The Health Effects of Waste Incinerators

4th Report of the British Society for Ecological Medicine

Moderators: Dr Jeremy Thompson and Dr Honor Anthony

Foreword

from **Professor C. V. Howard. MB. ChB. PhD. FRCPath.**

The authors are to be congratulated on producing this report. The reader will soon understand that to come to a comprehensive understanding of the health problems associated with incineration it is essential to become acquainted with a large number of different disciplines ranging from aerosol physics to endocrine disruption to long range transport of pollutants. In most medical schools, to this day, virtually nothing is routinely taught to equip the medical graduate to approach these problems. This has to change. We need the medical profession to be educated to health consequences associated with current environmental degredation.

There are no certainties in pinning specific health effects on incineration: the report makes that clear. However this is largely because of the complexity of exposure of the human race to many influences. The fact that 'proof' of cause and effect are hard to come by is the main defence used by those who prefer the *status quo*. However the weight of evidence, collected within this report, is sufficient in the authors' opinion to call for the phasing out of incineration as a way of dealing with our waste. I agree with that.

There is also the question of sustainability. Waste destroyed in an incinerator will be replaced. That involves new raw materials, manufacture, transport, packaging etc etc. In contrast, reduction, reuse and recycling represent a win-win strategy. It has been shown in a number of different cities that high levels of diversion of waste (>60%) can be achieved relatively quickly. When that happens, there is not very much left to burn, but a number of the products left will be problematic, for example PVC. Incineration, an end of pipe approach, sends the message 'No problem, we have a solution for disposal of your product, carry on business as usual'. What should happen is a 'front end solution'. Society should be able to say 'Your product is unsustainable and a health hazard – stop making it".

Incineration destroys accountability and this encourages industries to go on making products that lead to problematic toxic wastes. Once the waste has been reduced to ash who can say who made what? The past 150 years has seen a progressive 'toxification' of the waste stream with heavy metals, radionuclides and synthetic halogenated organic molecules. It is time to start reversing that trend. We won't achieve that while we continue to incinerate waste.

Vyvyan Howard December 2005

Professor of Bioimaging, Centre for Molecular Biosciences,
University of Ulster, Cromore Road, Coleraine, Co. Londonderry BT52 1SA

Contents

Executive Summary

- Large studies have shown higher rates of adult and childhood cancer and also birth defects around municipal waste incinerators: the results are consistent with the associations being causal. A number of smaller epidemiological studies support this interpretation and suggest that the range of illnesses produced by incinerators may be much wider.
- Incinerator emissions are a major source of fine particulates, of toxic metals and of more than 200 organic chemicals, including known carcinogens, mutagens, and hormone disrupters. Emissions also contain other unidentified compounds whose potential for harm is as yet unknown, as was once the case with dioxins. Since the nature of waste is continually changing, so is the chemical nature of the incinerator emissions and therefore the potential for adverse health effects.
- Present safety measures are designed to avoid acute toxic effects in the immediate neighbourhood, but ignore the fact that many of the pollutants bioaccumulate, can enter the food chain and can cause chronic illnesses over time and over a much wider geographical area. No official attempts have been made to assess the effects of emissions on long-term health.
- Incinerators produce bottom and fly ash which represent 30-50% by volume of the original waste (if compacted), requiring transportation to landfill sites. Abatement equipment in modern incinerators merely transfers the toxic load, notably that of dioxins and heavy metals, from airborne emissions to the fly ash. This fly ash is light, readily windborne and mostly of low particle size. It represents a considerable and poorly understood health hazard.
- Two large cohort studies in America have shown that fine ($PM_{2.5}$) particulate air pollution causes increases in all-cause mortality, cardiac mortality and mortality from lung cancer, after adjustment for other factors. Fine particulates are primarily produced by combustion processes and are produced in large quantities by incinerators.
- Ischaemic heart disease was responsible for nearly a quarter of deaths in one of the cohort studies and was strongly related to the level of $PM_{2.5}$ particulates. An increase of 24.5 mcg/m^3 in $PM_{2.5}$ particulate pollution, was associated with a 31% increase in cardiopulmonary mortality. Short-term increases in fine particulates, as will occur downwind from incinerators, have also been shown to cause significant increases in myocardial infarctions.
- Higher levels of fine particulates have been associated with an increased prevalence of asthma and COPD.
- Fine particulates formed in incinerators in the presence of toxic metals and organic toxins (including those known to be carcinogens), adsorb these pollutants and carry them into the blood stream and into the cells of the body.
- Toxic metals accumulate in the body and have been implicated in a range of emotional and behavioural problems in children including autism, dyslexia, attention deficit and hyperactivity disorder (ADHD), learning difficulties, and delinquency, and in problems in adults including violence, dementia,

depression and Parkinson's disease. These metals are universally present in incinerator emissions and present in high concentrations in the fly ash.

- Susceptibility to chemical pollutants varies, depending on genetic and acquired factors, with the maximum impact being on the foetus. Acute exposure can lead to sensitisation of some individuals, leaving them with life-long low dose chemical sensitivity.
- Few chemical combinations have been tested for toxicity, even though synergistic effects have been demonstrated in the majority of cases when this testing has been done. This synergy could greatly increase the toxicity of the pollutants emitted, but this danger has not been assessed.
- Both cancer and asthma have increased relentlessly along with industrialisation, and cancer rates have been shown to correlate geographically with both toxic waste treatment facilities and the presence of chemical industries, pointing to an urgent need to reduce our exposure.
- Incinerators burning radioactive material will produce radioactive particulates. This material is carcinogenic and no studies have been done to assess the danger to health of these radioactive emissions.
- Some chemical pollutants such as polyaromatic hydrocarbons (PAHs) and heavy metals are known to cause genetic changes. This represents not only a risk to present generations but to future generations.
- Monitoring of incinerators has been unsatisfactory in the lack of rigor, the infrequency of monitoring, the small number of compounds measured, the levels deemed acceptable, and the absence of biological monitoring. Approval of new installations has depended on modelling data, supposed to be scientific measures of safety, even though the method used has no more than a 30% accuracy and ignores the important problem of secondary particulates.
- It has been claimed that modern abatement procedures render the emissions from incinerators safe, but this is impossible to establish. Moreover two of the most hazardous emissions – fine particulates and heavy metals – are relatively resistant to removal.
- The safety of new incinerator installations cannot be established in advance and, although rigorous independent health monitoring might give rise to suspicions of adverse effects on the foetus and infant within a few years, this type of monitoring has not been put in place, and in the short term would not reach statistical significance for individual installations. Other effects, such as adult cancers, could be delayed for at least ten to twenty years. It would therefore be appropriate to apply the precautionary principle here.
- There are now alternative methods of dealing with waste which would avoid the main health hazards of incineration and would be far cheaper in real terms, if the health costs were taken into account.
- Incinerators presently contravene basic human rights as stated by the United Nations Commission on Human Rights, in particular the Right to Life under the European Human Rights Convention, but also the Stockholm Convention and the Environmental Protection Act of 1990. The foetus, infant and child are most at risk from incinerator emissions: their rights are therefore being ignored and violated, which is not in keeping with the concept of a just

society. Nor is the present policy of locating incinerators in deprived areas where their health effects will be maximal: this needs urgent review.

- The literature reviewed leads us to the opinion that new facilities emitting substantial quantities of fine particulates, volatile heavy metals and hazardous organic pollutants should not be approved and that urgent measures should be taken to reduce the emissions from waste burning installations in current use and to apply rigorous biological monitoring until they can be taken out of service and safer methods of waste disposal brought into operation. Vigorous efforts should also be made to reduce the amount of waste produced as there is presently no entirely satisfactory solution for its disposal.

1. Introduction

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

Incinerators produce pollution in two ways. Firstly, 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 bio-accumulative, building up over time in the human body in an insidious fashion 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 the 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 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 which 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 which 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 by 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 microns whereas fine particulates ($PM_{2.5}$s) are less than 2.5 microns and ultrafine particulates (PM_1s) are less than 1 micron. Incinerators produce huge quantities of fine and ultrafine particulates. Incinerators are permitted to emit particulates at a rate of $10mg/m^3$ 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_1s. In fact the majority of particles emitted by incinerators are the most dangerous ultrafine particulates (1). The baghouse filters are least effective at removing the smallest particles, especially those of 0.2 to 0.3 microns, 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 (3). Present modelling methods do not take secondary particulates into account (see section 12).

Studies have shown that toxic metals accumulate on the smallest particulates (4) and that 95% of polycyclic aromatic hydrocarbons (PAHs) are associated with fine particulates (PM_3 and below) (5-7). PAHs are toxic and carcinogenic, and it has been estimated that these increase the lung cancer risk by 7.8 times (8).

2.2 Heavy Metals

Incinerators are allowed to emit 10mg/m^3 of particulates and 1mg/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 (4) 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 from cars.

2.3 Nitrogen Oxides

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 volatile organic compounds (VOCs) to produce another pollutant, **ozone**.

2.4 Organic Pollutants

A wide range of organic pollutants are emitted from incinerators. These include PAHs (polycyclic aromatic hydrocarbons), PCBs (polycyclic aromatic hydrocarbons), dioxins, furans (polychlorinated biphenyls), 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 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 bio-accumulative and persistent. They include compounds that have been reported to affect the immune system (9), attach to chromosomes (10), disrupt hormone regulation (11), trigger cancer (12), alter behaviour (13), and lower intelligence (14). The very limited

toxicity data on many of these substances is a matter of concern (15). The changing nature of waste means 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 (16, 17).

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* (18-21). The data from the World Health Organisation shown in the graph below clearly illustrates that $PM_{2.5}$ particles have a greater effect on daily mortality than the larger PM_{10}s (18).

