead in petrol: 'it's all about economics...'

Late one night after a long day's work on the issue of lead in petrol, Herb Needleman and others from the EPA expert committee had dinner at the home of an EPA staffer. After dinner and a liberal amount of red wine, Needleman asked Jacobs from DuPont why, with its wealth of excellent research chemists, it had not developed a safer petrol additive to replace TEL. In Needleman's words:

'Jacobs, who had matched my intake, told me that their economists had modelled the future sales of leaded gasoline and projected that the consumption of gasoline would soon level off, and perhaps decline. Given such a projection, the company would not invest USD 100 million in research and development funds. I learned a valuable lesson that night: the entire debate about scientific studies, about the health risks for children, was merely a shadow play. The real decision had been made by DuPont's economists. Their plan was clear: don't budge on TEL and seek medical and environmental arguments to support the choice' (Needleman, 2000).

stocks of lead in the bones. His innovation was to analyse 'milk' teeth from more than 2 000 infants and to correlate their lead content with their later development in terms of intelligence and behaviour.

He observed that the average IQ of this group of children fell by 5 points, a shift that was dismissed by industry as 'small' and insignificant. This view ignored the effect on very large groups of children who were at both ends of the normal distribution of IQ, i.e. those either severely handicapped or exceptionally gifted, whose numbers would be doubled and halved respectively (Bellinger and Bellinger, 2006) ([5]).

Industry responded to this dramatic observation with unprecedented opposition, resorting eventually to a character assassination of Needleman.

Needleman later followed up the 1943 Byers discovery of the **chronic** anti-social behaviour of children who had 'recovered' from **acute** lead poisoning, confirming the association between childhood lead poisoning and anti-social adolescent behaviour (Needleman, 1996). Studies have further confirmed the link between lead and anti-social behaviour (WHO, 2010).

3.9 1977–1995: the phase down of lead in petrol

The EPA published its *Air quality criteria for lead* in December 1977, which stated that lead in air and in dust was a significant source of human exposure to lead, and that brain damage could occur in individuals with no acute symptoms of lead poisoning. The Air Office of EPA used the new criteria document to determine a standard for lead concentrations in air.

With the new standard in place and the gradual retirement of old cars that ran on leaded fuel, air lead levels began to fall. In 1977 air concentrations in Philadelphia ranged between 1.3 and 1.6 μg/m^3, whereas by 1980 the concentrations were between 0.3 and 0.4 μg/m^3 (Needleman, 2000). Similar trends were observed in most major cities.

Between 1976 and 1980, the amount of lead consumed in petrol production dropped by 50 % and the blood lead level of the average American dropped by 37 %. Furthermore, in its second volume of the *Air quality criteria for lead* the EPA concluded that, contrary to the claims of the industry, the relationship between petrol production and air lead levels was causal. It noted that between 1975 and 1984 the lead consumed in petrol had decreased 73 %, while the corresponding composite maximum quarterly average of ambient air lead had decreased by 71 % (USEPA, 1986).

In the 1970s the toxic threshold for lead in blood was defined as 60 μg/dl. The reduction of blood lead levels gradually allowed comparisons with children whose background blood lead levels were 1μg/dl or less. As a result, effects of lead on children's IQ have been found at levels below 10 μg/dl, with most of the cognitive impairment seeming to occur at blood lead levels as low as 5 μg/dl (Lanphear et al., 2000).

([5]) Chapter 23 on costs of inaction provides an illustration of this effect in Figure 23.1.

Box 3.10 Lead levels in blood decline

The phasing-out of leaded petrol between 1976 and 1995 was associated with a more than 90 % reduction in the mean blood lead concentration (Annest et al., 1983; CDC, 1997; Jones et al., 2009). The percentage of children in the United States aged between one and five with blood lead levels greater than or equal to 10 µg/dl declined from 77.8 % in the late 1970s to 4.4 % in the early 1990s, and the average lead level of a child in the United States declined to 1.9 µg/dl between 1999 and 2002 (CDC, 2005). At the same time, lead was eliminated from solder used in food cans and new residential paint products (President's Task Force, 2000). An estimated gain of 5–6 points in mean population IQ score was associated with the decline in mean blood lead concentrations, and this gain in IQ has been calculated to yield an annual economic benefit of between USD 100 billion and USD 300 billion in each birth cohort on the US (Grosse et al., 2002).

Similar reports of success in reducing the harm from lead in children level were achieved in Europe and elsewhere as they began to phase out lead in petrol.

In a number of rapidly industrialising countries, too, including China, El Salvador, India, Mexico and Thailand, declines in blood lead levels have followed the removal of lead from petrol (OECD, 1999; Mathee et al., 2006; He et al., 2009). Worldwide, unleaded petrol now accounts for an estimated 99 % of total sales.

