

BSEM REPORT

The health effects of waste incinerators

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Abstract

In the UK, incinerators are still seen as a satisfactory answer to the problem of getting rid of the increasing quantities of waste, including increasing amounts of synthetics. This report examines the scientific evidence from all sources concerning the health implications of waste incineration, and its costs, explicit and hidden.

The report reviews what is known about the range of pollutants released by incinerators and their health effects. The major air pollutants are fine airborne particulates (2.5 µm diameter and smaller), toxic metals, and organic chemicals. The precise content of the emissions varies with the material incinerated: emitted chemicals include persistent organic pollutants, hormone disrupters, and carcinogens, but not all the organic components have been identified. In addition, the ash produced includes large quantities of highly toxic fine fly ash (air pollution control residues), which pose important long-term health risks.

Dangers from chemicals have been overlooked in the past: chemicals such as dichlorodiphenyltrichloroethane (DDT) and chlorofluorocarbons (CFCs) were regarded as safe on introduction, but were banned many years later because of widespread ill-effects. Incineration is a source of emission of heavy metals and these have a range of harmful health effects. In the last 10 years, the health dangers of another major incinerator emission, fine particulates, have become widely recognized. These are associated with an increased incidence of lung cancer, but also with a linear increase in mortality (with no safe level), particularly from cardiovascular causes. Fine particulates are inspired deep into the lungs and carry other toxins, adsorbed to them, into the blood stream.

Increased adult lung cancer and all cancers have been found in the vicinity of incinerators: the peak seems to occur at least 14 years after incinerator start-up. There have been no direct studies of the incidence of cardiac illnesses around incinerators, but as incinerators are a major source of fine particulates, and ischaemic heart disease is a relatively common cause of death, substantial excess cardiac mortality and morbidity would be predicted. The foetus and infant are particularly susceptible to damage from toxins and carcinogens, and there are indications that some effects may be passed to the next generation. Increased birth defects and an increased incidence of childhood cancers have been demonstrated around incinerators.

Health costs should always be considered in determining strategies for waste disposal. Other methods are available that are safer and cheaper in the long term and far cheaper if the high health costs of incineration are taken into consideration. We recommend that these more modern methods should be used, and that a more stringent and independent monitoring system should be introduced. In our view, incinerators, with their high risks and high health costs, are a poor choice of technology for waste disposal: more modern and safer technologies should be used in the future. Tackling the problems of both the amount and the nature of waste generated is also of critical importance.

1. Introduction

Both the amount of waste and its potential toxicity are increasing. Available landfill sites are being used up and incineration is increasingly being seen as a solution to the waste problem. This report examines the literature concerning the health effects of incinerators.

Incinerators produce pollution in two ways. First, they discharge hundreds of pollutants into the atmosphere. Although some attention has been paid to the concentrations of the major chemicals emitted in an effort to avoid acute local toxic effects, this is only part of the problem. Many of these chemicals are both toxic and bioaccumulative, building up over time in the human body in an insidious manner with the risk of chronic effects at much lower exposures. Little is known about the risks of many of these pollutants, particularly when combined. In addition, incinerators convert some of the waste into ash and some of this ash will contain high concentrations of toxic substances such as dioxins and heavy metals, creating a major pollution problem for future generations. Pollutants from landfill have already been shown to seep down and pollute water sources. It is also important to note that incineration does not solve the landfill problem because of the large volumes of ash that are produced.

There have been relatively few studies of populations exposed to incinerator emissions or of occupational exposure to incinerators (see Section 4), but most show higher-than-expected levels of cancer and birth defects in the local population and increased ischaemic heart disease has been reported in incinerator workers. These findings are disturbing, but taken alone, they might only serve to alert the scientific community to possible dangers but for two facts. The first is the acknowledged difficulty of establishing beyond question the chronic effects associated with any sort of environmental exposure. The second is the volume of evidence linking health effects with exposure to the individual combustion products known to be discharged by incinerators and other combustion processes.

The purpose of this report is to look at all the evidence and to come to a balanced view about the future dangers that would be associated with the next generation of waste incinerators. There are good reasons for undertaking this review. The history of science shows that it often takes decades to identify the health effects of toxic exposures but, with hindsight, early warning signs were often present that had gone unheeded. It is rare for the effects of environmental exposures to have been anticipated in advance. For instance, it was not anticipated that the older generation of incinerators in the UK would prove to be a major source of contamination of the food supply with dioxins. In assessing the evidence, we shall also look at data from a number of other areas that we believe to be relevant, including research on the increased vulnerability of the foetus to toxic exposures, and the risk of synergistic effects between chemicals, the higher risks to people more sensitive to chemical pollution, the difficulties of hazard assessment, the problems of monitoring and the health costs of incineration.

2. Emissions from incinerators and other combustion sources

The exact composition of emissions from incinerators will vary with what waste is being burnt at any given time, the efficiency of the installation and the pollution control measures in place. There is little detailed evidence available. A municipal waste incinerator will take in a great variety of waste contaminated by heavy metals and man-made organic chemicals. During incineration, more toxic forms of some of these substances can be created. The

three most important constituents of the emissions, in terms of health effects, are particulates, heavy metals and combustion products of man-made chemicals; the latter two can be adsorbed onto the smaller particulates making them especially hazardous. The wide range of chemicals known to be products of combustion include sulphur dioxide, oxides of nitrogen, over a hundred volatile organic compounds (VOCs), dioxins, polyaromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs) and furans.

2.1. *Particulates*

Particulates are tiny particles in the air that are classified by size. PM_{10} s have a diameter of less than $10\ \mu\text{m}$, whereas fine particulates ($PM_{2.5}$ s) are less than $2.5\ \mu\text{m}$ and ultrafine particulates (PM_{1} s) are less than $1\ \mu\text{m}$. Incinerators produce huge quantities of fine and ultrafine particulates. Incinerators are permitted to emit particulates at a rate of $10\ \text{mg}/\text{m}^3$ of gaseous discharge. The commonly used baghouse filters act like a sieve, effectively allowing the smallest particulates to get through and blocking the less dangerous, larger particulates. Only 5–30% of the $PM_{2.5}$ s will be removed by these filters and virtually none of the PM_{1} s. In fact, the majority of particles emitted by incinerators are the most dangerous ultrafine particulates [1]. The baghouse filters are the least effective at removing the smallest particles, especially those of $0.2\text{--}0.3\ \mu\text{m}$, and these will have a considerable health impact. Health effects are determined by the number and size of particles and not the weight. Measurements of the particle size distribution by weight will give a false impression of safety due to the higher weight of the larger particulates. Pollution abatement equipment, installed to reduce emissions of nitrogen oxides, may actually increase emissions of the $PM_{2.5}$ particulates [2]. The ammonia used in this process reacts with sulphurous acid formed when steam and sulphur dioxide combine as they travel up the stack, leading to the production of secondary particulates. These secondary particulates are formed beyond the filters and emitted unabated: they can easily double the total volume of particulates emitted (P. Rossington, pers. comm.). Present modelling methods do not take secondary particulates into account (see Section 12).

Studies have shown that toxic metals accumulate on the smallest particulates [3] and that 95% of PAHs are associated with fine particulates (PM_3 and below) [4–6]. PAHs are toxic and carcinogenic, and it has been estimated that these increase the lung cancer risk by 7.8 times [7].

2.2. *Heavy metals*

Incinerators are allowed to emit $10\ \text{mg}/\text{m}^3$ of particulates and $1\ \text{mg}/\text{m}^3$ of metals. The limits mean little as, even within these limits, the total amount of particulates and metals emitted will vary with the volume per second of emissions generated by the incinerator and this can vary hugely. A further concern is that there are no statutory ambient air quality standards for heavy metals apart from lead, which means the levels of heavy metals in the surrounding air do not need to be monitored.

The proportion of metals to particulates allowed to be emitted by incinerators is very high and much higher than found in emissions from cars. At the high temperatures found in incinerators, metals are released from metallic waste, plastics and many other substances. Many of the heavy metals emitted, such as cadmium, are toxic at very low concentrations. The selective attachment of heavy metals to the smallest particulates emitted from incinerators [3] increases the toxicity of these particulates. This fact is likely to make the particulates from incinerators more dangerous than particulates from other sources such as cars.

2.3. Nitrogen oxides

The removal of nitric oxide by incinerators is only about 60% effective and the nitric oxide is then converted to nitrogen dioxide to form smog and acid rain. Sunlight acts on nitrous oxides and VOCs to produce another pollutant, ozone.

2.4. Organic pollutants

A wide range of organic pollutants is emitted from incinerators. These include PAHs, PCBs, dioxins, furans, phthalates, ketones, aldehydes, organic acids and alkenes.

The waste being burned now differs considerably from that burned in the past, with a higher load of heavy metals and plastics producing far greater potential for health and environmental problems. An example of this is polyvinyl chloride (PVC), which is more than 90% organic chlorine. It has been used extensively for doors and windows and with an expected life of 40 years it is likely to appear in increasing quantities in the waste stream. This could easily raise the organic chlorine in the waste stream to over 1%, which according to the European Waste Directive would mean the waste would be regarded as hazardous.

Many of the compounds are known to be not only toxic but bioaccumulative and persistent. They include compounds that have been reported to affect the immune system [8], attach to chromosomes [9], disrupt hormone regulation [10], trigger cancer [11], alter behaviour [12], and lower intelligence [13]. The very limited toxicity data on many of these substances is a matter of concern [14]. The changing nature of waste means that new substances are likely to be emitted and created. For example, polybrominated diphenyl ethers (PBDEs) are found in many electrical goods and are increasingly finding their way into incinerator waste. They have been found to affect brain development and affect the thyroid gland and cause behavioural and learning defects in animals [15,16].

3. Health effects of pollutants

3.1. Particulates

A large and growing body of literature has highlighted the dangers of particulates to health. Various studies have confirmed that the smaller the size of the particles the more dangerous the health effects [17–20]. The data from the World Health Organization (WHO) shown in Figure 1 clearly illustrate that $PM_{2.5}$ particles have a greater effect on daily mortality than the larger PM_{10s} [17].

The smaller particles are not filtered out by the nose and bronchioles and their minuscule size allows them to be breathed deeply into the lungs and to be absorbed directly into the blood stream where they can persist for hours [21]. They can then travel through the cell walls and into the cell nucleus affecting the cell's DNA. The WHO state that there is no safe level of $PM_{2.5s}$ and health effects have been observed at surprisingly low concentrations with no threshold [22,23]. The smallest particulates, particularly the ultrafine particulates (PM_1), are highly chemically reactive, a property of their small size and large surface area [24]. A further danger of the smallest particulates is that there are thousands more of them per unit weight. In incinerators, heavy metals, dioxins and other chemicals can adhere to their surface [25], increasing their toxicity. The body does not have efficient mechanisms for clearing the deeper part of the lung, as only a tiny fraction of natural particles will be as small as this.