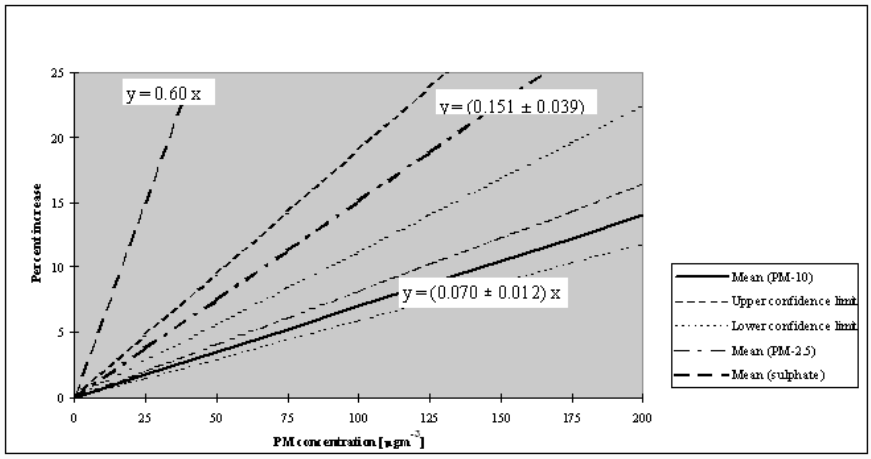

Figure 1. Increase in daily mortality as a function of PM concentration.
(reproduced from ref 18, Figure 3.6)

The smaller particles are not filtered out by the nose and bronchioles and their miniscule 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 (22). 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 $PM_{2.5}$s and health effects have been observed at surprisingly low concentrations with no threshold (23,24). The smallest particulates, particularly the ultrafine particulates (PM_1) are highly chemically reactive, a property of their small size and large surface area (25). 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 (26) 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.

As incinerators are effectively particulate generators and produce predominately the smaller particulates that have the biggest 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 (27) and with lung cancer (19,28).

Two large cohort studies in the USA showed increasing mortality with increasing levels of $PM_{2.5}$ pollution. In the Six City Study published in 1993 (19), 8,111 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 to the least. This related to a $PM_{2.5}$ difference of 18.6mcg/m^3: cardiopulmonary mortality was increased by 37% and lung cancer mortality was also 37% higher.

In the American Cancer Society study (20), 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 mcg/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 (29) 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 (30).

The health benefits of bringing in these new regulations have been estimated as $32 billion annually (31) 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 $120 to $193 billion over the last 10 years (see section 9.1). As this study looked at only three health indicators it is likely to underestimate the true benefits.

It follows from this 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 (28). 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 RRs 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 RR of 1.14. The authors reported that exposure to a 10mcg/m^3 higher level of $PM_{2.5}$s was associated with a 14% increase in lung cancer and a 9% increase in cardiopulmonary disease (28).

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 (30). When the causes of death in the Cancer Prevention II Study were looked at in more detail (32) to look for clues to possible pathophysiological mechanisms, the link was strongest with ischaemic heart disease: a 10mcg/m^3 increase in $PM_{2.5}$s 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 $PM_{2.5}$s were 20-25mcg/m^3 higher (33). Particulates also increased mortality from stroke (34,35). One study concluded that 11% of strokes could be attributed to outdoor air pollution (36). Episodes of increased particulate pollution also increased admissions with heart disease (37). Mortality from diabetes (27) and admissions for diabetic heart disease were also increased (38) and these were double the non-diabetic CHD admissions, suggesting that diabetics were particularly vulnerable to the effect of particulate pollution (38). Higher levels of particulates have been associated with life-threatening arrhythmias (39) exercise-induced ischaemia (40), excess mortality from heart failure (35,41) and thrombotic disease (35).

d) Effect on Children and the Foetus

Particulates carry various chemicals including polycyclic aromatic hydrocarbons (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 (42). 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 (43). In addition further studies have shown an adverse effect on foetal development at levels currently found in cities today, such as New York (44). Air pollution has been found to cause irreversible genetic mutations in mice. Researchers found, in contrast, that if mice breathed air which had been freed of particulates by filtration they developed only background levels of genetic mutations,

confirming that particulates were causative (45). 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 could be attributed to air pollution (46).

e) Acute Respiratory Incidents

Elevated particulate air pollution has been associated with increased hospital admissions with asthma (24) and with COPD (47), increases in respiratory symptoms (48,49), higher incidence of asthma (50), reduced immunity (51,52), higher rates of ear, nose and throat infection (50), loss of time from school in children through respiratory disease (53,54), and declines of respiratory function (55-57). A sad aside to the above is that children who did more outdoor sport had greater declines in respiratory function (57). 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 (19,20,27,28,35,41,58) and increased respiratory mortality (41,42). 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 (19,20,28) (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 10mcg/m^3 rise in PM_{10}s (59) for acute exposures and a 3.5% rise for chronic exposures (31). For $PM_{2.5}$s the increase in mortality is much greater, especially for cardiopulmonary mortality (see Table).

Table 1 Cardiopulmonary Mortality and Fine Particulate Pollution

Study	Reference & Year	No of Participants	FU	Adjusted excess c/p mortality	Difference in $PM_{2..5}$s in mcg/m^3	Adjusted excess c/p mortality for rise of 10mcg/m^3
Six Cities	19 1993	8,111	1974-1991	37%	18.6	19.8%
ACS Cancer Prevention II	20 1995	552,138	1982-1989	31%	24.5	12.7%
Cancer Prevention II	28 2002	500,000	1982-1998	9%	10	9%

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 (30). 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 mcg/m^3 difference in $PM_{2.5}$ and

in the ACS study between 33% and 47% for a 24.5mcg/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 primarily due to an 18% increase in deaths from ischaemic heart disease for each 10mcg/m^3 rise in $PM_{2.5}$s (32).

Incinerators selectively emit smaller particulates and cause a greater effect on levels of $PM_{2.5}$s than PM_{10}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 World Health Organisation Air Quality Guidelines (60) we have estimated that a 1mcg/m^3 increase in $PM_{2..5}$ 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 10mcg/m^3 increase in $PM_{2.5}$ 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 $PM_{2.5}$ 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*" (Schwartz)(59) 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*" (58).

h) Summary

In summary there is now robust scientific evidence on the dangers to health of $PM_{2.5}$ 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 $PM_{2.5}$ particulates. The data makes it quite clear that attempts should be made to the reduce levels of these particulates whenever possible. However $PM_{2.5}$s are not the only reasons to be concerned about incinerators. There are other dangers:-

3.2 Heavy Metals

Pope 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. 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. (61,62). 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, have been shown to mediate cardiopulmonary injury in rats (63) and small amounts of metal (<1%) in particulates are known to cause pulmonary toxicity (64).

Emissions and ash from incinerators contain over 35 metals (65). Several are known or suspected carcinogens. Toxic metals accumulate in the body with increasing age (66). 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 (67-69), learning disabilities and hyperactivity (70,71).

Inhalation of heavy metals such as nickel, beryllium, chromium, cadmium and arsenic increases the risk of lung cancer (12)**.** Cumulative exposure to cadmium has been correlated with lung cancer (72). Supportive evidence comes from Blot and Fraumeni who found an excess of lung cancer in US counties where there was smelting and refining of non-ferrous metals (73). Inhaled cadmium also correlates with ischaemic heart disease (74).

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 (75), dyslexia (76), impulsive behaviour (77) attention deficit and hyperactivity disorder (ADHD) (78,79) as well as learning difficulties (14,70,80-83), lowered intelligence (79) and delinquency (84,79), although not every study reaches standard significance levels. Many of these problems were noted in the study of the population round the Sint Niklaas incinerator (85). Exposed adults have also been shown to be affected, showing higher levels of violence (13,86), dementia (87-93) and depression than non-exposed individuals. Heavy metal toxicity has also been implicated in Parkinson's disease (94).

Heavy metals emitted from incinerators are usually monitored at 3 to 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 on the lung but also on the spleen, liver and blood in animal studies. Both reversible and irreversible effects on the lung 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 mcg/m^3 increase in nitrogen dioxide. Studies in Japan showed a higher incidence of asthma with increasing NO_2 levels and that it synergistically increases lung cancer mortality rates (40). It has also been reported to aid the spread of tumours (95,96). Increases in NO_2 have been associated with rises in admissions with COPD (97), asthma in children and in heart disease in those over 65

(18). Other studies have shown increases in asthma admissions (98) and increased mortality with rising NO_2 levels (99).

Rising ozone levels have led to increasing hospital admissions, asthma and respiratory inflammation and have been reported to lower immunity (100). Higher levels have been significantly associated with increased mortality (101) and with cardiovascular disease. Both ozone and nitrogen dioxide are associated with increasing admissions with COPD (97).

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 polycyclic aromatic hydrocarbons (PAHs), brominated flame retardants, polychlorinated biphenols (PCBs), dioxins, polychlorinated dibenzofurans (furans). These substances are lipophilic and accumulate in fatty tissue and remain active in the living organisms and the environment for many years. They have been linked with early puberty (102), endometriosis (103), breast cancer (104,105), reduced sperm counts (106) and other disorders of male reproductive tissues (107), testicular cancer (108) and thyroid disruption (11). It has been claimed that about 10% of man-made chemicals are carcinogenic (see section 5.1), and many are now recognised as endocrine disrupters. Most of these health effects were not anticipated and are only now being recognised. 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 (65) 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 (65).

3.5 Effects on Genetic Material

Both 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 which can be passed on through several generations is not just a horrifying scenario but has been demonstrated to occur in animals (109,110). Incinerator emissions would greatly increase this risk.

DNA adducts to PAHs increase with exposure to pollution and patients with lung cancer have high 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.(111-113) 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 immunity in dolphins in the USA dropped as PCBs and DDT increased in their blood (114). The immune system appeared 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 (115) and accidental exposure data on humans has shown immunotoxicity with PBBs, dioxins and aldicarb. In fact whole volumes have been written on immunotoxicity (116). Note 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 (117). 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 (118).

In summary there is evidence that a large number of the pollutants emitted by incinerators can cause damage to the immune system (119). As is demonstrated in the next 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 stated “synergistic effects among chemical pollutants are more often present than not” (120). 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 1,000 toxic chemicals in unique combinations of three would require 166 million different experiments and even this would disregard varying doses (121).