Source: WHO, 2010.

Box 3.11 Continuous reductions in the 'safe' level of lead 1960–2010

In the 1960s, an elevated paediatric lead level was defined by the United States Department of Health and Human Services Centers for Disease Control and Prevention (CDC) as a concentration in whole blood of 60 µg/dl.

In the 1970s, the level was reduced to 40 µg/dl, and then to 30 µg/dl. In the 1980s, it was reduced to 25 µg/dl. Most recently, in the early 1990s, the CDC reduced the blood lead level of concern to 10 µg/dl, the level that remains in place today (Surkan et al., 2007).

An international pooled analysis of data from seven cohorts showed an increase in blood lead level from less than 1 µg/dl to 10 µg/dl was associated with a six IQ point decrement, which is considerably greater than the decrement associated with an increase in blood lead level from 10 µg/dl to 20 µg/dl. (Lanphear et al., 2005).

In 2004, 16 % of all children worldwide were estimated to have levels above 10 µg/dl (WHO, 2010).

In 2010 the European Food Standards Agency withdrew its support for a provisional tolerable weekly intake guideline value on the grounds that it was inadequate to protect against IQ loss (EFSA, 2010).

3.10 The pros and cons of leaded petrol

Leaded petrol was finally completely phased out in the US in 1995, seventy years since the 'one day trial' in 1925.

The benefit of taking lead out of petrol exceeded the predictions of even the most convinced lead advocates. Lead levels in children's and adults' blood continued to drop in direct relationship to the reduction in lead in petrol. The average American child's blood lead level in 1976 was 13.7 µg/dl. In 1991 it was 3.2 µg/dl and in 2000 it was 2.0 µg/dl (WHO, 2010).

The health and other costs of lead damaged workers, child bearing women and children to the taxpayer, the Health service and to the economy have been huge and have persisted for decades after the leaded petrol phase out, as contamination persists in soils and dusts (Mielke, 2010). This damage to health has also had large economic consequences as outlined in Box 23.1 in Chapter 23.

Panel 3.2 EU policymaking on lead in petrol — a brief summary

Nigel Haigh ([6])

EU policymaking on leaded petrol emerged mainly from the activities of the UK and Germany. In 1971, the UK government received advice from its Chief Medical Officer that air lead levels should not be allowed to increase above current levels. The government responded by deciding on a phased three-stage reduction in petrol's lead content, from 0.84 to 0.4 g/l to be achieved in 1976. This phase down was then delayed and the deadline postponed until required by the subsequent EC directive. In the same year the German government decided to reduce lead levels but chose a faster programme, which was implemented as planned: 0.4 g/l in 1972 and 0.15 g/l in 1976.

In both countries this initial action arose in response to scientific advice, without much public pressure. The more leisurely approach of the UK government was possibly linked to the fact that the largest European plant then manufacturing lead additives for petrol was in the United Kingdom.

In 1971 the UK was not yet a member of the European Communities (EC) and it was Germany's unilateral decision that resulted in the EC Commission establishing two committees in 1971 to study the health and technical aspects of lead pollution from motor vehicles. The memorandum concluded that although there was no immediate danger to public health, it was desirable to prevent an increased lead pollution in air. Increasing car use and cross-border sales of petrol by oil refiners therefore warranted EC-wide limits on lead in petrol.

On 10 November 1975, at the European Parliament, the rapporteur of the Environment Committee said that the proposed second stage reduction of lead to 0.15 g/l for regular grade had met with insurmountable opposition in the Committee because it would have required industry to make substantial investments and increased petrol consumption. Since these objections could not be refuted, the Committee preferred to require the Commission to postpone the introduction of the second stage. The Committee did, however, approve the first stage limit of 0.4 g/l.

All subsequent discussion in the Council — where decisions then had to be taken unanimously — was coloured by the existing German limit of 0.15 g/l. Directive 78/611 therefore had to allow Member States to introduce national limits of 0.15 g/l but its main provision was an upper limit of 0.4 g/l.

This example shows how in favourable circumstances a determined Member State can lead its peers despite considerable opposition and scientific uncertainty. In doing so, Germany ensured that higher environmental standards were achieved more quickly than if the Member States had proceeded at their own pace.

In the United Kingdom in 1981, following the report of a scientific committee on lead and health, chaired by Professor Lawther (Lawther, 1980), and of a government working party on lead in petrol (WOPLIP) (UK Department of Transport, 1979), the government decided to require petrol's lead content to be limited to 0.15 g/l, the lowest level that could be required under the Directive. It did not propose lead-free petrol. This recommendation followed a major battle within government: the health and environment ministries were defeated on the second point by transport, energy and the treasury.