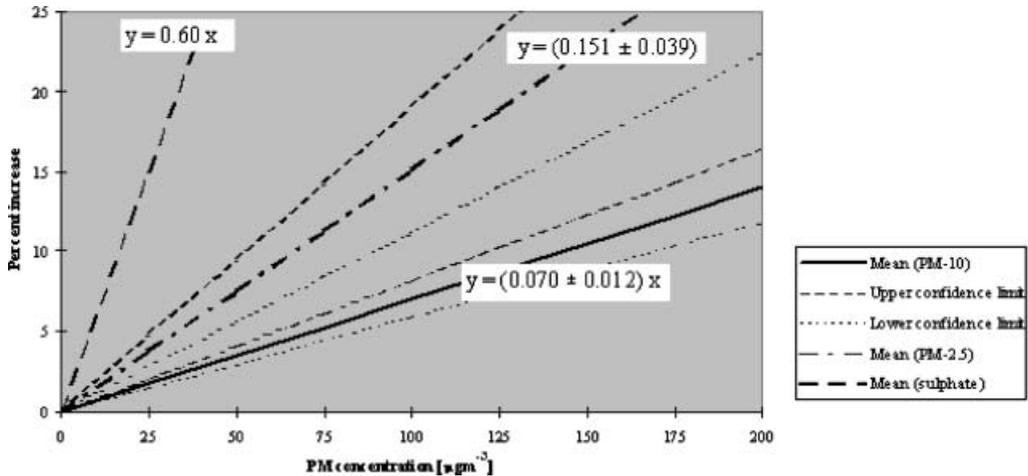


Figure 1. Increase in daily mortality as a function of PM concentration (reproduced from [17]).

As incinerators are effectively particulate generators and produce predominately the smaller particulates that have the largest effect on mortality, it is clear that incinerators have considerable lethal potential.

(a) *Epidemiological studies of particulate pollutants.* Fine particulates have been associated with both respiratory and cardiovascular disease [26] and with lung cancer [18,27].

Two large cohort studies in the USA showed increasing mortality with increasing levels of $PM_{2.5}$ pollution. In the Six Cities Study, published in 1993 [18], 8111 individuals were followed for 14–16 years (1974–1991), involving a total of 111,076 person years, to examine the effect of air pollution, allowing for smoking and other individual factors. As expected, the greatest risk factor was smoking (adjusted mortality rate ratio 1.59), but, after allowing for individual factors, mortality rates showed highly significant associations ($p < 0.005$) with the levels of fine particles and sulphate particles in the cities, with the most polluted city giving an adjusted all-cause mortality rate of 1.26 compared with the least. This related to a $PM_{2.5}$ difference of $18.6 \mu\text{g}/\text{m}^3$: cardiopulmonary mortality was increased by 37% and lung cancer mortality was also 37% higher.

In the American Cancer Society study [19], 552,138 adults (drawn from the Cancer Prevention II study) were followed from 1982 to 1989 and deaths analysed against mean concentrations of sulphate air pollution in 1980 and the median fine particulate concentration from 1979–1983, both obtained for each participant's area of residence from Environmental Protection Agency (EPA) data. Again, the strongest correlation was between lung cancer and smoking (adjusted mortality risk ratio 9.73), but both pollution measures showed highly significant association with all-cause mortality and with cardiopulmonary mortality: sulphates were also associated with lung cancer. After adjusting for smoking and other variables, higher fine particulate pollution was associated with a 17% increase in all-cause mortality and a 31% increase in cardiopulmonary mortality for a $24.5 \mu\text{g}/\text{m}^3$ difference in $PM_{2.5}$ s. These results are highly significant and led the EPA to place regulatory limits on $PM_{2.5}$ s, establishing the National Ambient Air Quality Standards in 1997. These regulations were challenged by industry but ultimately upheld by the US Supreme Court [28] after the data from all the studies had been subjected to intense

scrutiny, including an extensive independent audit and a re-analysis of the original data [29].

The health benefits of bringing in these new regulations have been estimated as \$32 billion annually [30] based on mortality and chronic and acute health effects, and a White House report from the Office of Management and Budget in September 2003 calculated the benefits in terms of reductions in hospitalizations, premature deaths and lost working days as between \$120 and 193 billion over the last 10 years (see Section 9.1). As this study looked at only three health indicators, the true benefits have probably been underestimated.

It follows from these data that incinerators and all other major sources of $PM_{2.5}$ particulates will generate substantial health costs as well as increasing mortality.

(b) *Further studies.* An analysis published in 2002 of the Cancer Prevention II study participants linked the individual factors, pollution exposures and mortality data for approximately 500,000 adults as reported in the ACS study above, bringing the follow-up to 1998 [27]. The report doubled the follow-up period and reported triple the number of deaths, a wider range of individual factors and more pollution data, concentrating on fine particles. Smoking remained the strongest factor associated with mortality, but fine particulate pollution remained significantly associated with all-cause and cardiopulmonary mortality, with average adjusted relative risks of 1.06 and 1.09. In addition, after the longer follow-up period, fine particulates were significantly associated with lung cancer mortality, with an adjusted relative risk of 1.14. The authors reported that exposure to a $10\ \mu\text{g}/\text{m}^3$ higher level of $PM_{2.5}$ was associated with a 14% increase in lung cancer and a 9% increase in cardiopulmonary disease [27].

(c) *Cardiovascular disease.* Researchers were surprised to find that the increased cardiopulmonary mortality associated with particulate pollution was primarily due to cardiovascular disease. This was found in both the Six City and ACS studies when they were re-analysed [29]. When the causes of death in the Cancer Prevention II study were looked at in more detail [31] to look for clues to possible pathophysiological mechanisms, the link was strongest with ischaemic heart disease: a $10\ \mu\text{g}/\text{m}^3$ increase in $PM_{2.5}$ was associated with an 18% increase in deaths from ischaemic heart disease (22% in never smokers).

Acute myocardial infarction rose during episodes of high particulate pollution, doubling when levels of $PM_{2.5}$ were 20–25 $\mu\text{g}/\text{m}^3$ higher [32]. Particulates also increased mortality from stroke [33,34]. One study concluded that 11% of strokes could be attributed to outdoor air pollution [35]. Episodes of increased particulate pollution also increased admissions with heart disease [36]. Mortality from diabetes [26] and admissions for diabetic heart disease were also increased [37] and these were double the non-diabetic coronary heart disease admissions, suggesting that diabetics were particularly vulnerable to the effect of particulate pollution [37]. Higher levels of particulates have been associated with life-threatening arrhythmias [38], exercise-induced ischaemia [39], excess mortality from heart failure [34,40] and thrombotic disease [34].

(d) *Effect on children and the foetus.* Particulates carry various chemicals, including PAHs, into the human body. Frederica Perera from the Columbia Center for Children's Environmental Health has found that the foetus is 10 times more vulnerable to damage by these substances [41]. She also found that $PM_{2.5}$ particulates have an adverse effect on the developing foetus, with significant reductions in weight, length and head circumference,

and reiterated the importance of reducing ambient fine particulate concentrations [42]. In addition, further studies have shown an adverse effect on foetal development at levels currently found in cities today, such as New York [43]. Air pollution has been found to cause irreversible genetic mutations in mice. Researchers found, in contrast, that if mice breathed air that had been freed of particulates by filtration they developed only background levels of genetic mutations, confirming that particulates were causative [44]. At the fourth Ministerial Conference of Environment and Health in June 2004, the WHO announced that between 1.8 and 6.4% of deaths in the age group from 0 to 4 years could be attributed to air pollution [45].

(e) *Acute respiratory incidents.* Elevated particulate air pollution has been associated with increased hospital admissions with asthma [23] and chronic obstructive pulmonary disease (COPD) [46], increases in respiratory symptoms [47,48], higher incidence of asthma [49], reduced immunity [50,51], higher rates of ear, nose and throat infection [49], loss of time from school in children through respiratory disease [52,53], and declines in respiratory function [54–56]. A sad aside to the above is that children who did more outdoor sport had greater declines in respiratory function [56]. We are doing a great disservice to our children if they cannot pursue healthy activities without damaging their health.

(f) *Mortality from particulate pollution.* Episodes of increased particulate pollution have been associated with increased cardiovascular mortality [18,19,26,27,34,40,57] and increased respiratory mortality [40,41]. About 150 time-series studies around the world have shown transient increases in mortality with increases in particulates. Cohort studies have shown a long-term effect on mortality [18,19,27] (see Section 3.1a).

Can we quantify this mortality? It has been estimated that the increased mortality works out as about a 0.5–1% increase in mortality for each $10 \mu\text{g}/\text{m}^3$ rise in $\text{PM}_{10\text{s}}$ [58] for acute exposures and a 3.5% rise for chronic exposures [30]. For $\text{PM}_{2.5\text{s}}$, the increase in mortality is much greater, especially for cardiopulmonary mortality (see Table I).

When the data from the Six Cities Study and the ACS study were subject to audit and re-analysis (see Section 3.1a), the cardiopulmonary deaths were separated into pulmonary and cardiovascular [29]. Unexpectedly, most of the excess deaths due to particulates had been from cardiovascular causes. This was apparent in each of the analyses performed giving figures for the increase in cardiovascular mortality in the Six Cities Study of between 35 and 44% for an $18.6 \mu\text{g}/\text{m}^3$ difference in $\text{PM}_{2.5}$ and in the ACS study between 33 and 47% for $24.5 \mu\text{g}/\text{m}^3$. This was much higher in each case than the increase in respiratory deaths of 7%. In the ACS data it was later found that the excess cardiovascular deaths were

Table I. Cardiopulmonary mortality and fine particulate pollution.

Study	Reference/ year	No. of participants	Follow-up	Adjusted excess cardiopulmonary mortality (%)	Difference in $\text{PM}_{2.5\text{s}}$ in $\mu\text{g}/\text{m}^3$	Adjusted excess cardiopulmonary mortality for rise of $10 \mu\text{g}/\text{m}^3$ (%)
Six Cities	[18]/1993	8111	1974–1991	37	18.6	19.8
ACS Cancer Prevention II	[19]/1995	552,138	1982–1989	31	24.5	12.7
ACS Cancer Prevention II	[27]/2002	500,000	1982–1998	9	10	9

primarily due to an 18% increase in deaths from ischaemic heart disease for each $10 \mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5\text{s}}$ [31].

Incinerators selectively emit smaller particulates and cause a greater effect on the levels of $\text{PM}_{2.5\text{s}}$ than $\text{PM}_{10\text{s}}$ and would therefore be expected to have a large impact on cardiopulmonary mortality, especially cardiovascular mortality. This has not so far been studied directly.

(g) *Assessment by the WHO and other authorities.* Based on the WHO Air Quality Guidelines [59], we have estimated that a $1 \mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ particulates (a very conservative estimate of the level of increase that would be expected around incinerators) would lead to a reduced life expectancy of 40 days per person over 15 years (this equals a reduction of life expectancy of 1.1 years for each $10 \mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ particulates). Although this figure appears small, they note that the public health implications are large and the effect on a typical surrounding population of 250,000 would be a loss of 27,500 years of life over a 15 year time period. This figure gives an indication of the likely loss of life from any major source of $\text{PM}_{2.5}$ particulates. In addition, incinerators normally operate for much longer periods than the 15 years quoted here. Note that the estimated loss of life here is from particulates alone and not from other toxic substances.