Synergy has been demonstrated when organic chemicals are combined with heavy metals (122,123), and with combinations of pesticides (124, 125) and food additives (126). 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 two weeks. In this case the chemicals appeared to amplify each other’s toxicity in logarithmic fashion. In a recent experiment scientists dosed animals with a mixture of 16 organochlorine pesticides, lead and cadmium at “safe levels” and found they developed impaired immune responses, altered thyroid function and

altered brain development (127). Another study in 1996, published in Science, reported on the dangers of combinations of pesticides and their ability to mimic oestrogen. They found that combinations could increase the toxicity by 500 to 1000 times (128). 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 round an incinerator is being exposed to multiple chemical carcinogens, and to $PM_{2.5}$s, to carcinogenic heavy metals (in particular cadmium) and in some cases to 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 round 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 two days of exposure (129). 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 of 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 since 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 (85). 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 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. published a major study (130) in which they compared the numbers of registered cancer cases within 3 km and within 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 3km 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 (130,131), 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 to 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. looked at the data from 22,458 children who died of cancer between 1953 and 1980 in the UK (132). 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 (132). 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 (133). Of the 9,224 children for whom they had found accurate birth and death addresses, 4,385 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 round hospital incinerators but not landfill sites. This is strong evidence that the incinerators' emissions contributed to children's cancer deaths.

Biggeri et al. in 1996 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 (134).

Using a spatial scan statistic, Viel et al 2000 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 high emission of dioxin (135). They found highly significant clusters of soft tissue sarcoma (RR 1.44) and of non-Hodgkins lymphoma (RR 1.27) but no clusters of Hodgkins disease (used as negative control). This study was interesting in that it was designed to look both in a focussed way at the

area round the incinerator, and to check the association by looking for space time relationships which should be present if the relationship was causal. In addition they looked in an unfocussed way for other clusters in the wider area which contained other areas of deprivation. Both the first two analyses were positive close to the incinerator - demonstrating that a causal relationship was likely - and since no other clusters were found they concluded that deprivation could be virtually excluded as a factor.

According to Ohta et al, 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 1985-95 in the area up to 1.2 km to leeward of an incinerator (built in 1971) were due to cancer, compared to 20% further away and 25% overall in the local prefecture (136). Their data on soil contamination reinforced the importance of considering wind directions in evaluating the health effects of incinerators.

In 1989 Gustavsson reported a twofold increase in lung cancer in incinerator workers in Sweden compared to the expected local rate (137). In 1993 he reported a 1.5 fold increase in oesophageal cancer in combustion workers, including those working in incinerators (138).

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 to leeward of the incinerator (85). Orofacial defects and other midline defects were found to be more than doubled near an incinerator in Zeeburg, Amsterdam (139). Most of these deformed babies were born in an area corresponding to wind-flow from the incinerator and other defects included hypospadius and spina bifida. In the Neerland area, Belgium, there was a 26% increase in congenital anomalies in an area situated between two incinerators (140). A study of incinerators in France has shown chromosomal defects and other major anomalies (facial clefts, megacolon, renal dysplasias) (141). 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 (142). The study pointed out that the figures for birth defects are likely to be substantial underestimates since 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 (143-146). 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.*"(147)

4.3 Ischaemic Heart Disease

Gustavsson found an excess of ischaemic heart disease (137) 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 round 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 unrecognised 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 onward (although children's cancers occurred earlier), and in Japan, Ohta noted that cancer caused 42% of all deaths in the lee of incinerators from 14 to 24 years after the incinerator was commissioned (136). 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. Since 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 means 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 of 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 look at other studies which 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 (148). Put another way, the chance of dying from cancer at the turn of the century was 1 in 33. It is now 1 in 4. WHO data has demonstrated that 80% of cancers are due to environmental influences (149) and evidence from migrant studies confirm that it is the environment rather than the genes that determine the cancer risk. (149).

Many people have noted that the rise in cancer has paralleled the rise in synthetic chemicals. These chemicals have doubled in quantity every 7 to 8 years with a 100 fold increase over the last 2 generations (150). 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 industrialised countries with 50% of cases in the industrialised 20% of the world (151) and the WHO has noted that cancer incidence rises with the GNP of a country.
- There is the same correlation within countries. The highest mortality from cancer in the USA is in areas of highest industrialised activity. There is also a correlation in the USA between cancer incidence and the number of waste sites in the county (152,153). Counties with facilities for treating toxic waste have four times as much breast cancer (154). Cancer is also commoner in counties with chemical industries (155). Public Data Access in the USA shows a close correlation between cancer mortality and environmental contamination (156).
- Numerous studies have shown higher cancer incidence in both industrial workers and in populations living in polluted areas (157, 158).
- 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) (159,160).

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 (161). Dogs have been found to have higher rates of bladder cancer in industrialised counties in the USA (162). 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 (163).

c) Large increases in cancer in certain tissues

Steep rises in cancer have occurred in tissues directly exposed to the environment: the lung and skin. But some of the steepest rises have occurred in parts of the body with high fat content. This including cancers of the brain, breast, bone marrow and liver. This again points to toxic chemicals which 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 Centre 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 (164,165). 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 (42).

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 (166,167) and the Upper Cape Study showing that tetrachloroethylene in the water was associated with elevated rates of several types of cancer (168-170). 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 (171), increases in lymphoma in counties in Iowa where drinking water was contaminated with dieldrin (172), elevated levels of leukaemia in children at Woburn, Massachusetts coinciding with a known period of water contamination with chlorinated solvents (173), a cancer cluster linked to consumption of river water contaminated by industrial and agricultural chemicals in Bynum, North Carolina (174) and high rates of non-Hodgkin's lymphoma where water was contaminated with chlorophenols in Finland (175).

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 showed that inhalation of ambient level nitrogen dioxide, or polluted urban ambient air, facilitated blood-borne cancer cell metastasis. (95).

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 to

10% are probable carcinogens. The International Agency for Cancer Research tested 1000 chemicals in 1993 and found that 110 were probable carcinogens (176). The National Toxicity Program tested 400 chemicals in 1995 and found that 5-10% were carcinogenic (177). Only 200 of the 75,000 synthetic chemicals in existence are regulated as carcinogens whereas, on this data, between 3,000 and 7,500 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 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. The evidence for this is as follows:-

In a study, a group of middle aged Americans were found to have 177 organochlorine residues in their bodies (178,179). 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 (180). 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 (181). 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 (182). 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 (183). 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 which

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 has 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 (184). The increase in Alzheimer's disease was found in almost all developed countries, and rises varied across countries from 20% (which was defined as substantial) to 1200%. The paper suggested environmental factors were likely to be 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% (185). It is very likely that these diseases have aetiological factors in common.

Heavy metal exposure is known to correlate with both Parkinson's disease (94,186) and Alzheimer's disease (67,68,88-92). 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 which are toxic to the brain and nervous system (180). It is crucial to look at every possible way to prevent Alzheimer's because of its huge care costs (US figures are $60 billion annually) and because of 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 inpatient admissions with organic brain syndrome, schizophrenia, major affective disorders, neurosis, behavioural disorder of childhood and adolescence, personality disorder and alcoholism (187). Increases in the total number of psychiatric emergency room visits and in schizophrenia (188) have been noted on days when air pollution has been high. Depression has also been linked to inhaled pollutants (189,190). Clearly something very profound occurs when we pollute the air.

5.4 Violence and Crime

An increasing number of studies, including studies of murderers (191), case-control and correlation studies (13, 86,192,193) and prospective studies (84,194) have shown links between violence and heavy metals and these include lead, cadmium and manganese. The majority of the studies have 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 (195) found early lead exposure was one of the most important predictors of disciplinary problems from ages 13 to 14, delinquency from ages 7 to 17 and adult criminal offences, from ages 18 to 22.

Stretesky found an association between air lead levels and murder rates in US counties (196). 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 (197). 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

.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. Firstly 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. Secondly 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 (198). Other factors that increase foetal susceptibility are higher rates of cell proliferation, lower immunological competence and decreased capacity to detoxify carcinogens and repair DNA (199).

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 (200) 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 miniscule 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 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 (11). A single exposure is enough and timing is critical (201). Small doses of oestrogenic chemicals can alter sexual development of the brain and the endocrine system (202).

It is estimated that 5% of babies born in the USA have been exposed to sufficient pollutants to affect neurological development (203). 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 (204). 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 (205). 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 (66).

In July 2005, in a ground-breaking study (206), 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 WWF and Greenpeace, on the foetal body burden. They tested for the presence of 35 chemicals in the umbilical cord blood of newborns (207). 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, bio-accumulative 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 (208). In the USA studies of human breast milk have shown that 90% of samples contained a disturbing 350 chemicals. This was higher in industrialised areas showing that inhalation of these toxic substances is an important factor (209). The toxic dose taken in by a breast-feeding baby is 50 times higher than that taken in by an adult (210).

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 (211). From 1979 one in four samples of breast milk have been found to be over the legal limit set for PCBs in commercial feeds (205) and these are known to impair intellectual development (212-214). Contamination with persistent organic pollutants (POPs) in breast milk in animals has consistently shown structural, behavioural and functional problems in their offspring (215). For instance, in monkeys it has shown that it decreases their ability to learn (216-218). Polybrominated diphenyl ethers (PBDEs) are toxic chemicals which have been doubling in breast milk every five 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 (219, 220). 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 (221-223). 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 which 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 (224). This is supported by animal studies which have already confirmed that cancer can be initiated by giving carcinogens before conception, in utero or directly to the neonate (225, 226).

Developing systems are very delicate and in many instances are not able to repair damage done by environmental toxicants (227). 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 (228). 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 recognised 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 which 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 (229). 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 (85) 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 need also to consider subclinical toxicity. The pioneering work of Herbert Needleman showed that lead could cause decreases in intelligence and alteration of behaviour in the absence of clinically visible signs of toxicity (82). This has also been shown to be the case with PCBs (230) and methyl mercury (71). 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 5 point drop of 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 (117), 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 (232) and lead (83). It has been demonstrated that there is a tenfold difference between different individuals in the metabolism of the carcinogenic PAH benz(a)pyrene (233).

Ashford and Miller also noted that studies in both toxicology and epidemiology have recognised 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 to 30% in several surveys with 5% having daily symptoms) (117). Research has shown 150 to 450 fold variability in response to airborne particles (234). Friedman has stated that environmental regulation requires the protection of these sensitive individuals (235). This highlights the dangers of incinerators which emit a multitude of chemical compounds. Chemical sensitivity is typically triggered by an acute exposure after which symptoms start to occur at very low levels of exposure (117). 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 sensitisation. Certain susceptible individuals will be highly affected by these pollutants and these effects will be difficult to anticipate. In addition, people affected 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 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 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.