Then there was a dramatic change in policy. In April 1983 the Royal Commission on Environmental Pollution (1983) recommended that the government initiate negotiations with the European Commission and other Member States to secure removal of the lower limit of lead in petrol in Directive 78/611 so that at the earliest practicable date all new cars should be required to run on lead-free petrol. The Government immediately accepted this recommendation.

Between these two decisions in the UK (1981 and 1983) there was an extraordinary public campaign. A new organisation called CLEAR (campaign for lead-free air), supported by a millionaire, provided very effective political lobbying and also publicity for the scientific information. It is possible that the Royal Commission only decided to look at the issue of lead because of the campaign, although that is not the view of its Chairman (Richard Southwood). What can be said with some certainty is that the government only endorsed the Commission's conclusions so quickly (within half an hour of publication) because of the campaign and because of an imminent general election.

([6]) This panel is based on extracts from Haigh (1998).

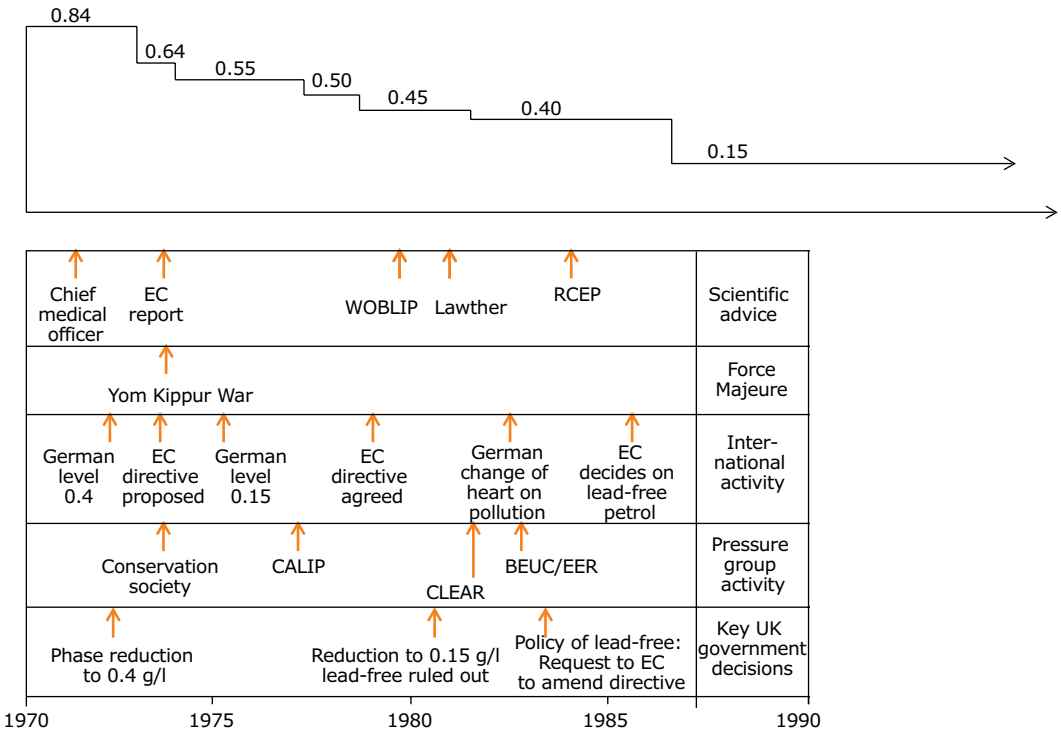
Panel 3.2 EU policymaking on lead in petrol — a brief summary (cont.)

When the first approaches were made to the European Commission in April 1983 the reaction was negative but the coincidence that changed the debate was the concern in Germany about forest die-back, partly caused by air pollution. Germany realised that to achieve its objective of significant NO_x reductions from cars, catalytic converters would be required. Since lead poisons catalytic converters, it would have to be removed. Germany, together with the Netherlands and Denmark, then supported the UK initiative and Directive 85/210 was adopted.

The catalysts for action in the United Kingdom and Germany were similar, but public conscience was excited by two quite different issues: public health and death of forests. It is pure chance that they came together at the same time and if either had been missing it is quite possible that the directive would not have been agreed, or not agreed so quickly. If we are tempted to speculate further, what would have happened if someone had invented a lead tolerant catalytic converter?

Science plays a unique and essential role in informing the public and influencing and guiding public opinion which is a major determinant of policy. But science itself does not always reach the public at a specific point in time when a specific decision is called for. Since we cannot yet claim that there is a European public, but only a collection of national and regional publics, the way the policy debate develops under the pressure of public opinion, more or less informed by science, is very likely to differ between countries. European policymaking is very much about reconciling these differences.