Statements by leading researchers include the following: “the magnitude of the association between fine particles and mortality suggests that controlling fine particles would result in saving thousands of early deaths each year” [58] and “there is consistent evidence that fine particulates are associated with increased all cause, cardiac and respiratory mortality. These findings strengthen the case for controlling the levels of respiratory particulates in outdoor air” [57].

(h) *Summary.* There is now robust scientific evidence on the dangers to health of $\text{PM}_{2.5}$ particulates and of the substantial health costs involved. For these reasons it is impossible to justify increasing levels of these particulates still further by building incinerators or any other major source of $\text{PM}_{2.5}$ particulates. The data make it quite clear that attempts should be made to reduce the levels of these particulates whenever possible. However, $\text{PM}_{2.5\text{s}}$ are not the only reasons to be concerned about incinerators. There are other dangers.

3.2. *Heavy metals*

Pope [60,61] reported that hospital admissions of children with respiratory disease fell dramatically in the Utah valley when a steel mill was closed for a year due to a strike. An air pollution analysis showed that the metal content of particulates was lower that year and that the type of inflammation found in the lungs while the steel mill was working could be reproduced in both rat and human lung tissue by using air pollutants of the type emitted by the steel mill. This is a very clear illustration of the dangers of pollution of the air with heavy metals. Exposure to inhaled metals, similar to the type produced by incinerators, has been shown to mediate cardiopulmonary injury in rats [62] and small amounts of metal (<1%) in particulates are known to cause pulmonary toxicity [63].

Emissions and ash from incinerators contain over 35 metals [64]. Several are known or suspected carcinogens. Toxic metals accumulate in the body with increasing age [65]. Breathing in air containing toxic metals leads to bioaccumulation in the human body. They can remain in the body for years: cadmium has a 30 year half-life. Incineration adds to the burden of toxic metals and can lead to further damage to health.

Mercury is a gas at incineration temperatures and cannot be removed by the filters. Incinerators have been a major source of mercury release into the environment. In theory, mercury can be removed using activated carbon, but in practice it is difficult to control and even when effective the mercury ends up in the fly ash to be landfilled. Mercury is one of the most dangerous heavy metals. It is neurotoxic and has been implicated in Alzheimer's disease [66–68], learning disabilities and hyperactivity [69,70].

The inhalation of heavy metals such as nickel, beryllium, chromium, cadmium and arsenic increases the risk of lung cancer [11]. Cumulative exposure to cadmium has been correlated with lung cancer [71]. Supportive evidence comes from Blot and Fraumeni [72] who found an excess of lung cancer in US counties where there was smelting and refining of non-ferrous metals. Inhaled cadmium also correlates with ischaemic heart disease [73].

So what are the dangers caused by toxic metals accumulating in the body? They have been implicated in a range of emotional and behavioural problems in children, including autism [74], dyslexia [75], impulsive behaviour [76], attention deficit and hyperactivity disorder (ADHD) [77,78], as well as learning difficulties [13,69,79–82], lowered intelligence [78] and delinquency [78,83], although not every study reached standard significance levels. Many of these problems were noted in the study of the population around the Sint Niklaas incinerator [84]. Exposed adults have also been shown to be affected, showing higher levels of violence [12,85], dementia [86–92] and depression than non-exposed individuals. Heavy metal toxicity has also been implicated in Parkinson's disease [93].

Heavy metals emitted from incinerators are usually monitored at 3–12 monthly intervals in the stack: this is clearly inadequate for substances with this degree of toxicity.

3.3. Nitrogen oxides and ozone

Nitrogen dioxide is another pollutant produced by incinerators. It has caused a variety of effects, primarily in the lungs, but also in the spleen, liver and blood in animal studies. Both reversible and irreversible effects in the lungs have been noted. Children between the ages of 5 and 12 years have been estimated to have a 20% increase in respiratory symptoms for each $28 \mu\text{g}/\text{m}^3$ increase in nitrogen dioxide. Studies in Japan showed a higher incidence of asthma with increasing nitrogen dioxide levels and that it synergistically increases lung cancer mortality rates [40]. It has also been reported to aid the spread of tumours [94,95]. Increases in nitrogen dioxide have been associated with increases in admissions with COPD [96], asthma in children and in heart disease in those over 65 years [17]. Other studies have shown increases in asthma admissions [97] and increased mortality with rising nitrogen dioxide levels [98].

Rising ozone levels have led to increasing hospital admissions, asthma and respiratory inflammation and have been reported to lower immunity [99]. Higher levels have been significantly associated with increased mortality [100] and with cardiovascular disease. Both ozone and nitrogen dioxide are associated with increasing admissions with COPD [96].

When it comes to incinerator emissions, the health effects of nitrous oxides are likely to compound the negative health effects of particulates and metals.

3.4. Organic toxicants

Hundreds of chemical compounds are released from incinerators. They include a host of chemicals produced from the burning of plastic and similar substances and include PAHs,

brominated flame retardants, PCBs, dioxins, and furans. These substances are lipophilic and accumulate in fatty tissue and remain active in living organisms and the environment for many years. They have been linked with early puberty [101], endometriosis [102], breast cancer [103,104], reduced sperm counts [105] and other disorders of male reproductive tissues [106], testicular cancer [107] and thyroid disruption [10]. It has been claimed that about 10% of man-made chemicals are carcinogenic (see Section 5.1), and many are now recognized as endocrine disrupters. Most of these health effects were not anticipated and are only now being recognized. No safety data exist on many of the compounds released by incinerators.

PAHs are an example of organic toxicants. Although emission levels are small, these substances are toxic at parts per billion or even parts per trillion [64] as opposed to parts per million for many other pollutants. They can cause cancer, immune changes, lung and liver damage, retarded cognitive and motor development, lowered birth weight and lowered growth rate [64].

3.5. Effects on genetic material

Heavy metals and many chemicals form covalent bonds with DNA called DNA adducts. This can increase the risk of cancer by activating oncogenes and blocking anti-tumour genes. This raises a very serious concern. This concern is that by releasing chemicals into the environment we may not only be poisoning this generation but the next. Carcinogenesis from chemicals that can be passed on through several generations is not just a horrifying scenario but has been demonstrated to occur in animals [108,109]. Incinerator emissions would greatly increase this risk.

DNA adducts to PAHs increase with exposure to pollution and patients with lung cancer have higher levels of adducts (see below). This is one demonstration of how pollutants alter genes and predispose to cancer. Other chemicals, such as vinyl chloride, interfere with DNA repair and yet others, such as organochlorines, are tumour promoters.

3.6. Effects on the immune system

Starting in the late 1980s, a series of dramatic marine epidemics killed off thousands of dolphins, seals and porpoises. Many were found to have been affected by a distemper-like virus. Autopsies of the dead animals showed weakened immune systems and high levels of pollutants, including PCBs and synthetic chemicals. A virologist, Albert Osterhaus and his co-workers, demonstrated that when seals were fed contaminated fish containing organochlorines (which were, however, considered fit for human consumption) they developed immune suppression and were unable to fight viruses [110–112]. Their natural killer cells were 20–50% below normal and their T cell response dropped by 25–60%. The immune suppression was due to dioxin-like chemicals, PCBs and synthetic chemicals. An immunologist, Garet Lahvis, found that immunity in dolphins in the USA dropped as PCBs and dichlorodiphenyltrichloroethane (DDT) increased in their blood [113]. The immune system appeared the most vulnerable during prenatal development. This demonstrates that the immune system may be damaged by exposure to synthetic chemicals and that we have seriously underestimated the dangers of these chemicals.

Animal experiments have shown immunotoxicity with heavy metals, organochlorine pesticides and halogenated aromatics [114] and accidental exposure data on humans have

shown immunotoxicity with polybrominated biphenyls (PBBs), dioxins and aldicarb. In fact, whole volumes have been written on immunotoxicity [115]. Note that these are the type of pollutants released by incinerators. Environmental toxins have been shown to decrease T-lymphocyte helper-suppressor ratios in four different exposed populations [116]. Nitrogen dioxide exposure leads to abnormally elevated immune and allergic responses. PM_{2.5} particulates themselves can cause mutagenic and cytotoxic effects and the smallest particulates cause the greatest effects [117].

In summary, there is evidence that a large number of the pollutants emitted by incinerators can cause damage to the immune system [118]. As demonstrated in the following section, the combination of these is likely to have an even more potent and damaging effect on immunity than any pollutant in isolation.

3.7. Synergistic effects

Various studies have shown that a combination of substances can cause toxicity even when the individual chemicals are at a level normally considered safe. The report 'Man's impact on the global environment' by the Massachusetts Institute of Technology [119] stated "synergistic effects among chemical pollutants are more often present than not". Testing has been minimal and most of the synergistic effects are likely to remain unknown. Toxicologist Dr Vyvyan Howard has calculated that to test just the commonest 1000 toxic chemicals in unique combinations of three would require 166 million different experiments and even this would disregard varying doses [120].

Synergy has been demonstrated when organic chemicals are combined with heavy metals [121,122], and with combinations of pesticides [123,124] and food additives [125]. The last study is of particular concern. Rats fed with one additive were unharmed. Those fed two developed a variety of symptoms, whereas those fed all three all died within 2 weeks. In this case, the chemicals appeared to amplify each other's toxicity in a logarithmic manner. In a recent experiment, scientists dosed animals with a mixture of 16 organochlorine pesticides, lead and cadmium at 'safe levels' and found that they developed impaired immune responses, altered thyroid function and altered brain development [126]. Another study reported on the dangers of combinations of pesticides and their ability to mimic oestrogen. They found that combinations could increase the toxicity by 500–1000 times [127]. The level of concern about the multiplicity of pollutants released into the air by incinerators is enhanced by the fact that no one has any idea what damage these combinations of chemicals can cause.

The population living around an incinerator is being exposed to multiple chemical carcinogens, PM_{2.5s}, carcinogenic heavy metals (in particular cadmium) and in some cases radioactive particles, all known to increase lung cancer. Nitrogen dioxide has also been shown to synergistically increase lung cancer. When all these are combined, the effects are likely to be more potent, and, in fact, an increase in the incidence of lung cancer has been reported around incinerators (see Section 4.1).

The potential for multiple pollutants to cause serious health effects is illustrated by the results of a key study on rats exposed to the dust, soil and air from a landfill site. These animals developed abnormal changes in the liver, thyroid and reproductive organs within only 2 days of exposure [128]. Although effects in animals do not always mimic those in humans, the authors concluded that present methods of calculating health risks underestimate the biological effects. This has obvious relevance to the dangers of exposing people to multiple pollutants from incinerators.

4. Increased morbidity and mortality near incinerators

4.1. Cancer

There have been a number of studies on the effect of incinerators on the health of the surrounding population, mainly concentrating on cancer incidence. In most studies the incinerators were situated near other sources of pollution and often in areas of deprivation, both likely to confound the findings as both are associated with higher cancer incidence. The study of an incinerator burning 55,000 tonnes of waste a year and built in 1977 in the middle of a residential area of a town of 140,000 with no heavy industry (Sint Niklaas) is scientifically unsatisfactory because funds were not made available for the study of controls [84]. However, the investigators mapped a convincing cluster of 38 cancer deaths immediately surrounding and to leeward of the incinerator, and this area also showed high concentrations of dioxin in soil samples when tested in 1992. They noted that the cancer standardised mortality rate (SMR) for this town for 1994–1996 (national statistics) was high (112.08 for males and 105.32 for females), supporting the genuine nature of their findings.

