INCINERATION AND HUMAN HEALTH

State of Knowledge of the Impacts of Waste Incinerators on Human Health.

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EXECUTIVE SUMMARY

Management of municipal and industrial waste is a growing problem throughout the world. In the European Union, while waste output is continually increasing, new regulations are imposing more stringent restrictions on the amount of waste permitted to go to landfill. At the same time, many incinerators have been closed over the past few years because of stricter regulations on their atmospheric emissions. In Europe, all incinerators will soon have to comply with new standards set out in a recent EC draft directive.

Fortunately, there are alternative solutions to turn around the waste crisis on a long-term basis. Primarily, this means the implementation of waste prevention strategies, and in conjunction with this, waste re-use and recycling. Despite this option, there is an emerging trend for constructing, and planning to construct, new incinerators in an attempt to provide a "quick fix" solution to the waste crisis. Incinerators are deemed as favourable in this respect because they are perceived as reducing waste to one tenth of its original volume, and therefore reduce the volume of waste going to landfill sites.

Incinerators, however, are controversial in terms of their potential impacts on the environment and human health, as well as in terms of the economic considerations which do not favour this technology. They are known to emit numerous toxic chemicals into the atmosphere and produce ashes and other residues. One country, the Philippines, has taken serious note of the many concerns about incineration at a governmental level. Following strong public opposition to incinerators, the Philippine Clean Air Act of 1999, banned the incineration of municipal, medical and hazardous waste. Waste reduction, re-use and recycling are being promoted while non-burn technologies are recommended for waste that needs some form of treatment. Meanwhile, some governments in Europe are advocating the construction of even more incinerators.

This report was undertaken to draw together scientific findings on incinerator or releases and their impacts on human health. A broad range of health effects have been associated with living near to incinerators as well as with working at these installations. Such effects include cancer (among both children and adults) adverse impacts on the respiratory system, heart disease, immune system effects, increased allergies and congenital abnormalities. Some studies, particularly those on cancer, relate to old rather than modern incinerators. However, modern incinerators operating in the last few years have also been associated with adverse health effects.

Despite reductions of some chemicals in stack emissions, modern incinerators nevertheless still emit numerous toxic substances to the atmosphere as well as in other residues such as fly ash and bottom ash. Moreover, reductions of dioxins and other chemicals in stack gases commonly leads to increased releases of these same chemicals in the other incinerator residues. In most cases, health effects which have been associated with incinerators cannot be tied down to a particular pollutant. Together with the limited data available, it is, therefore, impossible to predict health effects of incinerators either new or updated installations. With such factors in mind, this report demonstrates that there is an urgent need for the complete phase out of incineration and the implementation of sound waste management policies based on waste prevention, re-use and recycling.

INCINERATORS – WASTE GENERATORS

It is a common misconception that things simply disappear when they are burned. In reality, matter cannot be destroyed – it merely changes its form. This can be exemplified by looking at the fate of some substances in wastes which are burned in municipal solid waste (MSW) incinerators. These incinerators are typically fed mixed waste streams that contain hazardous substances, such as heavy metals and chlorinated organic chemicals. Following incineration, heavy metals present in the original solid waste are emitted from the incinerator stack in stack gases and in association with tiny particles, and are also present throughout the remaining ashes and other residues. Incineration of chlorinated substances in waste, such as polyvinyl chloride (PVC) plastic, leads to the formation of new chlorinated chemicals, such as highly toxic dioxins, which are released in stack gases, ashes and other residues. In short, incinerators do not solve the problems of toxic materials present in wastes. In fact they simply convert these toxic materials to other forms, some of which may be more toxic than the original materials. These newly created chemicals can then re-enter the environment as contaminants in stack gases, residual ashes and other residues.

All types of incinerators release pollutants to the atmosphere in stack gases, ashes and other residues. A multitudinous array of chemicals is released, including innumerable chemicals that currently remain unidentified. The chemicals present in stack gases are often also present in ashes and other residues. Such chemicals include dioxins, polychlorinated biphenyls (PCBs), polychlorinated napthalenes, chlorinated benzenes, polyaromatic hydrocarbons (PAHs), numerous volatile organic compounds (VOCs), and heavy metals including lead, cadmium and mercury. Many of these chemicals are known to be persistent (very resistant to degradation in the environment), bioaccumulative (build up in the tissues of living organisms) and toxic. These three properties make them arguably the most problematic chemicals to which natural systems can be exposed. Some of the emitted

chemicals are carcinogenic (cancer-causing) and some are endocrine disruptors. Others such as sulphur dioxide (SO_2) and nitrogen dioxide (NO_2) as well as fine particulate matter, have been associated with adverse impacts on respiratory health.