Figure 3.1 Key events which help to explain reduction in lead levels in petrol in Germany, the United Kingdom and the EU



Source: Reprinted with permission from Haigh, 1998.

Panel 3.3 Lead in petrol: a reflection on the German experience

Hans von Storch et al. ([7])

Environmental matters in the early 1970s featured strongly in German politics (Peters, 1980), and Germany was the first European country to impose restrictions on the lead content in petrol. From 1972, German production and importation of petrol with more than 0.4 g Pb/l was prohibited (down from the usual 0.6 g Pb/l), and from 1976 the stricter limit of 0.15 g Pb/1 was imposed. A preliminary analysis of newspaper coverage found that the health dangers of leaded petrol entered the German press in the 1960s. Comparable British articles at that time focused on urban smog.

Unleaded petrol (0.013 g Pb/1) was introduced in Germany in October 1984. Prohibiting the sale of leaded petrol in Germany was not an option because the European Union did not then allow such trade restrictions among its members. Instead, Germany introduced tax incentives for unleaded petrol in 1984, and in 1985 its availability at all German gas stations became mandatory. Enhanced tax incentives in 1986 made German unleaded petrol cheaper than the leaded variety, and its market share increased steadily.

In 1985, the EU mandated that by October 1989 super unleaded petrol had to be available for sale in all member states, alongside the leaded variety (Council Directive 85/210/EEC). In addition, member states were asked to adopt a 0.15 g Pb/1 limit voluntarily. Unleaded petrol was defined as containing no more than 0.013 g Pb/1. In 1987, Directive 87/416/EEC emphasised the importance of the availability of unleaded petrol for sale in every country. All Member States were then allowed to prohibit national production and sales of leaded 92-octane petrol because of damage to public health and the environment.

According to Löfgren and Hammar (2000), by 1995, unleaded petrol had conquered over 80 % of the market in Germany, Sweden, Finland, Denmark, the Netherlands and Austria, but less than 30 % in France, Greece and Portugal. Higher leaded petrol prices and the widespread adoption of cars using leadaverse catalysts were the two most important factors in reducing the market share of leaded petrol. Löfgren and Hammar also note the importance of effectively informing the public that unleaded petrol can safely be used with non-catalyst cars.

Road lead emissions totalled an estimated 31 000 metric tonnes in 1955 in Europe and this nearly quadrupled to 119 000 in 1975 with increasing car use. While road transport and petrol consumption continued to rise, subsequent petrol lead content regulations nearly halved road lead emissions to 62 000 tonnes in 1985. As unleaded petrol conquered increasingly higher market shares, road lead emissions dropped further to 42 000 tonnes in 1990 and to 19 500 in 1995.

Overall, favourable terms of competition were experienced by producers of cars with high technical standards, who had already gathered experience with catalyst systems on the US market (Hagner, 2000).

Blood levels in Germany with and without the reduction of lead in petrol

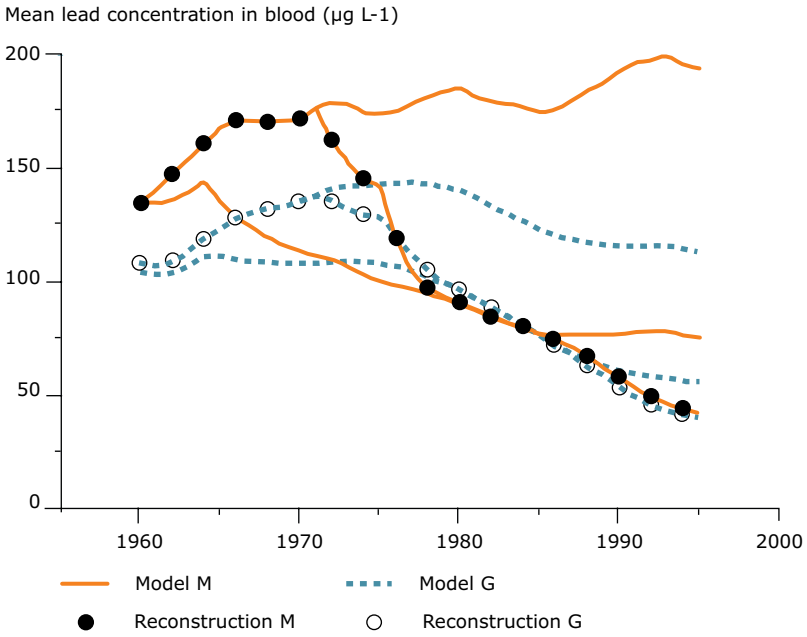
In the 1970s, lead in blood (PbB) values were reaching a level that health officials considered potentially harmful for foetuses and small children. To estimate how PbB levels may have developed if regulations of the use of lead in petrol had been implemented differently a model based on lead emissions was applied. In the case of no or delayed regulations, the model estimates that PbB levels well beyond the critical level would have emerged. Thus, the regulation instituted in Germany since the 1970s has reduced health hazards significantly.