.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 synthesised 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 and this exceeded the worst case scenario predicted by scientists.
- **Polychlorinated biphenyls (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 in 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 seventies scientists noted 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 be tributyl tin, a chemical added to paint to stop barnacles growing. Incredibly the damage was occurring at concentration of just five parts per trillion. By the end of the eighties more than one hundred species of fish were known to have been harmed.

This pattern of unanticipated disasters and long latent intervals before their discovery characterises 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 (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 air, the high volume of ash that needs landfilling and the very

toxic nature of the fly ash would rule it out. Similarly pyrollysis 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, Anaerobic Digestion (which can be a part of the above) and types of Gasification that produce no ash.

8.1 Mechanical Biological Treatment (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 partly being met. It fails on two counts only. Firstly 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 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 and more importantly this type of gasification 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 recycled 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 show that recycling saves about 3 to 5 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 mechanical biological treatment (MBT) with low environmental or 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 EC 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 Environmental Protection Agency again showed that every dollar spent on abatement saved 10 dollars 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 hospitalisations, 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. 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 (242).

The WWF 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 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 to 30 year contracts creating problems for the future. Incinerators produce two types of ash, bottom ash and fly ash (sometimes called air pollution control (APC) 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 (3). 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 EU Commission have 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, toxic ash laden with dioxins was spread over allotments, bridle paths and footpaths for six years.

9.3 Radioactivity

Over thirty 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 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 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 incidence of leukaemias and cancers around sites releasing radioactive material are well documented. At Seascale a public health enquiry found children were more than ten 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 kilometres 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 five years after Krummel was commissioned. At Dounreay there was a sixfold increase in children's leukaemia (246) and at Aldermaston there was also an increase in leukaemias in the under fives (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 ten years earlier where 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, that was 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 bio-accumulate. 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 here 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 centre of Biology of Natural Systems in Queen's College, New York, Dr Commoner and his team used a computer programme 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 (SLF) and recycled liquid fuel (RLF).

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.

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 practise 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 10mg/m^3. However cement kilns are allowed to emit up to 50mg/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 which 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 since 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. 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 persistent organic pollutants (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. 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 to 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 recognised, 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 account of 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 2,500 times lower than American EPAs lowest observed effect, with adverse outcomes including aggressive behaviour, early puberty and abnormal breast growth (180). Perchlorate produces changes in the size of parts of the brain at 0.01 mg/kg/day but not at 30mg (180). Aldicarb was found to suppress the immune system more at 1 ppb than it did at 1000ppb. Other chemicals also produce different effects at low dose to what they do at high dose. 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 two year period, documented in Greenpeace's "A Review of the Performance of Municipal Incinerators in the UK". 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. This data was 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 6,000 times in two 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 (254), particularly in relationship to birth and developmental defects. This type of assessment contains a value judgement about what is an acceptable level of risk (255). 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 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 (256). In spite of these severe limitations it is extensively used.

These risks assessments have almost always concluded that incinerators are safe which flies in the face of epidemiological data which shows 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 which were later found to have devastating and unanticipated effects, often beyond the worst case scenario (eg 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 persistent organic pollutants, including PCB, dioxins and furans. It identifies incinerators as 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 of *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 which 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 disruptors, 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% and gives rise to large quantities of highly toxic fly ash (air pollution control residues) which pose important 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. 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 new-born babies.
7) Waste incineration is prohibitively *expensive* when health costs are taken into consideration. The EC 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 ten 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, mechanical biological treatment and plasma gasification: a combination of these would be both safer and cheaper than incineration in the long run, much cheaper when the health costs were 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. [NB. Presently 9 out of 14 incinerators have been built in the most deprived 20% of wards (257)].

8) This report outlines the many deficiencies of 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.

References:

1) EC (1998) Proposal for a Council Directive on the incineration of waste. Brussels 07.10.1998 COM (1999) 558final. 98/0289 (SYN).
2) Howard C V (2000) *In* Health Impacts of Waste Management Policies. Hippocrates Foundation, Kos, Greece 12-14 Nov 1998. Academic Publishers.
3) Personal communication, Peter Rossiter BSc (Hon), MRSC, Chemical Consultant. 2005.
4) Espinosa AJ, Rodriquez MT, Barragan de la Rosa FJ et al. Size distribution of metals in urban aerosols in Seville (Spain). Atmos Environ 2001; 35: 2595-2601.
5) Baek SO, Field RA, Goldstone ME et al. A review of atmospheric polycyclic aromatic hydrocarbons: sources, fate and behaviour. Water, Air Soil Pollution, 1991; 60: 279-300.
6) Pistikopoulos P, Mascelet P, Mouvier G. A receptor model adapted to reactive species – polycyclic aromatic hydrocarbons - evaluation of source contributions in an open urban site. Atmos Environ A-Gen 1990; 24: 1189-97.
7) Venkataraman C, Friedlander SK. Source resolution of fine particulate polycyclic aromatic hydrocarbons – using a receptor model modified for reactivity. J Air Waste Management; 1994; 44: 1103-08.
8) Zmirou D, Masclet P, Boudet C, Dechenaux J. Personal exposure to atmospheric polycyclic hydrocarbons in a general adult population and lung cancer assessment. J Occup Environ Med 2000; 42(2): 121-6.
9) Kerkvliet NI. Immunotoxicology of dioxins and related compounds. *In* Schecter, Dioxins and Health p 199-225.
10) Whyatt RM, Santella RM, Jedrychowski W et al. Relationship between ambient air pollution and DNA damage in Polish mothers and newborns. Environ Health Perspect, 1998; 106 Suppl 3: 821-6
11) Porterfield SP. Vulnerability of the developing brain to thyroid abnormalities and environmental insults to the thyroid system. Environ Health Perspect 1994; 102 Supp 2: 125-30.
12) Peters JM, Thomas D, Falk H et al. Contribution of metals to respiratory cancer. Environ Health Perspect 1986;70: 71-83.
13) Gottscalk LA, Rebello T, Buchsbaum MS et al. Abnormalities in hair trace elements as indicators of aberrant behaviour. Comp Pyschiatry 1991; 32 (3): 229-37.
14) Tong S, Baghurst P, McMichael A et al. Lifetime exposure to environmental lead and children's intelligence at 11 – 13 years: the Port Pirie Cohort Study. BMJ 1996; 312 (7046): 1569-75.
15) Sedman RM, Esparza JR. Evaluation of the public health risks associated with semivolatile metal and dioxin emissions from hazardous waste incinerators. Environ Health Perspect 1991; 94: 181-7.
16) Ericksson P, Jakobsson E, Fredriksson A. Brominated flame retardants: A novel class of developmental neurotoxicants in our environment? Environ Health Perspect, 2001; 109(1): 903-908.
17) Olsson P-E, Borg B, Brunstrom B, Hakansson H, Klasson-Wehler E. Endocrine disrupting substances. ISBN 91-620-4859-7, Swedish EPA, Stockholm 1998.
18) WHO Air Quality Guidelines, 1999, Chapter 3.
19) Dockery DW, Pope Ca 3rd, Xu X et al. An association between air pollution and mortality in six US cities. N Eng J Med 1993; 329(24): 1753-9.
20) Pope CA, Thun MJ, Namboodiri MM et al. Particulate air pollution as a predictor of mortality in a prospective study of US adults. Am J Respir Crit Care Med 1995; 151 (3 pt 1): 669-74.
21) de Hartog JJ, Hoek G, Peters A, et al. Effects of fine and ultrafine particles on cardiorespiratory symptoms in elderly subjects with coronary heart disease: the ULTRA Study. Am J Epidemiology 2003; 157(7): 613-23.
22) Nemmar A, Hoet PH, Vanquickenborne B et al. Passage of inhaled particles into the blood circulation in humans. Circulation 2002; 105(4): 411-4.
23) Maynard RL, Howard CV, Air Pollution and Health, London: Academic Press 1999: 673-705.
24) Ponka A, Virtanen M. Asthma and air pollution in Helsinki. J Epidemiol Community Health 1996; 50 Suppl 1: s59-62.
25) Particulate Matter: Properties and Effects upon Health, BIOS Scientific Publishers Ltd, Oxford p 63-84.
26) Airborne Particulate Matter in the United Kingdom. Third Report of the Quality of Air Review Group (QUARG) May 1996, ISBN 0 9520771 3 2.
27) Goldberg MS, Burnett RT, Bailar JC et al. The association between daily mortality and ambient air particle pollution in Montreal, Quebec. 2. Cause-specific mortality. Environ Res 2001: 86(1): 26-36.
28) Pope CA, Burnett RT, Thun MJ, et al. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA 2002; 287(9): 1132-41.
29) Whitman v American Trucking Assoc Inc 532 US 457 (2001).
30) Re-analysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality: Special Report. Cambridge, Mass: Health Effects Institute July 2000, led Dr Daniel Kreweski.