It is a popular misconception that the weight and volume of the original raw waste are reduced during incineration. It is often quoted that the volume of waste is reduced by about 90% during incineration. Even if only the residual ashes are considered, however, the actual figure is closer to 45%. The weight of waste is supposedly reduced to about one-third during incineration. However, this once again refers only to ashes and ignores other incinerator emissions in the form of gases, which result in an increased output in weight. In sum, if the mass of all the outputs from an incinerator, including the gaseous outputs, are added together, then the output will exceed the waste input.

ENVIRONMENTAL AND HUMAN EXPOSURE TO INCINERATOR RELEASES

The research carried out on environmental contamination and human exposure to pollutants released by incinerators is limited and has focused mainly on dioxins and heavy metals. Research has demonstrated that both older and more modern incinerators can contribute to the contamination of local soil and vegetation with dioxins and heavy metals. Similarly, in several European countries, cow's milk from farms located in the vicinity of incinerators has been found to contain elevated levels of dioxins, in some cases above regulatory limits.

Populations residing near to incinerators are potentially exposed to chemicals through inhalation of contaminated air or by consumption of contaminated agricultural produce (e.g. vegetables, eggs, and milk) from the local area and by dermal contact with contaminated soil. Significantly increased levels of dioxins have been found in the tissues of residents near to incinerators in the UK, Spain and Japan most likely as a result of such exposure. Two studies in the Netherlands and Germany however, did not find increased levels of dioxins in body tissues of residents living near incinerators. At an incinerator in Finland, mercury was increased in hair of residents living in the vicinity, most likely due to incinerator releases. Children living near a modern incinerator in Spain were found to have elevated levels of urinary thioethers, a biomarker of toxic exposure. Elevated levels or more frequent occurrence of certain PCBs occurred in the blood of children living near a hazardous waste incinerator in Germany.

Several studies have reported elevated levels of dioxins (total TEQ), and/or certain dioxin congeners, in the body tissues of individuals employed at both modern and older incinerators. This is thought to be a consequence of exposure to contaminated ashes in the workplace. Similarly, some studies have reported increased levels of chlorinated phenols, lead, mercury and arsenic in the body tissues of incinerator workers.

HEALTH IMPACTS

Experimental data confirm that incinerators release toxic substances and that humans are exposed as a consequence. Studies on workers at incinerator plants, and populations residing near to incinerators, have identified a wide range of associated health impacts (see tables below). These studies give rise to great concerns about possible health impacts from incinerators even though the number of studies (particularly those that have been conducted to appropriately rigorous scientific standards) is highly limited. These should be seen, however, as strongly indicative that incinerators are potentially very damaging to human health.

Summary of studies on occupational health

HEALTH IMPACT	COMMENTS
Biomarkers of Exposure	
Elevated mutagens in urine	Incinerator ashes and stack emissions are mutagenic (have the ability to damage DNA). Workers are therefore exposed to mutagenic compounds. Elevated mutagens in urine indicate exposure to mutagenic compounds. (Study dates 1990 & 1992).
Elevated levels of hydroxypyrene in urine	Hydroxypyrene is an indicator of internal exposure to PAHs. The result suggests elevated exposure to PAHs. (Study date 1992).
Increased quantity of thioethers in urine	Thioethers in urine are an indicator of exposure to electrophilic compounds such as PAHs. The results suggest exposure to electrophilic compounds. (Study date 1981).
Cancer	
3.5-fold increased probability of mortality from lung cancer	Workers who were employed at a MSW incinerator in Sweden at sometime between 1920 and 1985. (Study date 1989).
1.5-fold increased likelihood of mortality from oesophageal cancer	Workers who were employed at a MSW incinerator in Sweden at sometime between 1920 and 1985. In conjunction with evidence from other research, the result implies an increased health threat to workers. (Study date 1989).
2.79-fold increase in mortality from gastric cancer	Workers employed at an MSW incinerator in Italy at sometime between 1962 and 1992. Some of the increase may have been due to other confounding factors. (Study date 1997).
Other Impacts	
Increased mortality from ischemic heart disease	Workers who were employed at a Swedish MSW incinerator in Sweden at sometime between 1920 and 1985. The result was statistically significant in workers with greater than 40 years employment. (Study date 1989).
Excess hyperlipidemia. A significant association between blood dioxin levels and natural killer cell activity (immune system effect). Altered sex ratio among offspring. Decreased liver function. Increased allergy.	Workers employed at an incinerator in Japan, that operated between 1988 and 1997. Excess of hyperlipidemia was significant. Change in immune system cells. Altered sex ratio was not statistically significant. Correlation between allergy and dioxin exposure must be confirmed. (Study date 2000).
Excess of proteinuria (urine abnormality) and hypertension. Possible increased incidence of small airway obstruction (unconfirmed diagnosis). Abnormal blood chemistry.	Workers at a MSW incinerator in the US. An excess of workers with significant proteinuria. (Study date 1992).
Chloracne (a skin condition due to dioxin-exposure)	Chloracne found in one worker from an old incinerator in Japan, who had high blood levels of dioxin. (Study date 1999).