The macroeconomic costs of the regulation seem to have been insignificant in spite of concerns that they would be substantial (Hagner, 2000). In fact, the case of leaded petrol demonstrated the limited utility of purportedly objective cost-benefit analyses, as the costs claimed at the time of the regulations turned out to be significantly biased, due to the vested interests that supported the analyses.

([7]) Adapted from von Storch et al. (2003) with permission from authors.

Panel 3.3 Lead in petrol: a reflection on the German experience (cont.)

Figure 3.2 Scenarios for mean PbB (µg L-1), as derived by the Münster model (M, orange, continuous) and for the Germany model (G, blue, dashed)



Note: Scenario 1 presents an evolution without regulation (i.e. continuing use of 0.6 g/l lead in petrol in Germany, upper curves).

In scenario 2 no unleaded petrol has been introduced in Germany in 1985 (middle curves).

In scenario 3, the regulation was instituted in Germany already in 1961 (lower curves). The reconstructed lead levels in blood are also given as open (Germany model) and full circles (Münster model).

Source: Helmholtz-Zentrum Geestacht Centre for Material and Coastal Research

The conclusion of a successful regulation in terms of limiting risks for human health should not downplay the consequences of the introduction of tetraethyl lead as an anti-knock additive in petrol, in particular since alternatives were known and available already in the 1920s and 1930s (Kitmann, 2000). Heavy metals such as lead pose a large-scale and long-term environmental problem as reduced emissions have limited influence on accumulations in the soil, which will remain for centuries. The strategy of protecting the environment from persistent substances must be based on continuous assessment and precautionary principles (Johansson et al., 2001).

Panel 3.4 The UK experience — expert risk assessments and public campaigns

Erik Millstone

In the United Kingdom, a committee examined the possible dangers of TEL use and submitted their report to the Minister of Health in 1930 (Departmental Committee on Ethyl Petrol, 1930). The committee received advice from several experts, including Dr Kehoe and US Surgeon General Cummings.

Cummings had moved from initial concern to the enthusiastic promotion of TEL writing dozens of letters touting Ethyl leaded petrol to public health leaders around the world. The fact that Cummings reported to Treasury Secretary Andrew Mellon, whose Gulf Oil Co. had exclusive contracts to distribute Ethyl petrol in the south-eastern US, may have had something to do with his enthusiasm.

The committee concluded that 'the widespread use of Ethyl petrol as a motor fuel for motor vehicles would not, in our opinion, increase the proportion of particulate lead in the atmosphere of our streets to such an extent as to constitute a risk even to the health of that part of the population which is most exposed — namely, police officers on traffic control duty and drivers of motor and other vehicles'.

Given the assurances from this report, TEL readily came into use in the United Kingdom and the rest of Europe.

By the late 1970s there was evidence of high levels of lead exposure in Britain and strengthening evidence of the toxicity of lead, even at low levels of exposure. The UK government responded by establishing a committee of enquiry. The Lawther report (as it came to be known) reported in 1980 that: 'We have not been able to come to clear conclusions concerning the effects of small amounts of lead on the intelligence, behaviour and performance of children' (Lawther, 1980). That statement was highly controversial. It was subsequently repudiated by several members of the committee for having been overly timid and was criticised by other lead experts (Rutter, 1983; Bryce-Smith and Stephens, 1980). Despite downplaying the dangers of lead in petrol, Lawther nevertheless advised the government and industry to reduce emissions of lead into the atmosphere progressively, without explaining why that advice was provided.

The government subsequently tried to represent the report as if it had proved that children's blood lead levels were entirely harmless. And British Petroleum and Associated Octel, which produced leaded petrol, continued to downplay the toxicity of TEL and atmospheric lead pollution, and the British government resisted efforts to reduce the lead concentration in petrol.

In general, scientists adopted a very cautious approach. For example, a report under the auspices of the Medical Research Council concluded that: 'While the observed statistical associations detailed in this review are consistent with the hypothesis that low-level lead exposure has a small negative effect on the performance of children in ability and attainment tests, the limitations of epidemiological studies on drawing causal inferences are such that it is not possible to conclude that exposure to lead at current urban levels is definitely harmful' (MRC Advisory Group on Lead, 1988).

That approach was marginally modified after the Royal Commission on Environmental Pollution pointed out in 1983 that: 'We are not aware of any other toxin which is so widely distributed in human and animal populations and which is also so universally present at levels that exceed even one tenth of that at which clinical signs and symptoms occur' (RCEP, 1983).