31) Ostro B, Chestnut L. Assessing the benefits of reducing particulate matter and pollution in the United States. Environ Res 1998; 76(2): 94-106.
32) Pope CA, Bumett RT, Thurston GD et al. Cardiovascular Mortality and Long-Term Exposure to Particulate Air Pollution: Epidemiological Evidence of General Pathophysiological Pathways of Disease. Circulation 2004; 109: 71-77.
33) Peters A, Dockery DW, Muller JE et al. Increased particulate air pollution and the triggering of myocardial infarction. Circulation 2001; 103 (23): 2810-5.
34) Hong YC, Lee JT, Kim H, Kwon HJ. Air pollution: a new risk factor in ischemic stroke mortality. Stroke 2002; 33(9): 2165-9.
35) Hoek G, Brunekreef B, Fischer P et al. The association between air pollution and heart failure, arrhythmia, embolism, thrombosis and other cardiovascular causes of death in a time series. Epidemiology 2001; 12(3): 355-7.
36) Maheswaran R, Haining RP, Brindley P et al. Outdoor air pollution and Stroke in Sheffield, United Kingdom, Small-Area Geographical Study. Stroke 2005; 36(2): 239-43.
37) Schwartz J. Air pollution and hospital admissions for heart disease in eight US counties. Epidemiology 1999; 10(1): 17-22.
38) Zanobetti A, Schwartz J. Cardiovascular damage by airborne particles: are diabetics more susceptible? Epidemiology 2002; 13(5): 588-92.
39) Peters A, Liu E, Verrier RL et al. Air pollution and incidence of cardiac arrhythmia. Epidemiology 2000; 11(1): 11-7.
40) Pekkanen J, Peters A, Hoek G, et al. Particulate air pollution and risk of ST segment depression during submaximal exercise tests among subjects with coronary heart disease: the Exposure and Risk Assessment for Fine and Ultrafine Particles in Ambient Air (ULTRA) study. Circulation 2002; 106: 933-38.
41) Goldberg MS, Burnett RT, Bailar JC 3rd et al. Identification of persons with cardiorespiratory conditions who are at risk of dying from the acute effects of ambient air particles. Environ Health Perspect 2001; 109 Supp 4: 487-94.
42) Perera FP, Tang D, Tu YH et al. Biomarkers in maternal and newborn blood indicate heightened fetal susceptibility to procarcinogenic DNA damage. Environ Health Perspect 2004; 112(10): 1133-6.
43) Jedrychowski W, Bendkowska I, Flak E et al. Estimated risk for altered fetal growth resulting from exposure to fine particles during pregnancy: an epidemiologic prospective cohort study in Poland. Environ Health Perspect 2004; 112(14): 1398-1402.
44) Perera FP, Rauh V, Whyatt RM et al. Molecular evidence of an interaction between prenatal environmental exposures and birth outcomes in a multiethnic population. Environ Health Perspect 2004; 112(5): 626-30.
45) Somers CM, McCarry BE, Malek F et al. Reduction of particulate air pollution lowers the risk of heritable mutations in mice. Science 2004; 304(5673): 1008-10.
46) Burden of disease attributable to selected environmental factors and injury among children and adolescents in Europe (no authors listed). Child Care Health Dev 2004; 30(6): 731-732.
47) Morgan G, Corbett S, Wlodarczyk J. Air pollution and hospital admissions in Sydney, Australia, 1990-1994. Am J Public Health 1998; 88(12): 1761-60.
48) Vichit-Vadakan N, Ostro BD, Chestnut LG et al. Air pollution and respiratory symptoms: result from three panel studies in Bangkok, Thailand. Environ Health Perspect 2001; 109 Supp3: 381-7.
49) Dockery DW, Speizer FE, Stram DO et al. Effects of inhalable particles on respiratory health of children. Am Rev Respir Dis 1989; 139(3): 587-94.
50) Brauer M, Hoek G Van Vliet P et al, Air pollution from traffic and the development of respiratory infections and asthmatic and allergic symptoms in children. Am J Respir Crit Care 2002; 166(8): 1092-8.
51) Seaton A, MacNee W, Donaldson K et al. Particulate air pollution and acute health effects. Lancet 1995; 345(8943): 176-8.
52) Boezen HM, van der Zee SC, Postma DS et al. Effects of ambient air pollution on upper and lower respiratory symptoms and peak expiratory flow in children. Lancet 1999; 353 (9156): 874-8.
53) Gilliland FD, Berhane K, Rappaport EB et al. The effects of ambient air pollution on school absenteeism due to respiratory illness. Epidemiology 2001: 12(1): 43-54.
54) Peters A, Dockery DW, Heinrich J, Wichmann HE. Short term effects of particulate air pollution on respiratory morbidity in asthmatic children. Eur Respir J 1997; 10(4): 872-9.
55) Gauderman WJ, McConnell R, Gilliland F et al. Association between air pollution and lung function growth in Southern Californian children. Am J Respir Crit Care Med 2000; 162 (4 Pt 1); 1383-90.
56) Brunekreef B, Hoek G. The relationship between low-level air pollution and short-term changes in lung function in Dutch children. J Expo Anal Environ Epidemiol 1993; 3 Suppl 1: 117-28.

57) Gauderman WJ, Gilliland GF, Vora H, et al. Association between air pollution and lung function growth in Southern Californian children: results from a second cohort. Am J Respir Crit Care Med 2002; 166(1): 76-84.
58) Samet JM, Dominici F, Curriero FC et al. Fine particulate air pollution and mortality in 20 US cities 1987-1994. N Eng J Med 2000; 343(24): 1742-9.
59) Schwartz J, Laden F, Zanobetti A. The concentration-response relation between PM2.5 and daily deaths. Environ Health Perspect 2002; 110(10): 1025-9.
60) Air Quality Guidelines for Europe, Section 7.3 p19, Second Edition, World Regional Publications, Regional European Series No 91, World Health Organisation, Regional Office for Europe, Copenhagen.
61) Proceedings of the Third Colloquium on Particulate Air Pollution and Human Health 6-8 June 1999, Durham, North Carolina, Irvine, CA: Air Pollution Effects Laboratory, University of California, 1999, 11/23.
62) Pope CA 3rd. Respiratory disease associated with community air pollution and a steel mill, Utah Valley. Am J Public Health, 1989, 79(5): 623-8.
63) Costa DL, Dreher KL. Bioavailable transition metals in particulate matter mediate cardiopulmonary injury in healthy and compromised animal models. Environ Health Perspect 1997;105 (suppl 5): 1053-60.
64) Dye JA, Lehmann JR, McGee JK et al. Acute pulmonary toxicity of particulate matter filter extracts in rats: Coherence with epidemiologic studies in Utah Valley.
Environ Health Perspect 2001; 109 Suppl 3: 395-403.
65) Rowat SC. Incinerator toxic emissions: a brief summary of human health effects with a note on regulatory control. Med Hypotheses 1999; 52(5): 389-96.
66) Casdorph R, Walker M. Toxic Metal Syndrome, New York: Avery Publishing Group 1995.
67) Ehmann WD, Markesbery WR, Alauddin M et al. Brain trace elements in Alzheimer's disease. Neurotoxicology 1986; 7 (1): 195-206.
68) Thompson CM, Markesbery WR, Ehmann WD et al. Regional trace-element studies in Alzheimer's disease. Neurotoxicology 1988; 9(1): 1-7.
69) Wenstrup D, Ehmann WD, Markesbery WR. Trace element imbalances in isolated subcellular fractions of Alzheimer's disease brains. Brain Res 1990; 533(1): 125-31.
70) Schettler T. Toxic threats to neurological development of children. Environ Health Perspect 2001; 109 (Suppl 6): 813-6.
71) Grandjean P, Weihe P, White RF et al. Cognitive deficit in 7-year old children with prenatal exposure to methyl mercury. Neurotoxicol Teratol 1997; 19(6): 417-28.
72) Thun MJ, Schnorr TM, Smith AB, et al. Mortality among a cohort of US cadmium production workers – an update. J Natl Cancer Inst 1985; 74(2): 325-33.
73) Blot WJ, Fraumeni JF Jnr. Arsenical air pollution and lung cancer. Lancet 1975; 2 (7926):142-4.
74) Severs R, Whitehead L, Lane R. Air quality correlates of chronic disease mortality: Harris County, Texas 1969-71. Tex Rep Biol Med 1978; 36: 169-84.
75) Wecker L, Miller SB, Cochran SR et al. Trace element concentration in hair from Autistic Children. J Ment Defic Res 1985: 29 (pt 1): 15-22.
76) Capel ID, Pinnock MH, Dorrell HM, et al. Comparison of concentrations of some trace, bulk, and toxic metals in the hair of normal and dyslexic children. Clinic Chem 1981: 27(6): 879-81.
77) Brockel BJ, Cory-Slechta DA. Lead, attention, and impulsive behaviour: changes in a fixed waiting-for-reward paradigm. Pharmacol Biochem Behav 1998: 60(2): 545-52.
78) David OJ, Hoffman SP, Sverd J, et al. Lead and hyperactivity: Behavioural response to chelation. Am J Psych 1976; 133(10): 1155-8.
79) Masters RD. Biology and politics: linking nature with nurture. Ann Rev Polit Sci 2001; 4: 345-65.
80) Leviton A, Bellinger D, Allred EN et al. Pre and postnatal low-level lead exposure and children's dysfunction in school. Environ Res 1993: 60(1): 30-43.
81) Eppright TD, Sanfacon JA, Horwitz FA. Attention deficit hyperactivity disorder, infantile autism and elevated blood lead: a possible relationship. Mol Med 1996: 93(3): 136-8.
82) Needleman HL, Gunnoe C, Leviton A et al. Deficits in psychologic and classroom performance in children with elevated dentine lead levels. N Eng J Med 1994; 331(13): 689-95.
83) Bellinger D, Leviton A, Waternaux C, et al. Longitudinal analyses of prenatal and postnatal lead exposure and early cognitive development. N Eng J Med 1987; 316 (17): 1037-43.
84) Needleman HL, Riess JA, Tobin MJ, et al. Bone lead levels and delinquent behaviour. JAMA 1996; 275 (5); 363-9.
85) Mispelstraat: Living under the smoke of a waste incinerator. Report on the health impact of the MIWA waste incinerator in Sint Niklaas, Belgium. www.milieugezondheid.
86) Schauss AG. Comparative hair-mineral analysis results of 21 elements in a random selected behaviourally "normal" 19-59 year old population and violent adult criminal offenders. Int J Biosoc Res 1981; 1: 21-41.