In 1996, Elliott et al. [129] published a major study in which they compared the numbers of registered cancer cases within 3 km and 7.5 km of the 72 municipal waste incinerator sites in the UK with the number of cases expected. It involved data on over 14 million people for up to 13 years. Expected numbers were calculated from national registrations, adjusted for unemployment, overcrowding and social class. No account was taken of prevailing winds, or of differences between incinerators. They first studied a sample of 20 of the incinerator sites, replicating the analysis later with the other 52. If the results of two sets like this concur, it strengthens the data. In each set there was an excess of all cancers near the incinerators, and excesses separately of stomach, colorectal, liver and lung cancers, but not leukaemias. The first set gave adjusted mortality ratios for all cancers of 1.08 for within 3 km and 1.05 within 7.5 km; for the second these were 1.04 and 1.02. These risks, representing an additional risk of 8 and 5% for the first set and 4 and 2% for the second, seem small but represented a total of over 11,000 extra cancer deaths near incinerators and were highly significant ($p < 0.001$ for each).

For each of the main cancer sites the excesses were higher for those living within 3 km than for all within 7.5 km [129,130], suggesting that the incinerators had caused the excess. The authors doubted this and attributed the findings to additional confounding in spite of the fact that they had already adjusted (possibly over-adjusted) for unemployment, overcrowding and social class, which give a partial correction for pollution. Moreover, the effect on people living leeward of the incinerator would be substantially higher than shown by this study, as the true number of people affected was diluted by those living at the same distance but away from the wind plume coming from the incinerator.

Knox et al. [131] looked at the data from 22,458 children who died of cancer between 1953 and 1980 in the UK. For each child they compared the distance of the birth and death addresses from the nearest source of pollution and found a consistent asymmetry: more had moved away from the nearest hazard than towards it [131]. They deduced that the excess of migrations away from the hazard (after allowing for social factors) was evidence that the children had been affected by the cancer-causing pollution before or shortly after birth.

Later they applied the method to the set of incinerators studied by Elliott et al. and again showed the same asymmetry in the children's birth and death addresses, indicating that the incinerators had posed a cancer risk to children [132]. Of the 9224 children for whom they had found accurate birth and death addresses, 4385 children had moved at least 0.1 km.

Significantly more children had migrated away from incinerators than towards. For all those who had at least one address within 3 km of an incinerator, the ratio was 1.27. When they limited the analysis to children with one address inside a 5 km radius from the nearest incinerator and the other address outside this radius the ratio was 2.01; this indicated a doubling of cancer risk. Both these findings were highly significant ($p < 0.001$ for each). The excess had only occurred during the operational period of each incinerator and was also noted around hospital incinerators but not landfill sites. This is strong evidence that the incinerators' emissions contributed to the children's cancer deaths.

Biggeri et al. [133] compared 755 lung cancer deaths in Trieste with controls in relation to smoking, probable occupational exposure to carcinogens and air pollution (measured nearest to their homes) and the distance of their home from each of four pollution sites. The city centre carried a risk of lung cancer but the strongest correlation was with the incinerator, where they found a 6.7 excess of lung cancer after allowing for individual risk factors.

Using a spatial scan statistic, Viel et al. [134] looked at the incidence of soft tissue sarcoma and non-Hodgkin's lymphoma from French Cancer Registry data, in two areas close to an incinerator with a high emission of dioxin. They found highly significant clusters of soft tissue sarcoma (relative risk 1.44) and non-Hodgkin's lymphoma (relative risk 1.27) but no clusters of Hodgkin's disease (used as the negative control). This study was interesting in that it was designed to look both in a focussed way at the area around the incinerator, and to check the association by looking for space-time relationships that should be present if the relationship was causal. In addition they looked in an unfocussed way for other clusters in the wider area that contained other areas of deprivation. Both the first two analyses were positive close to the incinerator, demonstrating that a causal relationship was likely. As no other clusters were found they concluded that deprivation could be virtually excluded as a factor.

According to Ohta et al. [135] Japan built 73% of all the municipal waste incinerators in the world and by 1997 had become very concerned about their health effects. In the village of Shintone, 42% of all deaths between 1985 and 1995 in the area up to 1.2 km leeward of an incinerator (built in 1971) were due to cancer, compared with 20% further away and 25% overall in the local prefecture. Their data on soil contamination reinforced the importance of considering wind direction in evaluating the health effects of incinerators.

In 1989, Gustavsson [136] reported a two-fold increase in lung cancer in incinerator workers in Sweden compared with the expected local rate. In 1993 he reported a 1.5-fold increase in oesophageal cancer in combustion workers, including those working in incinerators [137].

4.2. Birth defects

There have been five reports of increases in congenital abnormalities around incinerators. The investigators at Sint Niklaas noted multiple birth defects leeward of the incinerator [84]. Orofacial defects and other midline defects were found to be more than doubled near an incinerator in Zeeburg, Amsterdam [138]. Most of these deformed babies were born in an area corresponding to wind-flow from the incinerator and other defects included hypospadias and spina bifida. In the Neerland area, Belgium, there was a 26% increase in congenital anomalies in an area situated between two incinerators [139]. A study of incinerators in France has shown chromosomal defects and other major anomalies (facial clefts, megacolon, renal dysplasias) [140]. A recent British study looked at births in Cumbria between 1956 and 1993 and reported significantly increased lethal birth defects

around incinerators after adjusting for year of birth, social class, birth order, and multiple births. The odds ratio for spina bifida was 1.17 and that for heart defects 1.12. There was also an increased risk of stillbirth and anencephalus around crematoriums [141]. The study pointed out that the figures for birth defects are probably substantial underestimates as they do not include spontaneous or therapeutic abortions, both increased by foetal anomalies.

In addition, several studies have noted an increase in birth defects near waste sites, particularly hazardous waste sites. The pattern of abnormalities was similar to the pattern found with incinerators, with neural tube defects often being the most frequent abnormality found with cardiac defects second [142–145]. Harmful chemicals are normally stored in fatty tissue: in the foetus there is little or no fatty tissue except for the brain and nervous system, which may explain the pattern of damage. A review of this subject stated “the weight of evidence points to an association between residential proximity to hazardous waste site and adverse reproductive outcomes” [146].

4.3. *Ischaemic heart disease*

Gustavsson [136] found an excess of ischaemic heart disease in incinerator workers who had been exposed for longer. We have not found any epidemiological studies of cardiovascular disease in the neighbourhood of incinerators, but in view of the research on particulates (see Section 3.1), this should be investigated.

4.4. *Comment*

The authors of some of these reports did not consider that they had sufficient grounds for concluding that the health effects around incinerators were caused by pollution from the incinerators. However, statistically their findings were highly significant and, taking the studies together, it is difficult to believe that all their results could have been due to unrecognized confounding variables. This is even less likely when you consider the nature of the pollutants released from incinerators and the scientific evidence for the health effects of those compounds (see Sections 2 and 3). The concordance of increased cancer incidence in local areas demonstrated to be more polluted also points to a causal association, although it does not necessarily imply that the pollutant measured contributed to the increase.

The studies may have underestimated the risks. At 13 years, the follow-up period of the large British study was probably too short: at Sint Niklaas, adult cancer cases seemed to increase from 13 years onwards (although children’s cancers occurred earlier), and in Japan, Ohta et al. [135] noted that cancer caused 42% of all deaths in the lee of incinerators from 14 to 24 years after the incinerator was commissioned. The reported risks were higher in the studies in which allowance was made for the direction of prevailing winds, possibly because of dilution elsewhere by relatively unexposed persons.

The studies reviewed apply to the older incinerators: newer incinerators may have better filters but fine particulates and metals are incompletely removed. Because some of these pollutants, notably fine particulates, do not appear to have a safe threshold, it is clearly incorrect to claim that incinerators are safe. The higher quantity of toxic fly ash produced by modern incinerators, which is easily wind-borne, represents an additional hazard. Even if incinerators were equipped with perfect filters, their huge size and tendency to faults mean that the risk of intermittent high levels of pollution is a real concern.

Taking into account these results and the difficulty in identifying causes of cancers and other chronic diseases, it is a matter of considerable concern that incinerators have been

introduced without a comprehensive system to study their health effects, and that further incinerators are being planned without comprehensive monitoring either of emissions or the health of the local population.

5. Disease incidence and pollution

5.1. Cancer

Studies linking cancer with incinerators cannot be seen in isolation. It is important to obtain an overall picture and to look at other studies that link pollutants with cancer. And there is another aspect to this. Many types of cancer, including lung, pancreatic and stomach cancer, have a very poor prognosis and our only hope lies in prevention. Prevention means reducing our exposure to carcinogenic substances and we should take every opportunity to do this.

Cancer has shown an unrelenting rise over the last century, and is affecting younger people. The rise has been gradual, steady and real. Cancer incidence has been increasing by 1% per annum, with an age standardized increase in mortality of 43% between 1950 and 1988 [147]. Put another way, the chance of dying from cancer at the turn of the twentieth century was 1 in 33. It is now 1 in 4. WHO data have demonstrated that 80% of cancers are due to environmental influences [148] and evidence from migrant studies confirm that it is mainly the environment rather than genes that determine the cancer risk [148].

Many people have noted that the rise in cancer has paralleled the rise in synthetic chemicals. These chemicals have doubled in quantity every 7–8 years, with a 100-fold increase over the last two generations [149]. Many converging pieces of evidence link chemicals to the relentless rise of cancer.

(a) *Links between exposure to pollutants and cancer in man.*

- Cancer is commonest in industrialized countries, with 50% of cases in the industrialized 20% of the world [150] and the WHO has noted that cancer incidence rises with the gross national product of a country.
- There is the same correlation within countries. The highest mortality from cancer in the USA is in areas of highest industrialized activity. There is also a correlation in the USA between cancer incidence and the number of waste sites in the county [151,152]. Counties with facilities for treating toxic waste have four times as much breast cancer [153]. Cancer is also more common in counties with chemical industries [154]. Public Data Access in the USA shows a close correlation between cancer mortality and environmental contamination [155].
- Numerous studies have shown higher cancer incidence in both industrial workers and in populations living in polluted areas [156,157].
- One of the three most rapidly rising cancers, non-Hodgkin's lymphoma, has been clearly linked with exposure to certain chemicals (for instance, phenoxyherbicides and chlorophenols) [158,159].

(b) *Links between exposure to pollutants and cancer in animals.* Three decades of studies of cancers in wildlife have shown that they are intimately associated with environmental contamination. This is particularly important as animals do not smoke, drink or eat junk food and cannot be accused of living in deprived areas. This strengthens the long-suspected

link between environmental pollution and cancer. In a recent study of outbreaks of liver cancer in 16 different species of fish at 25 different sites, cancers were always associated with environmental contamination [160]. Dogs have been found to have higher rates of bladder cancer in industrialized countries in the USA [161]. It is inconceivable that we are not affected in the same way. Furthermore, cancer rates in animals rapidly decline when the pollutants are removed, showing the critical importance of an uncontaminated environment for good health [162].