The Lawther committee had recommended that if a child was found to have a blood lead level (or PbB) above 35 µg/dl then steps should be taken to ascertain the source of exposure, and to reduce them (Lawther, 1980). By then, however, evidence of adverse effects below that level was available implying a maximum blood lead target significantly below 35 µg/dL (Chishold, 1976; Needleman, 1979). The Lawther report also neglected to recommend the establishment of a screening programme to identify children with elevated blood lead levels. The committee did, however, recommend that 'There should be a programme for the detection of lead in paint coatings accessible to children in areas where a high incidence of old lead paint surfaces may be suspected, such as old inner city residential areas.' That recommendation was sensible but 30 years later has not yet been properly implemented.

Panel 3.4 The UK experience — expert risk assessments and public campaigns (cont.)

When in 1980, the Lawther Committee recommended that, where a child was found to have a PbB level above 35 µg/dL, an investigation should be conducted to identify and reduce their sources of exposure, it was merely reiterating a policy to which the British government and all other EEC Member States had already agreed three years previously. In 1981 the Department of Health went marginally further when it advised that any child with a PbB over 30 µg/dL should be followed up (Quinn and Sherlock, 1990).

In 1982 the UK government shifted its position and set a maximum figure or 'action level' for lead in the blood (PbB) at 25 µg/dL. When it did so, that decision was made by reference to the results of a blood lead survey rather than toxicological considerations. Surveillance work had indicated that the vast majority of the population then had PbB levels below 25 µg/dL, and therefore endorsing that figure as an 'action level' necessitated no further remedial action. This exemplifies the British government's practice of not setting lead targets until they had already been met.

Throughout the 1980s evidence that lead exerted adverse neurotoxic effects on children at ever lower levels of exposure continued to emerge, especially in the US, Greece and Australia. In the UK an influential pressure group, CLEAR, pressed the government to ensure that the use of lead as a petrol additive was ended. The response of the British authorities to those pressures was the classic tactic of establishing yet another investigative committee, this time under the auspices of the Medical Research Council (MRC). The question posed by the UK government to the MRC panel was: 'does the evidence on childhood neurotoxicity **prove** that levels of lead in British children are doing them obvious harm?' Implicitly, it set a particularly high evidential bar: indicative evidence short of proof would be insufficient. It did not ask: in which physiological system(s), and at which lowest level of exposure, are adverse effects detectable? If it had asked a question of that sort, a rather different answer would have been obtained. The government proposed only to act in the face of compelling evidence rather than, for example, the balance of probabilities.

Eventually, the MRC committee produced two reports. The first one sat resolutely on the fence; it just listed several of the important studies, emphasised their methodological limitations and suggested that if lead was having an adverse neurological effect on British children, the effect was a small one (MRC, 1984).

By 1988, several further studies had emerged, and the second report (MRC, 1988) focused on those recent studies. The committee emphasised many of the methodological limitations of the studies but acknowledged that in the intervening four years the evidence had strengthened. It accepted that 'low level lead exposure has a small negative effect on the performance of children in ability and attainment tests', and so concluded that 'it would be prudent to continue to reduce the environmental lead to which children are exposed.' That final remark was an acknowledgement that the levels of lead to which British children were then being exposed were unacceptably high, although it was couched in language designed not to provoke public anxiety.

In 1987 the United Kingdom eventually started to facilitate the increasing use of unleaded petrol after a preferential tax rate on unleaded fuel was introduced. That policy was adopted to facilitate the use of catalytic converters in motor vehicle exhaust systems rather than in response to evidence of lead's neurotoxicity. Curiously, official efforts to monitor childhood blood lead levels in British children then came to an end, so detailed evidence indicating the beneficial effects of phasing out leaded petrol in the UK have been only fragmentarily documented. It remains difficult, moreover, to estimate the proportion of children in the United Kingdom with elevated blood lead levels.

When the preferential tax change was introduced in 1987, the UK was one of the last industrialised countries to embrace unleaded petrol. It has been difficult to establish the extent to which the slow pace of change could be attributed to the fact that one of the world's main producers of lead tetra-ethyl (Associated Octel) was located in the UK. Nonetheless, it is noteworthy that in the summer of 2010 two former senior executives of Octel were convicted of having bribed government officials in Indonesia and Iraq to continue allowing the use of tetra ethyl lead as a fuel additive in those countries (Leigh et al., 2010).