87) Bowdler NC, Beasley DS, Fritze EC et al. Behavioural effects of aluminium ingestion on animal and human subjects. Pharmacol Biochem Behav 1979: 10(4): 502-12.
88) Trapp GA, Miner GH, Zimmerman RL et al. Aluminium levels in the brain in Alzheimer's disease. Biol Pyschiatry 1978; 13(6): 709-18.
89) Multhaup G. Amyloid precursor protein, copper and Alzheimer's disease. Biomed Pharmocother 1997: 51(3): 105-11.
90) Zapatero MD, Garcia de Jalon A, Pascual F, et al. Serum aluminium levels in Alzheimer's disease and other senile dementias. Biol Trace Elem Res 1995; 47 (1-3):
235-40.
91) Martyn CN, Barker DJ, Osmond C et al. Geographical relationship between Alzheimer's disease and aluminium in drinking water. Lancet 1989; 1(8763): 59-62.
92) Crapper DR, Krishnan SS, Dalton AJ et al. Brain aluminium distribution in Alzheimer's disease and experimental neurofibrillary degeneration. Science 1973: 180(85): 511-3.
93) Neri LC, Hewitt D. Alzheimer's disease and drinking water. Lancet 1991; 338 (8763): 390.
94) Zayed J, Ducic S, Campanella G, et al. Environmental factors in the etiology of Parkinson's disease. Can J Neurol Sci 1990: 17(3): 286-91.
95) Richters A, Richters V. A new relationship between air pollutant inhalation and cancer. Arch Environ Health 1983; 38(2): 69-75.
96) Ruaslahti E. How cancer spreads. Scientific American Sept 1996: 72-77.
97) Andersen HR, Spix C, Medina S, et al. Air pollution and daily admissions for chronic obstructive pulmonary disease in 6 European cities: results from the APHEA project. Eur Resp J 1997; 10(5): 1064-71.
98) Sunyer J, Spix C, Quenel P, et al. Urban air pollution and emergency admissions for asthma in four European cities: the APHEA Project. Thorax 1997; 52(9): 760-5.
99) Ostro BD, Broadwin R, Lipsett MJ. Coarse and fine particles and daily mortality in the Coachella Valley, California: a follow-up study. J Exp Anal Environ Epidemiol 2000; 10(5): 412-9.
100) Breslin K. The impact of ozone. Env Health Perspectives 1995; 103(7-8): 660-4.
101) Hoek G, Schwartz JD, Groot B, Eilers P. Effects of ambient particulate matter and ozone on daily mortality in Rotterdam, The Netherlands. Arch Environ Health 1997; 52(6): 455-63.
102) Den Hond E, Roels HA, Hoppenbrouwers K et al. Sexual maturation in relationship to polychlorinated aromatic hydrocarbons: Shape and Skakkebaek's hypothesis revisited. Environ Health Perspect 2002; 110(8): 771-6.
103) Eskenazi B, Mocarelli P, Warner M et al. Serum dioxin concentrations and endometriosis: a cohort study in Sevenso, Italy. Environ Health Perspect 2002; 110(7): 629-34.
104) Wolff MS, Weston A. Breast cancer risk and environmental exposures. Environ Health Perspect 1997; 105(Suppl 4): 891-6.
105) Hoyer AP, Granjean P, Jorgensen T et al. Organochlorine exposure and the risk of breast cancer. Lancet 1998; 352 (9143): 1816-20.
106) Oliva A, Spira A, Multigner L et al. Contribution of environmental factors to the risk of male infertility. Hum Reprod 2001; 16(8): 1768-76.
107) Sultan C, Balaguer P, Terouanne B et al. Environmental xenoestogens, antiandrogens and disorders of male sexual differentiation. Mol Cell Endocrinol 2001; 178 (1-2): 99-105.
108) Hardell L, van Bavel B, Lindstrom G et al. Increased concentrations of polychlorinated biphenyls, hexachlorobenzene and chlordanes in mothers of men with testicular cancer. Environ Health Perspect 2003; 111 (7): 930-4.
109) Tomatis L. Transplacental Carcinogenesis. Lyon, International Agency for Research on Cancer, IARC Scientific Publications No 4 pp100-111.
110) Tomatis L, Goodall CM. The occurrence of tumours in F1, F2 and F3 descendants of pregnant mice injected with 7,12 dimethylbenz(a)anthracene. Int J Cancer 1969; 4(2): 219-25.
111) Ross P, de Swart, Visser I, et al. Relative immunocompetence of the newborn harbor seal, Phoca vitulina. Veterinary Immunology and Immunopathology 1994; 42(3-4): 331-48.
112) Ross P, de Swart R, Reijnders P, et al. Contaminant-related suppression of delayed-type hypersensitivity and antibody responses in harbor seals fed herring from the Baltic Sea. Env Health Perspect 1995; 103 (2): 162-7.
113) De Swart R. Impaired immunity in seals exposed to bioaccumulated environmental contaminants, PhD Thesis, Erasmus University, Rotterdam, Netherlands, 1995.
114) Lahvis G, Wells RS, Kuehl DW et al. Decreased lymphocyte response in free-ranging bottle-nosed dolphins (Tursiops truncatus) are associated with increased concentration of PCBs and DDT in peripheral blood. Env Health Perspect 1995; 103(4): 67-72.
115) Cone JE,Harrison R, Reiter R. Patients with multiple chemical sensitivities: clinical diagnostic subsets among an occupational health clinic population. *In* Cullen M (ed) Workers with Multiple Chemical Sensitivities, Occupational Medicine: State of the Art Review 1987; 2(4):721-738 .

116) Sharma R. Immunological Considerations in Toxicology, Vols 1 and 2 (1981), CRC Press, Boca Raton, FL.
117) Ashford N, Miller C. Chemical Exposures: Low Levels and High Stakes. John Wiley & Sons 1998.
118) Massolo L, Muller A, Tueros M, et al. Assessment of mutagenicity and toxicity of different-size fractions of air particles from La Plata, Argentina, and Leipzig, Germany. Environ Toxicol 2002; 17 (3): 219-31.
119) Hillam RP, Bice DE, Hahn FF, Scnizelein CT. Effects of acute nitrogen dioxide exposure on cellular immunity after lung immunization. Environ Res 1983; 31(1): 201-11.
120) Carroll Wilson. Man's Global Impact on the Environment: A Study of Critical Environmental Problems, MIT Press, Cambridge, Mass 1971.
121) Mokhiber R. The Ecologist 1998; 28(2): 57-8.
122) Harrison PT, Heath JC. Apparent synergy in lung carcinogenesis: interactions between N-nitrosheptamethyleneimine, particulate cadmium and crocidolite asbestos fibres in rats. Carcinogenesis 1986; 7(11): 1903-8.
123) Wade MG, Foster WG, Younglai EV, et al. Effects of subchronic exposure to a complex mixture of persistent contaminants in male rats: systemic, immune and reproductive effects. Toxicol Sci 2002; 67(1): 131-43.
124) Soto AM, Chung KL, Sonnenschein C. The pesticides endosulphan, toxaphene and dieldrin have estrogenic effects on human estrogen-sensitive cells. Environ Health Perspect 1994; 102(4): 380-3.
125) Abou-Donia MB, Wilmarth KR, Jensen KF et al. Neurotoxicity resulting from co-exposure to pyridostigmine bromide, DEET and permethrin: Implications of Gulf War chemical exposures. J Toxicol Env Health 1996; 48(1): 35-56.
126) Ershoff BH. Synergistic toxicity of food additives in rats fed a diet low in dietary fibre. J Food Sci 1976; 41: 949-51.
127) Wade MG, Parent S, Finnson KW, et al. Thyroid Toxicity due to a subchronic exposure to a complex mixture of 16 organochlorines, lead, and cadmium. Toxicol Sci 2002; 67(2): 207-18.
128) Arnold SF, Klotz DM, Collins BM, et al. Synergistic activation of estrogen receptors with combinations of environmental chemicals. Science 1996; 272 (5267): 1489-92.
129) Li MH, Hansen LG. Enzyme induction and acute endocrine effects in prepubertal female rats receiving environmental PCB/PCDF/PCDD mixtures. Environ Health Perspect 1996; 104(7): 712-22.
130) Elliot P, Shaddick G, Kleinschmidt I etal, Cancer incidence near municipal solid waste incinerators in Great Britain. Brit J Cancer 1996; 73(5): 702-10.
131) Elliot P, Eaton N, Shaddick G et al. Cancer incidence near municipal waste incinerators in Great Britain. Part 2: Histopathological and case note review of primary liver cancer cases. British J Cancer 2000; 82(5): 1103-6.
132) Knox EG, Gilman EA. Migration patterns of children with cancer in Britain. J Epidemiology & Community Health 1998; 52(11): 716-26.
133) Knox EG. Childhood cancers, birthplaces, incinerators and landfill sites. Int J Epidemiology 2000; 29 (3): 391-7.
134): Biggeri A, Barbone F, Lagazio C, et al. Air pollution and lung cancer in Trieste, Italy: Spatial analysis of risk as a function of distance from sources. Environ Health Perspect 1996; 104 (7): 750-4.
135) Viel JF, Arveux P, Baverel J, et al. Soft tissue sarcoma and non Hodgkin's lymphoma clusters around municipal solid waste incinerators with high dioxin emission levels. Am J Epidemiology 2000; 152(1): 13-19.
136) Ohta S, Kuriyama S, Nakao et al. Levels of PCDDs, PCDFs and non-ortho coplanar PCBs in soil collected from high cancer-causing area close to batch-type municipal solid waste incinerator in Japan. Organohalogen Compounds 1997; 32: 155-60.
137) Gustavsson P. Mortality among workers at a municipal waste incinerator. Am J Ind Med 1989; 15(3): 245-53.
138) Gustavsson P, Evanoff B, Hogstedt C. Increased risk of esophageal cancer among workers exposed to combustion products. Archives Environ Med 1993; 48(4): 243-5.
139) ten Tusscher GW, Stam GA, Koppe JG. Open chemical combusting resulting in a localised increased incidence of orofacial clefts. Chemosphere 2000; 40(9-11): 1263-70.
140) Van Lorebeke N. Health effects of a household waste incinerator near Wilrijk, Belgium. *In* Health Impacts of Waste Management Policies, Hippocrates Foundation, Kos, Greece, 2000.
141) Cordier S, Chevrier C, Robert-Gnansia E et al. Risk of congenital anomalies in the vicinity of municipal solid waste incinerators. Occup Environ Med 2004: 61(1): 8-15.
142) Dummer TJ, Dickinson HO, Parker L. Adverse pregnancy outcomes around incinerators and crematoriums in Cumbria, North-west England, 1956-93. J Epidemiol Community Health 2003: 57 (6): 456-61.
143) Dolk H, Vrijheld M, Armstrong B et al. Risk of congenital anomalies near hazardous-waste landfill sites in Europe: the EUROHAZCON study. Lancet, 1998; 352(9126): 423-7.