(c) *Large increases in cancer in certain tissues.* Steep increases in cancer have occurred in tissues directly exposed to the environment: the lung and skin. However, some of the steepest increases have occurred in parts of the body with high fat content, including cancers of the brain, breast, bone marrow and liver. This again points to toxic chemicals that are predominantly stored in the fatty tissues.

(d) *Genetic mutation.* Many chemicals are known to attach to DNA, causing genetic change in the form of DNA adducts. The research of molecular epidemiologist, Dr Frederica Perera, of Columbia Center for Children's Environmental Health, has shown consistent associations between exposures to pollution and adduct formation on the one hand and adduct formation and cancer risk on the other [163,164]. Perera found two to three times the level of DNA adducts to polycyclic aromatic hydrocarbons in people in polluted areas and also found higher levels of adducts in people with lung cancer than in those without. Mothers exposed to pollution form DNA adducts but their babies have even higher adduct levels, potentially putting them at increased risk of cancer from birth [41].

(e) *Cancers and environmental pollution.* Several studies have already given direct evidence of a link between environmental pollution and cancer. These include the Long Island Study showing a link between airborne carcinogens and breast cancer [165,166] and the Upper Cape Study showing that tetrachloroethylene in the water was associated with elevated rates of several types of cancer [167–169]. It is noteworthy that initial investigations were negative in both these places and it was only demonstrated after detailed and sophisticated studies by scientists from many fields. Numerous other studies have shown links between cancer and chemicals: these include associations between VOCs in the water and increases in leukaemia in New Jersey [170], increases in lymphoma in counties in Iowa where drinking water was contaminated with dieldrin [171], elevated levels of leukaemia in children at Woburn, Massachusetts coinciding with a known period of water contamination with chlorinated solvents [172], a cancer cluster linked to consumption of river water contaminated by industrial and agricultural chemicals in Bynum, North Carolina [173] and high rates of non-Hodgkin's lymphoma where water was contaminated with chlorophenols in Finland [174].

(f) *Spread of cancer and pollutants.* Airborne pollutants not only affect the chance of contracting cancer but may also influence the chance of the cancer spreading. Animal studies have shown that inhalation of ambient level nitrogen dioxide, or polluted urban ambient air, facilitated blood-borne cancer cell metastasis [94].

(g) *Levels of carcinogens in the body.* The reality about most chemicals is that their risks are largely unknown. This is particularly true of chemicals new to the market. What we do

know is that about 5–10% are probable carcinogens. The International Agency for Cancer Research tested 1000 chemicals in 1993 and found that 110 were probable carcinogens [175]. The National Toxicity Program tested 400 chemicals in 1995 and found that 5–10% were carcinogenic [176]. Only 200 of the 75,000 synthetic chemicals in existence are regulated as carcinogens, whereas from these data, between 3000 and 7500 might be expected to be. We have even less knowledge about the carcinogenic potential of combinations of toxic chemicals, but what evidence we do have suggests that combinations may be more dangerous and yet these are what we are routinely exposed to.

Although the UK figures are not available, we know that 2.26 billion pounds of toxic chemicals were released in the USA in 1994: about 177 million pounds of these will have been suspected carcinogens. But what happens to all these chemicals? The reality is that much of this chemical pollution ends up inside us.

In a study, a group of middle-aged Americans were found to have 177 organochlorine residues in their bodies [177,178]. A recent study by the Mount Sinai School of Medicine measured chemicals in the blood and urine of healthy volunteers and found an average of 52 carcinogens, 62 chemicals toxic to the brain and nervous system and 55 chemicals associated with birth defects [179]. They point out that these were chemicals that could be measured and that there were many more that could not, making this a considerable underestimate. A study of pollutants in amniotic fluid found detectable levels of PCBs and pesticides at levels equivalent to the foetus's own sex hormones [180]. What this demonstrates is that what we put out into the world sooner or later comes back to us and will be stored in our bodies. This effect is slow, insidious and real. To allow carcinogens and other poisonous substances into our bodies in this way must be to gamble with our health.

Incinerators emit carcinogens. Particulates themselves are known to be carcinogenic, many heavy metals are known or suspected carcinogens, up to 10% of the chemical pollutants are carcinogenic and there is abundant evidence that carcinogens are far more dangerous when combined than when in isolation.

Common sense dictates that it is reckless to continue to pour more carcinogens into the air at a time when cancer is steadily increasing. Recent studies suggest that we already have to cope with 65 carcinogens in food, 40 carcinogens in water and 60 carcinogens in the air we breathe [181]. They should not be there at all. They should certainly not be increased. If we seriously want to prevent cancer it is of paramount importance that we rapidly decrease the levels of all carcinogens that we are exposed to.

5.2. *Neurological disease*

Most toxic compounds are stored in fatty tissue and this includes the brain, making the brain a key target organ for pollutants. There is now compelling evidence that heavy metals and other compounds such as PCBs and dioxins cause cognitive defects, learning problems and behavioural disturbances in children and these effects occur at levels previously thought to be safe [182]. It is inconceivable that these same pollutants have no impact on adult brain function.

Of great concern is the developing crisis of Alzheimer's disease, which now affects 4.5 million patients in the USA and 500,000 in the UK. This is a disease that had never been diagnosed until 1907 and in the UK had only reached 150 cases by 1948. At the present rate of increase, the numbers will double by 2030. These statistics are alarming, but need to be seen as part of an overall trend of increasing neurological disease. A recent study

noted substantial increases in neurological diseases in the last two decades coupled with earlier onset of these illnesses. These diseases include Alzheimer's disease, Parkinson's disease and motor neurone disease [183]. The increase in Alzheimer's disease was found in almost all developed countries, and increases varied across countries from 20% (which was defined as substantial) to 1200%. The paper suggested that environmental factors were probably responsible.

It is notable that these diseases of older people have increased at the same time that diseases affecting the brain (including ADHD, autism and learning difficulties) have also shown large increases at the other end of the age spectrum, of the order of 200–1700% [184]. It is very likely that these diseases have aetiological factors in common.

Heavy metal exposure is known to correlate with both Parkinson's disease [93,185] and Alzheimer's disease [66,67,87–91]. Both diseases have increased dramatically over the last 30 years. In addition we have already noted that the average person's body contains at least 62 chemicals that are toxic to the brain and nervous system [179]. It is crucial to look at every possible way to prevent Alzheimer's disease because of its huge care costs (US figures are \$60 billion annually) and its dire effect on both patients and carers.

Although multiple factors are probably involved in its causation, there is evidence of a link to heavy metal exposure and it is therefore imperative to reduce our exposure to these toxic metals and other neurotoxic chemicals by all means possible. To deliberately increase our exposure to these pollutants, at a time when these diseases are showing huge increases, shows a worrying lack of foresight.

5.3. *Mental diseases*

Many pollutants pass straight from the nose to the brain where they affect brain function. Air pollution correlates with in-patient admissions with organic brain syndrome, schizophrenia, major affective disorders, neurosis, behavioural disorders of childhood and adolescence, personality disorder and alcoholism [186]. Increases in the total number of psychiatric emergency room visits and schizophrenia [187] have been noted on days when air pollution has been high. Depression has also been linked to inhaled pollutants [188,189]. Clearly something very profound occurs when we pollute the air.

5.4. *Violence and crime*

An increasing number of studies, including studies of murderers [190], case-control and correlation studies [12,85,191,192] and prospective studies [83,193] have shown links between violence and heavy metals and these include lead, cadmium and manganese. The majority of the studies investigated lead. Violence and crime have been associated with both increased body levels of lead and with increased levels of lead in the air. For instance, Denno [194] found that early lead exposure was one of the most important predictors of disciplinary problems from 13 to 14 years of age, delinquency from 7 to 17 years of age and adult criminal offences from 18 to 22 years of age. Stretesky and Lynch [195] found an association between air lead levels and murder rates in US counties. It is interesting that air lead levels were a much stronger predictor of both violent and property crime than unemployment, which has often been considered an important cause for crime [196]. The likely mechanism is that these substances alter neurotransmitters such as dopamine and serotonin and reduce impulse control.

This growing literature should serve as a warning about the dangers of allowing heavy metals to be emitted into the environment. Crime, especially violent crime, can have a

dramatic effect on people's quality of life. We need to consider the effect of incinerators, not only on health, but on education and on quality of life, including the impact of violence and crime.

6. High risk groups

6.1. *The foetus*

The unborn child is the most vulnerable member of the human population. The foetus is uniquely susceptible to toxic damage and early exposures can have life-changing consequences. Why is the foetus so vulnerable? There are two main reasons. First, most of these chemicals are fat soluble. The foetus has virtually no protective fat stores until very late pregnancy, so the chemicals are stored in the only fatty tissues it has, namely its own nervous system and particularly the brain. Second, many pollutants are actively transported across the placenta from the mother to the foetus. This occurs with heavy metals, which the body mistakes for essential minerals. This is particularly critical for mercury, where one tenth of women already have body stores of mercury which can lead to neurodevelopmental problems in the newborn [197]. Other factors that increase foetal susceptibility are higher rates of cell proliferation, lower immunological competence and decreased capacity to detoxify carcinogens and repair DNA [198].

Safety limits currently do not take into account this increased risk to the foetus. Only 7% of high volume chemicals have been tested for neurodevelopmental toxicity [199] and very few pollutants have been tested for teratogenicity.

During a narrow window of time, in the first 12 weeks *in utero*, the foetus's body is affected by minuscule amounts of hormone measured in parts per trillion. Tiny amounts of chemicals can upset this delicate balance. It is now generally accepted that chemicals that are not toxic to an adult can have devastating effects on the newborn. Porterfield [10] has shown that small amounts of chemicals such as dioxins and PCBs, at doses that are not normally regarded as toxic, can affect thyroid hormones and neurological development. A single exposure is enough and timing is critical [200]. Small doses of oestrogenic chemicals can alter sexual development of the brain and the endocrine system [201].

It is estimated that 5% of babies born in the USA have been exposed to sufficient pollutants to affect neurological development [202]. It has also been shown that exposure to oestrogenic chemicals affects immunity, reduces the immune response to vaccines, and is associated with a high incidence of middle ear and recurrent respiratory infections [203]. The amount of chemical that the baby takes in relates to the total persistent contaminants that have built up in the mother's fat over her lifetime [204]. This will increase in areas around incinerators. Exposure to fine particulate pollution during pregnancy can have an adverse effect on the developing foetus and lead to impaired foetal growth [65].

In July 2005, in a ground-breaking study [205], researchers at two major laboratories in the USA looked at the body burden in the foetus. They reported an average of 200 industrial chemicals and pollutants (out of 413 tested) in the umbilical cord blood of 10 randomly chosen babies. These included 180 carcinogens, 217 chemicals that are toxic to the brain and nervous system, and 208 that can cause birth defects and abnormal development in animals. A statement by scientists and paediatricians said that the report raised issues of substantial importance to public health, showed up gaping holes in the government's safety net and pointed to the need for major reform to the nation's laws that aim to protect the public from chemical exposures.

Two months later, scientists at the University of Groningen released the results of a European study, commissioned by the World Wildlife Fund and Greenpeace, on the foetal body burden. They tested for the presence of 35 chemicals in the umbilical cord blood of newborns [206]. At least five hazardous chemicals were found in all babies and some had as many as 14 different compounds. The report questioned the wisdom of allowing the foetus to be exposed to a complex mixture of persistent, bioaccumulative and bioactive chemicals at the most critical stage of life.