Of course, leaded petrol also brought many benefits. It improved the energy and fuel efficiency of cars and other vehicles, provided thousands of jobs and generated much profit for the lead, oil and car industries of America, Europe and elsewhere. These benefits could, however, have been attained by alternative uses of the economic capital involved. Indeed, a 10-year phase out of leaded petrol at any time since 1925 would have encouraged innovators to develop less hazardous and perhaps more efficient fuel additives and engine designs. Since the early 1900s, such innovations have been widely recognised as a useful defence against high oil prices and insecure oil supplies.

3.11 European reflections on phasing out leaded petrol

Campaigns to take lead out of petrol in other countries went through similar phases and arguments. Panels 3.2, 3.3 and 3.4 provide European reflections on lead in petrol, focusing on the EU, Germany and the United Kingdom.

In Europe the legacy of leaded petrol and other sources of lead, such as old mines and lead shot, that can contaminate the food chain via soils and water still pose a threat to the neurodevelopmental health of some children in Europe (EFSA, 2010), as well as to wildlife (Mateo et al., 2007; Rodriguez-Estival et al., 2012).

Meanwhile, lead in electronic waste is an emerging hazard for children in poor countries in Asia and Africa, where waste from rich countries is dumped (Box 3.12).

3.12 Some late but contemporary lessons

The lessons from the story of leaded petrol are divided into two groups: lessons from the science and lessons from the influence of society on the science. In addition there are some lessons concerning some of the main arguments about the epidemiology that are relevant to many current controversies and which are therefore discussed in Chapter 26 on science for precautionary decision-making.

3.12.1 Some general lessons from the science

1. Much of the early evidence on lead poisoning came from the high exposures of fit, adult, usually male workers. Such findings were widely seen as irrelevant to the much lower exposures of the public to lead in petrol. However, the public can be more vulnerable to low doses of poisons because of sub-groups who are more sensitive to toxicants than workers, such as children, infants, foetuses, the elderly, the sick, pregnant women and the immuno-compromised. In addition, the public are often exposed for up to 24 hours a day and from multiple sources of the same poison via several routes e.g. ingestion and skin absorption from food, water, dust and consumer products, as well as via the inhalation of polluted air. Great care must therefore be taken in assuming that evidence from highly exposed occupational groups, or from low exposures to average populations, is not relevant to sensitive public groups.

2. Much reliance was initially placed on evidence from mortality, or from short-term (acute) poisoning. This can be a poor guide to

Box 3.12 Lead in electronic waste: an emerging hazard

With the global proliferation of computers, cellular telephones and other electronic equipment — as well as rapid cycles of replacement and obsolescence of these instruments — an enormous amount of electronic waste is now generated each year worldwide. Much of this waste — or electronic material near the end of its useful life — is shipped to low-income countries where large numbers of workers in both the formal and informal sectors separate lead, mercury and other metals from the waste for recovery and recycling. In the informal sector, much of the work is performed by children. Elevated lead levels in dust and blood have been reported in the communities and the children performing this work (Xia Huo et al., 2007). In 2004, 16 % of all children worldwide were estimated to have levels above 10 µg/dl (WHO, 2010).

In 2010 the European Food Standards Agency withdrew its support for a provisional tolerable weekly intake guideline value on the grounds that it was inadequate to protect against IQ loss (EFSA, 2010).

long-term (chronic) effects on morbidity such as neurological or reproductive damage.

3. Key assumptions that are critical to outcomes of harm or its absence were confidently asserted rather than demonstrated. For example, the initial assumption from the lead industry, in reply to the US Surgeon General's query about possible health hazards from leaded petrol, was to state that there were none, 'although no actual experimental data has been taken'. This was an early example of assuming that 'no evidence of harm' is the same as 'evidence of no harm' when no relevant research is available to support that assumption. This is a still a common mistake in public health.

4. Another key assumption was that the intake of lead into the body was counteracted by excretion, which was sufficient to achieve a harmless physiological balance, whereby no, or only minimal accumulation of lead in the body would take place. This assumption was not supported by actual evidence of the absence of lead accumulation.

5. Early studies of workers and Mexican farmers did not serve as unexposed control groups as they too were contaminated with lead. When 'unexposed' control groups are also contaminated then true risks will be underestimated.

6. The first study of consumer risks from lead in petrol was too small and short term to detect effects other than acute and gross ones and it was not followed up by the publicly funded longterm monitoring that its authors strongly recommended.

7. Experimental studies in animals documented adverse effects of lead from environmentally relevant concentrations but this evidence was frequently ignored or regarded as irrelevant for humans.

8. Extensive scientific debates, sometimes focusing on diversionary details, or based on the potential for exploiting or even manufacturing scientific doubt helped to maintain the impression that the adverse health effects of environmental lead pollution were unproven. It is often more convenient for a hazardous industry to debate the science than to discuss options for reducing hazards.