144)Elliot P, Briggs D, Morris S et al. Risk of adverse birth outcomes in populations living near landfill sites. BMJ, 2001; 323(7309): 363-8.
145) Croen LA, Shaw GM, Sanbonmatsu L et al. Maternal residential proximity to hazardous waste sites and risk for selected congenital malformations. Epidemiology 1997; 8(4): 347-54.
146) Orr M, Bove F, Kaye W et al. Elevated birth defects in racial or ethnic minority children of women living near hazardous waste sites. Int J Hyg Environ Health, 2002; 205(1-2): 19-27.
147) Johnson BL. A review of the effects of the effects of hazardous waste on reproductive health. Am J of Obstetrics and Gynecology 1999; 181: S12-S16.
148) NCI, 1991: "Cancer Statistics Review 1973-88", NIH Publications No 91-2789.
149) Tomatis L, Cancer, Causes, Occurrence and Control, IARC Scientific publications 100, (Lyon, France, IARC 1996) 21.
150) Graphs of chemical production: From International Trade Commission, Washington DC.
151) Davies DL, Hoel D, Foxj, Lopez A. International trends in cancer mortality in France, West Germany, Italy, Japan, England and Wales and the USA. Lancet 1990; 336 (8713): 474-81.
152) Pickle LW, Mason TJ, Fraumeni JF Jr. The new United States Cancer Atlas. Recent Results Cancer Res, 1989; 114: 196-207.
153) Najem GR, Louria DB, Lavenhar MA et al. Clusters of cancer mortality in New Jersey municipalities, with special reference to chemical toxic waste disposal sites and *per capita* income. Int J Epidemiol 1985; 14(4): 528-37.
154) Najem GR, Greer W. Female reproductive organs and breast cancer mortality in New Jersey Counties and the relationship with certain environmental variables. Prev Med 1985: 14(5): 620-35.
155) Hoover R, Fraumeni JF Jr. Cancer mortality in US counties with chemical industries. Environ Res 1975; 9(2): 196-207.
156) Goldman BA. The Truth About Where You Live: An Atlas for Action on Toxins and Mortality. New York: Random House 1991.
157) Zahm SH, Blair A. Cancer among migrant and seasonal farmers: an epidemiologic review and research agenda. Am J of Ind Med 1993; 24(6): 753-66.
158) Tornling G, Gustavsson P, Hogstedt C. Mortality and cancer incidence among Stockholm fire fighters. Amer J Industrial Med 1994: 25(2): 219-28.
159) Zahm SH, Weisenburger DD, Babbitt PA et al. A case control study of non-Hodgkin's Lymphoma and the Herbicide 2,4 Dichlorophenoxyacetic acid (2,4-D) in Eastern Nebraska. Epidemiology 1990; 1 (5): 349-56.
160) Hardell L, Eriksson M, Lenner P et al. Malignant lymphoma and exposure to chemicals, especially organic solvents, chlorophenols and phenoxy acids: a case control study. Brit J Cancer 1981; 43(2): 169-76.
161) Harshbarger JC and Clark JB. Epizootiology of neoplasms in bony fish of North America. Sci Total Environ 1990; 94(1-2): 1-32.
162) Hayes HM Jr, Hoover R, Tarone RE. Bladder cancer in pet dogs: a sentinel for environmental cancer. Am J Epidemiol 1981; 114(2): 229-33.
163) Baumann PC, HarshbargerJC. Decline in liver neoplasms in wild brown bullhead catfish after coking plant closes and environmental PAHs plummet. Environ health Perspect 1995; 103: 168-70.
164) Perera F.P, Hemminki K, Gryzbowska E et al. Molecular and Genetic Damage in Humans from Environmental Pollution in Poland. Nature 1992; 360 (6401): 256-58.
165) Perera FP, Mooney LA, Stamfer M et al. Associations between carcinogen-DNA damage, glutathione S transferase genotypes, and risk of lung cancer in the prospective Physician's Health Cohort Study. Carcinogenesis 2002; 23(10): 1641-6.
166) Lewis-Michl EL, Melius JM, Kallenbach LR et al. Breast cancer risk and residence near industry or traffic in Nassau and Suffolk Counties, Long Island, New York. Arch Environ Health 1996; 51(4): 255-65.
167) The Long island Breast Cancer Study Reports 1-3 (1988-90), New York State Department of Health, Department of Community and Preventative Medicine, Nassau County Department of Health and Suffolk County Department of Health Services.
168) Aschengrou A, Ozonoff DM. Upper Cape Cancer Incidence Study: Final Report. Boston: Mass. Depts of Public Health and Environment Protection 1991.
169) Aschengrau A, Ozonoff D, Paulu C et al. Cancer risk and tetrachloroethylene-containing drinking water in Massachusetts. Arch Environ Health 1995; 48(5): 284-92.
170) McKelvey W, Brody JG, Aschengrau A et al. Association between residence on Cape Cod, Massachusetts, and breast cancer. Ann Epidemiol 2004; 14(2): 89-94.
171) Fagliano J, Berry M, Boye F et al. Drinking water contamination and the incidence of leukaemia:an ecologic study. Am J Public Health 1990; 80 (10): 1209-12.
172) Cantor KP et al., Water Pollution *In* Schottenfeld D and Fraumeni JF Jr (eds.), Cancer Epidemiology and Prevention, 2nd ed. Oxford: Oxford Univ Press 1996.

173) Lagakos S.W et al. An analysis of contaminated well water and health effects in Woburn, Massachusetts. J Amer Stat Assoc 1986: 395: 583-96.
174) Osborne J.S, Shy CM, Kaplan BH. Epidemiologic analysis of a reported cancer case cluster in a small rural population. Am J Epidemiol 1990; 132 (Supp 1): S87-95.
175) Lampi P, Hakulinen T, Luostarinen et al. Cancer incidence following chlorophenol exposure in a community in Southern Finland. Arch Environ Health 1992; 47(3): 167-75.
176) IARC Monographs on Evaluation of Carcinogenic Risks to Humans Suppl 7 (Lyon, France: IARC 1987).
177) US.DHHS Seventh Annual Report on Carcinogens, Research Triangle Park, NC:us. Department of Health and Human Services, 1990.
178) Holzman D. Banking on tissues. Environ Health Perspect 1996; 104(6): 606-10.
179) Moses M, Johnson ES, Anger WK et al. Environmental equity and pesticide exposure. Toxicol Ind Health 1993; 9(5): 913-59.
180) Body Burden: Executive Summary, 2003, Environmental Working Group, Mount Sinai School of Medicine and Commonweal. www.ewg.org/reports/bodyburden/
181) Foster W, Chan S, Platt L, Hughes C. Detection of endocrine disrupting chemicals in samples of second trimester human amniotic fluid. J Clinic Endocrinol Metabol 2000; 85(8): 2954-7.
182) Zieger M. Biomarkers: The clues to genetic susceptibility. Environ Health Perspectives 1994; 102 (1): 50-7.
183) Rodier PM. Developing brain as a target of toxicity. Environ Health Perspect 1995: 103 Suppl 6: 73-6.
184) Pritchard C, Baldwin D, Mayers A. Changing patterns of adult neurological deaths (45-74 years) in the major western world countries (1979-1997). Public Health 2004; 118(4): 268-83.
185) Taylor B, Miller E, Farrington CP et al. Autism and measles, mumps and rubella vaccine: no epidemiological evidence for a causal association. Lancet 1999; 353(9169): 2026-9.
186) Rybicki RA, Johnson CC, Uman J, Gorrell JM. Parkinson's disease mortality and the industrial use of heavy metals in Michigan. Mov Disord 1993; 8(1): 87-92.
187) Strahilevitz M, Strahilevitz A, Miller JE. Air pollutants and the admission rate of psychiatric patients. Am J Psychiatry 1979; 136(2): 205-7.
188) Briere J, Downes A, Spensley J. A. summer in the city: urban weather conditions and psychiatric emergency room visits. J Abnorm Pyschol 1983; 92(1): 77-80.
189) Morrow LA, Kamis H, Hodgson MJ. Psychiatric symptomatology in persons with organic solvent exposure. J Consult Clinic Pyschol 1993; 61(1): 171-4.
190) Morrow LA, Stein L, Scott A et al. Neuropsychological assessment, depression and past exposure to organic solvents. Applied Neuropyschol 2001; 8(2): 65-73.
191) Hall RW. A study of mass murder: evidence of underlying cadmium and lead poisoning and brain-involved immunoreactivity. Int J Bioscoc Med Res 1989; 11: 144-52.
192) Marlowe M, Schneider HG, Bliss LB. Hair mineral analysis in emotionally disturbed and violence prone children. Int J Biosoc Med Res 1991; 13: 169-79.
193) Pihl RO, Ervin F. Lead and cadmium levels in violent criminals. Pyschol Rep 1990; 66(3Pt 1): 839-44.
194) Denno DW. Gender, crime and the criminal law defences. J Crim Law Criminol 1994; 85: 80-180.
195) Deborah Denno. Biology and Violence: From Birth to Adulthood. Cambridge University Press, 1990.
196) Stretesky PB, Lynch MJ. The relationship between lead exposure and homicide. Arch Ped Adolesc Med 2001; 155(5): 579-82.
197) Stretesky PB, Lynch MJ. The relationship between lead and crime. J Health & Soc Behav 2004; 45(2): 214-29.
198) Centers for Disease Control. Blood and hair mercury levels in young children and women of childbearing age. United States 1999 Morbidity and Mortality Report, 2001; 50: 140-43.
199) Anderson LM, Diwan BA, Fear NT, Roman E. Critical windows of exposure for children's health: cancer in human epidemiological studies and neoplasms in experimental animal models. Environ Health Perspect 2000; 108 suppl 3: 573-94.
200) US Environmental Protection Agency, Office of Pollution Protection and Toxic Substances, Chemical Hazard Data Availability Study: What do we really know about high production volume chemicals? USEPA: Washington DC,1998.
201) Sonnenschein C, Soto AM. An Updated review of environmental estrogen and androgen mimics and antagonists. J Steroid Biochem Mol Biol 1998; 65 (1-6): 143-50.
202) Markey CM, Coombs MA, Sonnenschein C, Soto AM. Mammalian development in a changing environment: exposure to endocrine disruptors reveals the developmental plasticity of steroid-hormone target organs. Evol Dev 2003; 5(1): 67-75.