Incinerators can only have the effect of increasing the foetal body burden and their use is therefore a retrograde step for society. It is particularly important to apply the precautionary principle in issues that affect the foetus, infant and child.

6.2. *The breast-fed infant*

It is a major concern that breast milk, perhaps the greatest gift a mother can give for the future health of her child, has now become the most contaminated food on the planet, in terms of persistent organic pollutants [207]. In the USA, studies of human breast milk have shown that 90% of samples contained a disturbing 350 chemicals. This was higher in industrialized areas, showing that inhalation of these toxic substances is an important factor [208]. The toxic dose taken in by a breast-feeding baby is 50 times higher than that taken in by an adult [209].

The incinerator would add to the total load of chemicals in the mother's fat and those toxins accumulated over a lifetime by the mother will then be transferred to the tiny body of her baby through her milk. Six months of breast-feeding will transfer 20% of the mother's lifetime accumulation of organochlorines to the child [210]. From 1979, one in four samples of breast milk have been found to be over the legal limit set for PCBs in commercial feeds [204] and these are known to impair intellectual development [211–213]. Contamination with persistent organic pollutants (POPs) in breast milk in animals has consistently shown structural, behavioural and functional problems in their offspring [214]. For instance, in monkeys it has been shown that it decreases their ability to learn [215–217]. PBDEs are toxic chemicals that have been doubling in breast milk every 5 years, and have also been rapidly increasing in the waste fed to incinerators as they are now present in many common electrical and electronic goods. PBDEs cause cancer, birth defects, thyroid dysfunction and immune suppression [218,219]. It is truly tragic that one of the few ways of removing these contaminants from the mother's body is by breast-feeding.

6.3. *Children*

Toxic and carcinogenic exposures in early life, including prenatal exposures, are more likely to lead to cancer than similar exposures later [220–222]. At the First International Scientific Conference of Childhood Leukaemia, held in September 2004, Professor Alan Preece suggested that pollutants crossing the placenta were damaging the immune system and could be linked with soaring rates of leukaemia, which were being initiated *in utero*. This theme was expanded by Professor George Knox in his recent study that found that children born in 'pollution hotspots' were two to four times more likely to die from childhood cancer. The 'hotspots' included sites of industrial combustion, and sites with higher levels of particulates, VOCs, nitrogen dioxides, dioxins and benz(a)pyrenes – in other words just what would be found around incinerators. He said that, in most cases, the mother had inhaled these toxic substances and they were then passed on to the foetus through the placenta [223]. This is supported by animal studies that have already

confirmed that cancer can be initiated by giving carcinogens before conception, *in utero* or directly to the neonate [224,225].

Developing systems are very delicate and in many instances are not able to repair damage done by environmental toxicants [226]. In one study there was an age-related difference in neurotoxicity for all but two of 31 substances tested; these included heavy metals, pesticides and other chemicals [227]. Children are not just a vulnerable group, but the current inhabitants of a developmental stage through which all future generations must pass. This fact is recognized in the passage of the Food Quality Protection Act in the USA. It requires that pesticide standards are based primarily on health considerations and that standards are set at levels that will protect the health of children and infants.

Developmental disorders, including autism and attention deficit syndrome, are widespread and affect 3–8% of children. The US National Academy of Sciences concluded in July 2000 that 3% of all developmental disorders were a direct consequence of toxic environmental exposures and another 25% are the result of interactions between toxic exposures and individual susceptibility. The causes include lead, mercury, PCBs, certain pesticides and other environmental neurotoxicants [228]. These are exactly the chemicals put out by incinerators.

The study of the Sint Niklaas incinerator found a multitude of problems in children, including learning defects, hyperactivity, autism, mental retardation and allergies [84] and this is exactly what would be anticipated from research already done on the health effects of heavy metals, PCBs and dioxins on both children and animals.

We also need to consider subclinical toxicity. The pioneering work of Herbert Needleman and colleagues [81] showed that lead could cause decreases in intelligence and alteration of behaviour in the absence of clinically visible signs of toxicity. This has also been shown to be the case with PCBs [229] and methyl mercury [70]. These effects are all the more likely when children are exposed to multiple pollutants, notably the heavy metals, which will be found in the cocktail of chemicals released by incinerators.

Although this has only minor implications for an individual, it can have major implications for a population. For instance, a five point drop in IQ in the population reduces by 50% the number of gifted children (IQ above 120) and increases by 50% the number with borderline IQ (below 80) [230]. This can have profound consequences for a society, especially if the drop in IQ is accompanied by behavioural changes.

6.4. *The chemically sensitive*

In the book, 'Chemical exposures, low levels and high stakes' by Professors Ashford and Miller [116], the authors noted that a proportion of the population react to chemicals and pollutants at several orders of magnitude below that normally thought to be toxic. For example, research has discovered individuals who react to levels of toxins previously considered to be safe. Two examples are benzene [231] and lead [82]. It has been demonstrated that there is a 10-fold difference between different individuals in the metabolism of the carcinogenic PAH benz(a)pyrene [232].

Ashford and Miller also noted that studies in both toxicology and epidemiology have recognized that chemicals are harmful at lower and lower doses and that an increasing number of people are having problems. A significant percentage of the population have been found to react this way (15–30% in several surveys with 5% having daily symptoms) [116]. Chemical sensitivity may be triggered by repeated modest non-toxic exposures or by one or more exposures to a somewhat higher concentration (often not generally regarded as toxic), after which symptoms start to occur at very low levels of exposure, not noticed by

other people [233]. Research has shown 150–450-fold variability in response to airborne particles [234]. Friedman [235] stated that environmental regulation requires the protection of these sensitive individuals. This highlights the dangers of incinerators, which emit a multitude of chemical compounds. Faults are all too common with modern incinerators, leading to discharges of pollutants at levels that endanger health – giving a very real risk of long-term sensitization. Certain susceptible individuals will be highly affected by these pollutants and these effects will be difficult to anticipate. In addition, people affected in this way are extremely difficult to treat.

7. Past mistakes and the precautionary principle

7.1. *The precautionary principle*

The precautionary principle has now been introduced into national and international law, including that of the European Union [236]. This principle involves acting in the face of uncertain knowledge about risks from environmental exposures. This means that public health measures should be taken in response to limited, but plausible and credible, evidence of likely and substantial harm [237]. In the case of incinerators, a recent review of health effects found that two-thirds of studies showed a positive exposure–disease association with cancer (mortality, incidence and prevalence) [238] and some studies pointed to a positive association with congenital malformations. It is absolutely clear from this and from the evidence presented here that building municipal waste incinerators violates the precautionary principle and perhaps European law.

7.2. *Learning from past mistakes*

Time and time again it has been found that what we did not know about chemicals proved to be far more important than what we did know. As an incinerator generates hundreds of chemicals, including new compounds, we can expect many unpleasant future surprises. Here are a few examples from the past:

- Chlorofluorocarbons (CFCs): These chemicals were touted as the safest chemicals ever invented when first synthesized in 1928. Thomas Midgeley received the highest award from the chemical industry for his discovery. After 40 years on the market suspicion fell on them. They were producing holes in the ozone layer exceeding the worst case scenario predicted by scientists.
- PCBs: These chemicals were introduced in 1929. Toxicity tests at the time showed no hazardous effects. They were on the market for 36 years before questions arose. By that time they were in the body fat of every living creature on the planet and evidence began to emerge of their endocrine-disrupting effects.
- Pesticides: Early pesticides included arsenical compounds but these killed farmers as well as pests. They were replaced by DDT. Paul Muller was awarded the Nobel Prize for this discovery, as it was considered a milestone in human progress. But DDT brought death in a different way and it was another two decades before it was banned. Less persistent pesticides then came onto the market but they had yet another unanticipated problem – endocrine disruption.
- Tributyl tin (TBT): In the early 1970s, scientists noted that irreversible damage was occurring to the reproductive system of fish, especially clams, shrimps, oysters, Dover

sole and salmon. It was 11 years before the cause was found and it was found to be due to TBT, a chemical added to paint to stop barnacles growing. Incredibly the damage was occurring at a concentration of just five parts per trillion. By the end of the 1980s more than 100 species of fish were known to have been harmed.

This pattern of unanticipated disasters and long latent intervals before their discovery characterizes the history of many toxic chemicals and warrants great caution in the use of new compounds. Animal studies often underestimate the uniquely human neurotoxic effects on behaviour, language and thinking. In the case of lead, mercury and PCBs, the levels of exposure needed for these effects to occur have been overestimated by a factor of 100 to 10,000 [239]. To quote Grandjean et al. [237] “Past experiences show the costly consequences of disregarding early warnings about environmental hazards. Today the need for applying the Precautionary Principle is even greater than before”.

8. Alternative waste technologies

An ideal waste strategy would produce no toxic emissions, no toxic by-products, no residues that need landfilling (zero waste), good recovery of materials and be capable of dealing with all types of waste. This might seem a tall order, but it is now possible to come quite close to this goal.

Once this aim is made clear then incineration becomes a poor choice. The potentially dangerous emissions to the air, the high volume of ash that needs landfilling and the very toxic nature of the fly ash would rule it out. Similarly, pyrolysis produces toxic by-products and is best avoided.

No single strategy can achieve these aims, so what is needed is an integrated strategy. The first component must be some form of separation and recycling. Three forms of waste strategy then need to be considered: mechanical–biological treatment (MBT), anaerobic digestion (which can be a part of the above) and types of gasification that produce no ash.

8.1. MBT

This treatment is used extensively in Germany, Italy and Austria. It has been in use for over 10 years and is due to be introduced into the UK. The process involves a mechanical stage in which the waste is chopped up into fragments and then separated by being put through screens of various sizes and past magnets. This process will separate the waste into fractions, which can be used for different purposes. For instance, metals, minerals and hard plastics can then be recycled. Paper, textiles and timber can also be recovered. Organic matter can then be broken down by composting – this is the biological treatment. This can be achieved by exposing the waste to atmospheric oxygen or it can be broken down in the absence of oxygen (anaerobic digestion). The remaining rubbish can then be landfilled. This process is virtually pollution free, unless the remaining pellets are burned with all the risks this entails. With MBT, most of the original goals are being met. It fails on two counts only. First, there is some residue that needs landfilling – this is a minor point but the second is more serious: MBT cannot cope with all types of waste as it is not suitable for hazardous waste. This is important, as the amount of hazardous waste is likely to increase. So MBT needs to be part of a system.

It should be pointed out that the major problem with landfilling is presently not a lack of space, but the release of methane gas from landfill sites, which adds to greenhouse gases. This would not be a problem with MBT, as the residue has had the organic matter removed.

8.2. *Gasification methods (that produce no ash)*

This means plasma gasification or high temperature gasification using the thermoselect process. This achieves the final objective by disposing of residual waste. It is worth noting that plasma gasification can produce up to three times as much energy as incineration and can deal safely with the most hazardous types of waste.