9. It was assumed that there was a threshold between biological effects and 'adverse' effects. This is still a dominant assumption in conventional toxicology despite the accumulating evidence that biological effects can be critical steps on the way to adverse effects, as Patterson pointed out in the 1960s. There is usually a biological continuum and not a discrete change. This means that action to avoid significant biological 'effects' will often be needed if we are to prevent, as opposed to merely observe, 'adverse effects'.

3.12.2 The influence of society on science

1. For several decades after the introduction of leaded petrol in the 1920s, virtually no independent research was carried out, and the main source of information was industry and industry-sponsored researchers. It took more independent scientists from outside this group, such as Patterson in the 1960s and Needleman in the 1970s to show, for example, that 'typical' body burdens of lead arising from human activities were not 'normal', as industry claimed, but were hundreds of times higher than before the industrial revolution, and were therefore likely to be harmful, especially to the brains of children.

2. There is a need for sufficient incentives and funds for independent long-term prospective monitoring of potential health hazards when new technologies are introduced.

3. The established and specific technical and economic benefits from leaded petrol, which largely accrued to particular and powerful minorities, were contrasted to the unproven, more general and future health threats to the public. This was an unequal contest, which even influenced many public health specialists, who allowed their appreciation of 'the gift from God', as the car industry described leaded petrol, to override their scientific concerns about health effects.

4. Public health is well served when scientists who discover hazards, especially when funded by the public, play an active role in disseminating both their results and their implications for precautionary or preventive action. Alice Hamilton, Yandell Henderson, Craig Patterson and Herbert Needleman played this role in the US leaded petrol story.

5. Each wave of 'early warning' scientists in the leaded patrol saga, from Yandall Henderson in the 1920s, to Byers in the 1940s, Patterson in the

1960s and Needleman in the 1980s, had either their funding withdrawn, their jobs threatened or their characters assassinated. They share such experiences with other 'early warning' scientists. Such scientists need more support from society via recognition for their work, help with their defence and legal protection against discrimination. This issue is picked up in Chapter 24 on protecting early warners and late victims.

6. The concrete record of decision-making by industries, scientists and governments need to be made publicly available if history is to stand a reasonable chance of being understood and providing relevant lessons for the future. This usually only occurs many years after the relevant events and then only via legal cases for compensation.

Table 3.1 Early warnings and actions

Year	Event
2nd century BC	First published record of occupational lead poisoning by Nicander.
1695	The count of Württemberg bans lead addition to wine based on Eberhard Gockel's study of lead poisoning in the city of Ulm.
1892	First report of poisoning cases in children from old lead paint.
1920	Leaded paint is banned in Australia and later in Europe.
1921	The octane-boosting property of tetraethyl (TEL) lead is discovered.
1921–1923	Ethanol-based alternative additives are considered by Du Pont and GM but rejected as less profitable than TEL, which goes into production.
1923–1924	Deaths of TEL workers lead to its temporary suspension.
1925	The 'one day trial' of TEL leads to its approval by an expert committee but only under careful monitoring and regulations, which do not take place.
1930–1960s	Kehoe and the TEL industry dominate the research field for next 50–60 years asserting that widespread human lead exposures are 'natural' and therefore safe — and that only acute, clinical effects are serious.
1943	Byers and Lord report chronic brain damage and anti-social behaviour in lead-poisoned children
1965	Patterson reports that current lead exposures are 100 times higher than natural levels and dismisses Kehoes' argument that 'normal' is 'natural'.
1966	Senator Muskie and US Congress start asking questions about leaded petrol and Patterson asserts the likelihood of no safe threshold with a continuum between effects and adverse effects.
1970	US Clean Air Act comes into force. GM announces the phase out of leaded petrol as it poisons the catalytic converters needed to secure the Act's targets for NO_x ,SO_2 and other air pollutants.
1971	UK and Germany begin to reduce permitted levels of lead in petrol.
1973	The US EPA introduces regulations to reduce lead in petrol but is opposed in the courts by industry.
1976	The EPA wins the court case on appeal.
1977	The EPA recognises the existence of subclinical lead poisoning due to environmental lead exposure.
1979	Needleman and colleagues report dose-related mental deficits in children with background lead exposures.
1983	A European Commission study with lead isotopes in northern Italy demonstrates that petrol additives cause substantial human exposures.
1984	Germany introduces low-lead petrol and other countries follow.
1985	A European Commission directive requires Member States to make unleaded petrol available and lowers the limits of lead permissible in petrol.
1995–2000	Virtually all western Europe only uses lead-free petrol.
2013	Nearly all countries worldwide have phased out leaded petrol. Legacy lead persists in water and soils threatening the neurodevelopmental health of some children.

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