203) Tilson HA, Jacobson JL, Rogan WJ. Polychlorinated biphenyls and the developing nervous system: cross species comparisons. Neurotoxicol Teratol 1990; 12 (3): 239-48.
204) Weisgals-Kuperas N, Patandin S, Berbers GA, et al. Immunological effects of background exposure to polychlorinated biphenyls and dioxins in Dutch preschool children. Environ Health Perspect 2000; 108(12): 1203-7.
205) Rogan WJ, Gladen BC, McKinney JD, et al. Polychlorinated biphenyls (PCBs) and dichlorodiphenyldichloroethene (DDE) in human milk: effects of maternal factor and previous lactation. Am J Public Health 1986; 76(2): 172-7.
206) Body Burden: The Pollution in Newborns: Executive Summary, July 2005, Environmental Working Group, Mount Sinai School of Medicine and Commonweal.www.ewg.org/reports/bodyburden2/execsumm.php
207) A Present for Life: Hazardous chemicals in umbilical cord blood. WWF/Greenpeace, September 2005. www.greenpeace.org/raw/content/international/press/reports/umbilicalcordreport.pdf
208) Jensen AA, Slorach SA. Assessment of infant intake of chemicals via breast milk *in* Chemical Contaminants in Human Milk. Boca Raton: CRC Press 1991. pp215-22.
209) Koopman-Esseboom C, Huisman M, Weisglas-Kuperus N, et al. Dioxin and PCB levels in blood and human milk in relation to living in the Netherlands. Chemosphere 1994; 29 (9-11): 2327-38.
210) Patandin S, Dagnelie PC, Mulder PG, et al. Dietary exposure to polychlorinated biphenyls and dioxins from infancy until adulthood: a comparison between breast-feeding, toddler and long-term exposure. Environ Health Perspect 1999; 107(1): 45-51.
211) Rogan WJ, Bagniewska A, Damstra T. Pollutants in breast milk. N Engl J Med 1980; 302(26): 1450-3.
212) Jacobson JL, Jacobson SW. Prenatal exposure to polychlorinated biphenyls and attention at school age. J Paediatr 2003; 143(6): 780-8.
213) Jacobson JL, Jacobson SW. Association of prenatal exposure to an environmental contaminant with intellectual function in childhood. J Toxicol Clin Toxicol 2002; 40(4): 467-75.
214) Jacobson JL, Jacobson SW. Intellectual impairment in children exposed to polychlorinated biphenyls in utero. N Eng J Med 1996; 335(11): 783-9.
215) Kinbrough RD. Toxicological implications of human milk residues as indicated by toxicological and epidemiological studies *in* Jensen AA & Slorach SA: Chemical Contaminants in Human Milk, 1990 pp271-83.
216) Rice DC. Behavioural impairment produced by low-level postnatal PCB exposure in monkeys. Env Res 1999; 80(2 Pt 2): S113-S121.
217) Rice DC. Effects of postnatal exposure of monkeys to a PCB mixture on spatial discrimination reversal and DRL performance. Neurotoxicol Teratol 1998; 20(4): 391-400.
218) Rice DC, Hayward S. Effects of postnatal exposure to a PCB mixture in monkeys on non-spatial discrimination reversal and delayed alternation performance. Neurotoxicology 1997; 18(2): 479-94.
219) Hallgren S, Sinjari T, Hakansson H, Darnerud PO. Effects of polybrominated diphenyl ethers (PBDEs) and polychlorinated biphenyls (PCBs) on thyroid hormone and vitamin A levels in rats and mice. Arch Toxicol 2001; 75(4): 200-8.
220) Hooper K, McDonald TA. The PBDEs: an emerging environmental challenge and another reason for breast milk monitoring programs. Env Health Perspect 2000; 108(5): 387-92.
221) Moolgavkar SH, Venzon DJ. Two-event model for carcinogenesis: incidence of curves for childhood and adult tumours. Maths Biosci 1979; 47: 55-77.
222) Rodier PM. Chronology of neuron development: animal studies and their clinical implications. Dev Med Child Neurol 1980; 22(4): 525-45.
223) Ekbom A, Hsieh CC, Lipworth L, et al. Intrauterine environment and breast cancer risk in women: a population-based study. J Natl Cancer Inst 1997; 89(1): 71-6.
224) Knox EG. Childhood cancers and atmospheric carcinogens. J Epidemiol Community Health 2005; 59(2): 101-5.
225) Tomatis L, Overview of perinatal and multigeneration carcinogenesis. ARC Sci Publ 1989; 96: 1-15.
226) Anderson LM, Donovan PJ, Rice JM, Risk assessment for transplacental carcinogenesis. *In* New Approaches in Toxicity Testing and their Application in Human Risk Assessment (ed Li AP). 1985 pp179-202.
227) Landrigan PJ, Garg A. Chronic effects of toxic environmental exposures in children's health. J Toxicol Clinical Toxicol 2002; 40(4): 449-56.
228) Calabrese E.J. Age and Susceptibility to Toxic Substances. New York, John Wiley & Sons 1986.
229) National Academy of Sciences. Scientific Frontiers in Developmental Toxicology and Risk Assessment. National Academy Press, Washington DC 2000.
230) Jacobson JL, Jacobson SW, Humphrey HE. Effects of *in utero* exposure to polychlorinated biphenyls and related contaminants on cognitive functioning in young children. J Paediatr 1990; 116 (1): 38-45.

231) Needleman HL, Leviton A, Bellinger D. Lead-associated intellectual deficit. N Eng J Med 1982; 306(6): 367.
232) Rinsky RA et al. Benzene and leukemia: an epidemiologic risk assessment. N Eng J Med 1987; 316(17): 1044-50.
233) Pelkonenn O. Comparison of activities of drug-metabolizing enzymes in human fetal and adult livers. Clinic Pharmacol Ther 1973; 14(5): 840-6.
234) Hattis D, Russ A, Goble R, et al. Human interindividual variability in susceptibility to airborne particles. Risk Anal 2001; 21(4): 585-99.
235) Friedman R. Sensitive Populations and Environmental Standards. The Conservative Foundation, Washington DC (1981).
236) European Commission 2000. Communications from the Commission on the Precautionary Principle (COM (2000) 1) Brussels. URL: http://europa.eu.int/comm./dgs/health_consumer/library/pub/pub07_en.pdf (accessed 30 November 2003).
237) Grandjean P, Bailar JC, Gee D, et al. Implications of the precautionary principle in research and policy-making. Am J Ind Med 2004; 45(4): 382-5.
238) Franchini M, Rial M, Buiatti E, Bianchi F. Health effects of exposure to waste incinerator emissions: a review of the epidemiological studies. Ann Ist Super Sanita, 2004; 40(1): 101-15.
239) Rice DC, Evangelista de Duffard AM, Duffard R et al. Lessons for neurotoxicology from selected model compounds SGOMSEC joint report. Env Health Perspect 1996; 104 (Supp 2): 205-15.
240) Waste Working Group, Friends of the Earth, Ireland and Voice. Submission to the Limerick, Clare, Kerry Regional Waste Plan 2000.
241) Wulf-Schnabel J, Lohse J. Economic evaluation of dust abatement techniques in the European Cement Industry. A report produced for the European Commission, May 1999.
242) Pianin E. Study finds Net Gain from Pollution rules. Washington Post, Sept 27th, 2003.
243) World Wildlife Fund Report: Compromising Our Children: Chemical Impacts on Children's intelligence and Behaviour, June 2004. www.wwf.org.uk/chemicals
244) Berd V et al (eds.), Childhood Cancer and Nuclear Installations (London, BMJ Publishing Group 1993).
245) Gardner MJ. Childhood leukaemia around the Sellafield nuclear plant. *In* P Elliot et al (eds.) Geographical and Environmental Epidemiology: Methods for Small Area Studies. Oxford, Oxford University Press 1992, pp291-309.
246) Heasman MA, Kemp IW, Urquart JD, Black R. Childhood cancer in Northern Scotland. Lancet 1986; 1 (8475): 266.
247) Roman E, Watson A, Beral V, et al. Case control study of leukemia and Non-Hodgkin lymphoma among children aged 0-4 Years living in West Berkshire and North Hampshire health districts. BMJ 1993; 306(6878): 615-21.
248) Morris MS, Knorr RS. Adult leukemia and proximity-based surrogates for exposure to Pilgrim plant's nuclear emissions. Arch Environ Health 1996; 51(4): 266-74.
249) Clapp RW et al. Leukaemia near Massachusetts nuclear power plant. Lancet 1987; 2(8571): 1324-5.
250) National Research Council (2000): Waste Incineration and Public Health ISBN: 0-309-06371-X,Washington DC, National Academy Press.
251) Mittal AK, Van Grieken R, Ravindra.. Health risk assessment of urban suspended particulate matter with special reference to polycyclic aromatic hydrocarbons: a review. Rev Environ Health 2001; 16 (Pt 3): 169-89.
252) Final report to the North American Commission for Environmental Cooperation (Flushing, N.Y.: Centre for the Biology of Natural systems, Queens College, CUNY, 2000).
253) Raloff FJ. Even Nunavut gets plenty of dioxin. Science News 2000; 158 : 230.
254) National Research Council (NRC). Toxicity Testing: Strategies to Determine Needs and Priorities (1984), National Academy Press, Washington, D.C.
255) Moore CF. Silent Scourge: Children, Pollution and Why Scientists Disagree. Oxford University Press, 2003, Oxford.
256) Schettler T, Solomon G, Valenti M and Huddle A. Generations at Risk: Reproductive Health and the Environment, 1999, MIT Press, Cambridge, Massachusetts, & London.
257) Friends of the Earth Briefing. Incinerators and Deprivation, Jan 2004.

Publications by the British Society for Ecological Medicine

(under its previous name The British Society for Allergy Environmental and Nutritional Medicine, BSAENM)

Environmental Medicine in Clinical Practice. Honor Anthony, Sybil Birtwistle, Keith Eaton and Jonathan Maberly. BSAENM Publications 1997, 483 pages, 242 figures and tables, over 1,000 references. ISBN 0 9523397 2 2. The first comprehensive text for doctors on ecological medicine.

Scientific Reports

Effective Allergy Practice*:* A Document on Standards of Care and Management for the Allergy Patient. BSAENM Publications 1994. ISBN 09523397 0 6

and in the Journal of Nutritional and Environmental Medicine 1995; 5: 79-104.

Effective Nutritional Medicine: the application of nutrition to major health problems. BSAENM Publications 1995. ISBN 0 9523397 1 4.

and in the Journal of Nutritional and Environmental Medicine 1996; 6: 191-232.

Multiple Chemical Sensitivity: Recognition and Management. A document on the health effects of everyday chemical exposures and their implications. BSAENM Publications 2000. ISBN 0 9523397 3 0

and in the Journal of Nutritional and Environmental Medicine 2000; 10 39-84.

All available from The British Society for Ecological Medicine, Po Box 7, Knighton, LD7 1WT Phone (premium line) 09063020010, Fax 01547 550339.