Gasification has been employed by the natural gas industry for over 80 years, but has not, so far, been used extensively for dealing with waste, although plants are now in operation in Italy, Switzerland, Germany and Japan. Gasification produces high temperatures and converts complex organic molecules to simple gases. Plasma refers to the gas when it has become ionized and this happens when an electric current is passed through the gas. Unlike incineration it does not produce contaminated ash. The gas cleaning process can convert many contaminants into environmentally benign and useful by-products. There is a very basic difference in the abatement equipment of incinerators and gasification units. If the abatement equipment in an incinerator fails then people downwind can suffer health effects. If the abatement equipment in a gasification unit fails it will cause serious damage to the plant itself – so the plant has to be built to a much higher quality.

Toxic substances, including metals, become encapsulated in silicate, which is like being encased in stone. A good quality plasma gasification unit will not produce any adverse residues or by-products, only silica, sulphur and salt. It produces a useful by-product called synthesis gas, which can be used as a fuel; this is a major financial advantage allowing the capital costs of the unit to be paid within a 7 year period. Although it is a relatively expensive process, it is far cheaper than incineration once the health costs are taken into account. If it is combined with MBT and recycling then only a small unit is needed.

8.3. *Recycling*

The UK presently recycles about 18% of its waste. Many other countries recycle a far higher proportion of their waste, with Norway, Austria and Holland achieving over 40% and Switzerland over 50%.

Recycling could be increased vastly. In America many cities have achieved high levels of recycling, the figures being 50% in Seattle, 45% for the state of New Jersey and 70% in Edmonton, Canada. Flanders in Belgium has cut its waste by 59% and Canberra by 56%.

Recycling is far more energy efficient. Two American studies have shown that recycling saves about three to five times as much energy as incineration.

However, one of the most important lessons to be learned is that we need to produce less waste in the first place.

9. **Other considerations of importance**

9.1. *The costs of incineration*

The cost of incineration is huge. A recent report by the European Commission suggested that for every tonne of waste burnt there would be between £21 and £126 of health and

environmental damage. This means that a 400,000 tonnes per year incinerator would cost the tax payer between £9,000,000 and £57,000,000 per year [240]. Another report suggested an incinerator of this size would cost 48,000,000 euros in health damage [240]. And yet methods such as gasification and MBT with low environmental and health costs (see Section 8) are not being given sufficient consideration in the UK. MBT is relatively cheap, but plasma gasification is more expensive to install. However, if plasma gasification was combined with MBT or similar methods, it would have an equivalent cost to incineration at 10 years because of the extra electricity produced, and from then on would be more profitable. However, once the health costs are taken into account, plasma gasification is very much cheaper. It makes no logical sense to use a method of waste disposal that has a total cost far in excess of other methods. The human and health costs must be part of the equation.

The European Commission Okopol report of 1999 [241] showed that every pound spent on pollution abatement saved £6 in health care costs and £4 in social security costs. A report from the US EPA again showed that every dollar spent on abatement saved \$10 in health costs.

In addition, a White House study by the Office of Management and Budget in 2003 concluded that enforcing clean air regulations led to reductions in hospitalizations, emergency room visits, premature deaths and lost workdays, which led to a saving of between \$120 and \$193 billion between October 1992 and September 2002 [242]. This is certainly an underestimate, as it did not look at other health savings such as prescription costs and primary care costs. Few measures today would give so dramatic a health benefit and such a large saving in health costs.

The World Wildlife Fund investigated three conditions: mental retardation, cerebral palsy and autism, to assess the impact of chemical pollution, and calculated the cost of toxic chemicals on children's brain development to be approximately £1 billion annually [243].

9.2. *The problem of ash*

The incineration of waste produces a large amount of ash, amounting to 30% of the volume of the original waste. This ash would occupy 40–50% of the volume of that waste if that waste had been compacted. In other words, incineration is no solution to the problem of a lack of landfill sites. This is important as only a few landfill sites will be available after 2011, so it is clear that incineration will not solve the landfill problem. Little thought has been given to this and incinerator operators are still being given 20–30 year contracts, creating problems for the future. Incinerators produce two types of ash, bottom ash and fly ash (sometimes called air pollution control residues). The latter is highly toxic, as it is laden with heavy metals and dioxin.

There is a basic problem with modern incinerators. The less air pollution produced, the more toxic the ash. Early incinerators emitted large volumes of dioxins. These have been significantly reduced in gaseous emissions but have greatly increased in the fly ash, together with heavy metals and other toxic chemicals. An incinerator burning 400,000 tonnes of waste annually for its 25 years of operation would produce approximately half a million tonnes of highly toxic fly ash (P. Rossington, pers. comm.). No adequate method of disposing of fly ash has been found. It is presently landfilled at special sites and this involves lengthy road journeys where accidents are always a possibility. The European Union Commission has stated that leaching from landfill sites may be one of the most important sources of dioxins in the future. These and other pollutants could leach into the water table, where their removal would be near impossible.

In spite of the massive health risks associated with fly ash it is poorly regulated. At Byker (Newcastle-upon-Tyne, UK), toxic ash laden with dioxins was spread over allotments, bridle paths and footpaths for 6 years.

9.3. *Radioactivity*

Over 30 sites in the UK incinerate radioactive waste. Most countries consider this too hazardous. The abatement systems of incinerators are not equipped to remove the radioactive material and previous experience suggests that most radioactive waste will pass straight through the incinerator abatement system and into the surrounding air as particulates. The rest will make the ash highly toxic. The radioactive matter emitted will be breathed in by people in the area, passing into their lungs, circulation and cells. In effect they will receive a dose of radioactivity. The risk from this policy is obvious. There is no safe level of radioactive PM_{2.5} particulates.

Increased incidences of leukaemias and cancers around sites releasing radioactive material are well documented. At Seascale (UK), a public health enquiry found that children were more than 10 times more likely to get leukaemia and three times more likely to get cancer [244,245]. The incidence of leukaemias in children living within 5 km of the Krummel and Goesthact nuclear installations in Germany is much higher than in Germany as a whole. Significantly, the first cases of leukaemia only appeared 5 years after Krummel was commissioned. At Dounreay (UK) there was a six-fold increase in children's leukaemia [246] and at Aldermaston there was also an increase in leukaemias in children under 5 years old [247]. Sharply rising leukaemia rates were noted in five neighbouring towns surrounding the Pilgrim nuclear plant in Massachusetts in the 1980s. It was thought to be linked to radioactive releases from the Pilgrim nuclear plant 10 years earlier when there had been a fuel rod problem. "Meteorological data showed that individuals with the highest potential for exposure to Pilgrim emissions had almost four times the risk of leukaemia compared to those having the lowest potential for exposure" [248,249].

The weight of evidence here strongly suggests that airborne radioactivity is a potent carcinogen and likely to be extremely hazardous. To combine this with a cocktail of other carcinogens is reckless.

9.4. *Spread of pollutants*

The National Research Council, an arm of the National Academy of Sciences, established to advise the US government, concluded that it was not only the health of workers and local populations that would be affected by incinerators. They reported that populations living more distantly are also likely to be exposed to incinerator pollutants. They stated:

Persistent air pollutants, such as dioxins, furans and mercury can be dispersed over large regions – well beyond local areas and even the countries from which the sources emanate. Food contaminated by an incinerator facility might be consumed by local people close to the facility or far away from it. Thus, local deposition on food might result in some exposure of populations at great distances, due to transport of food to markets. However, distant populations are likely to be more exposed through long-range transport of pollutants and low-level widespread deposition on food crops at locations remote from an incineration facility [250].

They later commented that the incremental burden from all incinerators deserves serious consideration beyond a local level. This has obvious relevance to the present policy of promoting incinerators in the UK. An important point is that the more toxic smaller particulates, which typically have more toxic chemicals and carcinogens attached, will travel the furthest [251].

Most chemical pollutants are lipophilic and are therefore not easily washed away by the rain after they settle. When they land on crops they enter the food chain where they bioaccumulate. It has already been admitted that most dioxin in food today in the UK came from the older generation of incinerators. All chemicals capable of entering the food chain will sooner or later reach their highest concentration in the foetus or breast-fed infant.

A striking example of the unforeseen and tragic consequences of releasing pollutants into the air has been seen in Nunavut, in the far north of Canada in the Polar Regions. The Inuit mothers have twice the level of dioxins in their breast milk as Canadians living in the south, although there is no source of dioxin within 300 miles. At the Center for the Biology of Natural Systems in Queen's College, New York, Dr Commoner and his team used a computer program to track emissions from 44,000 sources of dioxin in North America. This system combined data on toxic releases and meteorological records. Among the leading contributors to the pollution in Nunavut were three municipal incinerators in the USA [252,253].

10. Cement kilns

Although this report is primarily about incinerators, it is useful to compare incinerators with cement kilns. Both produce toxic emissions of a similar type and much of the report is relevant to both. Cement kilns convert ground limestone, shale or clay into cement. They require large quantities of fuel to produce the high temperatures needed and this lends itself to the use of non-traditional fuels such as tyres, refuse-derived fuel and industrial and hazardous wastes variously called Cemfuel, secondary liquid fuel and recycled liquid fuel.

However, pollution and planning controls are significantly weaker than those for hazardous waste incinerators. Cement kilns produce a number of toxic emissions, including mercury, manganese, barium, lead, sulphuric acid, styrenes, dioxins and 1,3 butadiene.

The thermal treatment of hazardous waste is always a highly dangerous activity and the very best available technology needs to be used. Cement kilns are effectively being used to burn hazardous waste on the cheap. Sadly hazardous waste typically finds its way to the least regulated and cheapest disposal methods, in practice those that create the most health risks and the most environmental damage.

Cement kiln technology has remained virtually unchanged since the turn of the twentieth century. They can only be refitted or retrofitted to a minimal degree to improve efficiency and toxic waste destruction.

The limit set for the weight of particulates emitted by incinerators is 10 mg/m^3 . However cement kilns are allowed to emit up to 50 mg/m^3 . This would be excessive by itself, but the volumes of emissions from cement kilns can be up to five times greater than incinerators. Therefore, some cement kilns can produce emissions of particulates and other toxic substances that are in excess of 20 times that of incinerators. Worse still they have poorer abatement equipment and usually lack the activated charcoal needed to reduce emissions of metals and dioxins.

They are therefore capable of extremely serious health consequences. Incredibly some of these cement kilns have been sited in the middle of towns where they would be expected to

have a major effect on the health of the local population. The fact that they are allowed at all is astonishing, for the maximum impact will inevitably be on the most vulnerable members of society, and in particular the unborn child.

11. Monitoring

At the heart of the problems with incineration is the unsatisfactory nature of monitoring at these installations, unsatisfactory in the way it is done, the compounds monitored, and the levels deemed acceptable, and the lack of monitoring of body burdens in the local population.

- Very few pollutants are being measured: Out of the hundreds of chemicals released from an incinerator, only a tiny proportion are measured. Only half a dozen of these are measured continuously in the stack and about another half dozen are measured occasionally (usually 6 monthly for the first year and then yearly) by spot monitoring – these include heavy metals and dioxins. This is clearly unsatisfactory and as waste operators are warned in advance of a visit, they are handed an opportunity to change to burning cleaner waste, which is unrepresentative of the toxic risk.
- In addition to monitoring in the stack, there is a requirement to monitor pollutants in the surrounding air. This is normally done by the local council. However, this is also unsatisfactory. For instance, to monitor for safe levels of particulates it would require at least 24 monitors placed at strategic points around an incinerator (assuming the wind is distributed evenly) to achieve a 25% sampling rate, which is the minimum that can be considered acceptable. Typically there are less than three monitors around most incinerators today. The measurement of heavy metals in the surrounding air, with the exception of lead, is not even required.
- Measuring their concentrations in the stack of the incinerator at one point in time gives virtually no information about the total amounts of pollutants to which the local population is exposed.
- Current monitoring tells us nothing about the body burdens of pollutants. Even if present in low amounts, most of the pollutants emitted by incinerators will accumulate slowly in people in the vicinity. Chronic toxicity is a risk whenever pollutants are accumulated faster than they are eliminated: this is particularly the case for heavy metals and POPs. For some pollutants, excretion rates are very poor. For example, the half life of cadmium in the body is 30 years and for PCBs it is 75 years, and even without further exposure it would take much longer to clear cadmium or PCBs from the human body.
- There has been no attempt to measure the health effects of this accumulation. For this to be achieved it would be necessary to monitor the concentrations of toxic chemicals in people's bodies as they slowly accumulate them over time, and the effects on their health. Although susceptibility will vary from individual to individual, toxic accumulation is likely in almost everyone exposed to incinerator emissions, faster in some than others, and faster for some pollutants than for others. The testing of body burdens is therefore an essential part of monitoring.
- Safety levels often rely on animal studies, which underestimate the risk. Animal studies commonly underestimate human vulnerability because of the obvious difficulty in testing cognitive, behavioural and language deficiencies and conditions such as fatigue. In the case of lead, mercury and PCBs, animal studies have underestimated the neurotoxic effect on humans by a factor of 100–10,000 times [239].

- Safety levels only apply to adults. Average levels or spot monitoring ignores exposures at critical times. The timing of the exposure is often more important than the concentration. Exposures at critical times during foetal growth or infancy are known to produce more serious effects than similar exposures in adulthood and this damage can be permanent. This is well recognized, especially with lead, mercury and PCBs.
- None of the safety limits has been demonstrated to protect against foetal damage. We know from animal and human studies that toxins have the greatest impact on the foetus and young child, but this is not taken into account in the current legislation and so the most vulnerable members of the community are likely to bear the brunt of the toxic load.
- Low-dose toxicity is being ignored. Low-dose studies often show toxic effects at levels far below the 'no effect' level in high-dose studies. An example of this is bisphenol A, a plasticizer. Studies showed health effects at levels 2500 times lower than the US EPA's lowest observed effect, with adverse outcomes including aggressive behaviour, early puberty and abnormal breast growth [179]. Perchlorate produces changes in the size of parts of the brain at 0.01 mg/kg/day but not at 30 mg [179]. Aldicarb was found to suppress the immune system more at 1 part per billion than it did at 1000 parts per billion. Other chemicals also produce different effects at low doses than at high doses. This shows how very little we know about the dangers of exposing people to chemical pollution.
- Monitoring is inadequate. Ten incinerators in the UK committed 553 pollution offences in a 2 year period, documented in Greenpeace's 'A review of the performance of municipal incinerators in the UK' [254]. This appalling record led to only one prosecution by the Environment Agency. This clearly gives waste companies a green light to ignore regulations and pollute as much as they want. These data were based on self-assessment by the companies concerned. When an environmental group investigated an incinerator in Indianapolis the situation was far worse. They found it had violated its permits 6000 times in 2 years and bypassed its own air control pollution devices 18 times. In effect, public safety is dependent on how well the incinerator is run and the evidence suggests that it is often run badly.

12. Risk assessment

One might reasonably expect that, when the decision to build an incinerator is made, all the above information would be carefully taken into account. Sadly this is not necessarily the case. Directors of Public Health, who usually have little knowledge of environmental health, are asked to write an IPPC (Integrated Pollution Prevention and Control) Application Report and give their opinion on the health risks from the proposed incinerator. Typically this decision is based on an inexact method called risk assessment. They tend to rely almost exclusively on this type of assessment and often have little understanding of its limitations.

Risk assessment is a method developed for engineering, but is very poor for assessing the complexities of human health. Typically it involves estimating the risk to health of just 20 out of the hundreds of different pollutants emitted by incinerators.

There are a host of problems with this type of assessment: lack of accurate data on pollutants, lack of toxicological data on the majority of chemicals, the fact that an increasing proportion of people react to low levels of chemicals, the fact that in the real world pollutants come in mixtures and can have damaging synergistic effects, the fact that the foetus and breast-fed baby take in 50 times more pollutants than adults relative to their

weight, and that there is virtually no toxicological data on the effect of these pollutants on either the foetus or the baby.

Further problems are that many pollutants have no safe thresholds so there can be no safe level. Indeed some pollutants are more dangerous at low concentrations than high (see Section 11). In fact, it is impossible to assess risk when the toxic effects of 88–90% of chemicals and pollutants are unknown [255], particularly in relationship to birth and developmental defects. This type of assessment contains a value judgement about what is an acceptable level of risk [256]. For instance, what is an acceptable number of birth defects and who is it acceptable to?

Risk assessment usually involves ‘modelling’, which uses an estimation of exposure data, rather than actual exposure data, to assess the impacts of pollutants and their likely distribution. These reports are typically produced by the polluter. Unfortunately, modelling has a 30% confidence level. This means that this technique has only a 30% chance of accurately predicting the ground level concentrations of pollutants. In other words, less accurate than tossing a coin. Different models give very different results.

In addition, present modelling methods seriously underestimate the levels of pollutants. In particular, modelling almost never takes into account secondary particulates formed as the products of combustion rise up the stack. These secondary particulates can easily double the total volume of particulates (see Section 2.1).

Modelling produces the illusion of a scientific knowledge and a certainty that is entirely unjustified, as modelling itself is imprecise and it is based on substantial scientific uncertainty and limited scientific data. It produces a mass of complex mathematical data, which implies unjustified precision, and it is difficult for people not familiar with the mathematics to disentangle the inaccuracies. It is often treated by regulators and Directors of Public Health as if it was an accurate assessment [257]. In spite of these severe limitations it is extensively used.

These risk assessments have almost always concluded that incinerators are safe, which flies in the face of epidemiological data that show the opposite. It also flies in the face of the history of chemical use. The latter is littered with examples of chemicals once said to be safe that were later found to have devastating and unanticipated effects, often beyond the worst case scenario (e.g. DDT, PCBs, CFCs) (see Section 7.2).

13. Public rights and international treaties

In 2001, the United Nations Commission on Human Rights stated that “everyone has the right to live in a world free from toxic pollution and environmental degradation”.

It is unethical that people should die from the emissions from incinerators when safe alternatives are available and for this reason incineration violates Article 2 of the European Human Rights Convention, the Right to Life.

The Stockholm Convention, agreed to by over 100 countries including Britain in 2001, commits countries to eliminating POPs, including PCBs, dioxins and furans. It identifies incinerators as the primary sources of these. Incineration is a violation of the Stockholm Convention.

Incineration is also a violation of the Environmental Protection Act of 1990, which states that the UK must prevent emissions from harming human health.

14. Conclusions

- (1) Large epidemiological studies have shown higher rates of adult and childhood cancers and birth defects around incinerators. Smaller studies and a large body of related research support these findings, point to a causal relationship, and suggest that a much wider range of illnesses may be involved.
- (2) Recent research has confirmed that particulate pollution, especially the fine particulate ($PM_{2.5}$) pollution that is typical of incinerator emissions, is an important contributor to heart disease, lung cancer, and an assortment of other diseases, and causes a linear increase in mortality. Incinerators are in reality particulate generators, and their use cannot be justified now that it is clear how toxic and carcinogenic fine particulates are.
- (3) Other pollutants emitted by incinerators include heavy metals and a large variety of organic chemicals. These substances include known carcinogens, endocrine disrupters, and substances that can attach to genes, alter behaviour, damage the immune system and decrease intelligence. The dangers of these are self-evident. Some of these compounds have been detected hundreds to thousands of miles away from their source.
- (4) Additional dangers arise from radioactive particulates emitted from incinerators licensed to deal with hazardous waste.
- (5) Incineration only reduces the volume of waste by 30–50%. Modern incinerators produce far more toxic fly ash (air pollution control residues) than older incinerators; these pose long-term health risks. No adequate methods exist for the disposal of this ash.
- (6) The greatest concern is the long-term effects of incinerator emissions on the developing embryo and infant, and the real possibility that genetic changes will occur and be passed on to succeeding generations. A far greater vulnerability to toxins is documented for the very young, particularly foetuses, causing cancer, spontaneous abortion, birth defects or permanent cognitive damage. A worryingly high body burden of pollutants has recently been reported in two studies of cord blood from newborn babies.
- (7) Waste incineration is prohibitively expensive when health costs are taken into consideration. The European Commission figures indicate that a single incinerator could cost the tax payer up to £50 million a year. Recent American data showed that strict air pollution control has saved tens of billions of dollars a year in health costs.
- (8) Waste incineration is unjust because its maximum toxic impact is on the most vulnerable members of our society, the unborn child, children, the poor and the chemically sensitive. It contravenes the United Nations Commission on Human Rights, the European Human Rights Convention (the Right to Life), and the Stockholm Convention, and violates the Environmental Protection Act of 1990, which states that the UK must prevent emissions from harming human health.

15. Recommendations

- (1) The safest methods of waste disposal should be used.
- (2) Health costs should be routinely taken into account when deciding on waste disposal strategies.

- (3) The present limited method of risk assessment by which the safety of proposed installations is judged is inadequate, cannot be relied on, and should be reviewed.
- (4) Tackling the problems of both the amount and the nature of waste generated is of critical importance, with the emphasis on reducing the production of waste and on recycling.
- (5) The serious health consequences of fine particulate pollution have become apparent in the last 10 years: incinerators are a major source and, in our considered opinion, incineration is the least preferred option for getting rid of waste. Taking account of all the information available, including research indicating that there are no safe levels for fine particulates, we can see no reason to believe that the next generation of incinerators would be substantially safer than the previous ones.
- (6) Far safer alternative methods are now available, including recycling, MBT and plasma gasification: a combination of these would be safer, would produce more energy, and would be cheaper than incineration in the long run, much cheaper when the health costs are taken into account. These more up-to-date methods should be employed.
- (7) It is particularly important that incinerators should not be sited in deprived areas or areas with high rates of mortality where their health impact is likely to be greatest. This can only add to health inequalities. Presently nine out of 14 incinerators have been built in the most deprived 20% of wards [258].
- (8) This report outlines the many deficiencies of the present monitoring procedures. We recommend the introduction of a stricter and more comprehensive system for the monitoring of all waste-burning plants by a fully independent body, including random unannounced visits. The monitoring should include:
 - (a) more monitors around incinerators to measure particulates and heavy metals;
 - (b) periodic monitoring of the content of dust in homes in the locality;
 - (c) periodic monitoring of the heavy metals and dioxins in the fly ash;
 - (d) a programme of monitoring the body burdens of some key pollutants in local inhabitants.
- (9) We recommend that no further waste incinerators be built.

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