Summary of studies on health of populations living in the vicinity of incinerators

HEALTH IMPACT	COMMENTS
Biomarkers of Exposure	
Elevated levels of thioethers in children's urine	Urinary thioether levels were higher among children living near a recently built incinerator in Spain. (Study date 1999)
No abnormal chromosomal damage	No excess chromosomal damage among children living near two Belgian incinerators. (Study date 1998)
Cancer	
44% increase in soft tissue sarcoma and 27% increase in non-Hodgkin's lymphoma.	Significant clusters of these cancers in residents living close to an incinerator in France. Possibly due to exposure to dioxin from the incinerator, but more research is needed to confirm if this is the case. (Study date 2000).
6.7-fold increase in likelihood of mortality from lung cancer	Significantly increased occurrence in residents living close to a MSW incinerator in an urban area of Italy. (Study date 1996).
Increased incidence of cancer of the larynx	Found around one UK hazardous incinerator of waste solvents (1990), but not nine others. In Italy, excess mortality from this cancer was found in residents living near to an incinerator, a waste disposal site and an oil refinery.
37% excess mortality due to liver cancer	A study on 14 million people living within 7.5 km of 72 MSW incinerators in the UK. Further research to eliminate possible confounders found the increased probability of liver cancer to lie between 20 and 30%. Social deprivation could not be totally ruled out as a confounder. (Study dates 1996 and 2000).
2-fold increased probability of cancer mortality in children	A study conducted on 70 MSW incinerators in the UK (1974-87) and 307 hospital waste incinerators (1953-1980). These results are consistent with another study in which an increased probability of childhood cancer was observed for hospital incinerators and large-scale, high-temperature combustion industries (Study dates 1998 and 2000).
Respiratory Impacts	
Increased purchase of medicine for respiratory problems.	A study at a village in France that had a MSW incinerator. Results suggest increased use of medicine for respiratory illness but a cause-effect relationship cannot be concluded (Study date 1984).
Increased respiratory symptoms, including 9-times increase in reporting of wheezing or cough.	A study in the US on residents living near to a hazardous waste incinerator. The results are of limited utility because of methodological concerns about the study. (Study date 1993).
Adverse impacts on lung function of children.	A study on children living near to a wire reclamation incinerator in Taiwan. Results indicate that higher air pollution, but not the incinerator itself, is linked to altered lung function in children. (Study date 1992).
Increased respiratory symptoms including lung disease, wheezing, persistent cough and bronchitis.	A study on 58 individuals living near to cement kilns burning hazardous waste in the US. Significant increase in respiratory symptoms. (Study date 1998).

HEALTH IMPACT	COMMENTS
Respiratory Impacts	
No adverse effect on the prevalence or severity of asthma in children.	A study on children living near to sewage sludge incinerators in Australia. (Study date 1994).
No increase in respiratory effects or decrease in lung function	A study on 3 communities (6963 individuals) living near to a municipal, hazardous and hospital waste incinerator in the US. The lack of association between exposure to particulate air pollution and respiratory health in this study should be interpreted cautiously due to limitations in data on individual exposures.
Sex Ratio	
Increase in female births	A study on populations living near to 2 incinerators in Scotland, UK. The effect was found in the area potentially most exposed to incinerator releases. Other studies have found an increase in female births where fathers were accidentally exposed to high levels of dioxins. (Study dates 1995 and 1999).
Congenital Abnormalities	
Increased incidence in orofacial clefts Other midline defects including spina bifida and hypospadias (genital defect)	The significant increase in orofacial clefts was observed for births in an area located near to an incinerator site where open burning of chemicals took place 1960-69. A link between the conditions and living near the incinerator is likely but not confirmed.
1.26-fold increased probability of congenital malformations among new born infants	A study conducted on a population living near to 2 MSW incinerators in Wilrijk, Belgium. (Study date 1998).
Increased congenital eye malformations (anecdotal report)	Reported at an area near two chemical waste incinerators in Scotland, UK. Further research in the UK found no link, although the study was hampered by lack of data on the condition. (Study date 1989).
Multiple Pregnancy	
Possible increase in rate of twinning/multiple pregnancy.	An increase in twinning was significant in 1980 in a population living near to an incinerator in Scotland, UK. A 2.6-fold probability of multiple pregnancy found near incinerator in Belgium (Study date 2000). No impact on multiple pregnancy found on a survey of an incinerator in Sweden. Data from different studies is conflicting and inconclusive.
Other Impacts	
Lower thyroid hormone levels in children	Children living near a German incinerator had significantly lower blood levels of certain thyroid hormones. (Study date 1998)
Increased allergies, increased incidence of common cold, increased complaints about health in general, increased use of medication in school children	A study conducted on school children living near to two MSW incinerators in Wilrijk, Belgium. (Study date 1998).

INCINERATOR RELEASES AND REGULATION

• Stack Gases

As previously mentioned, numerous chemicals are emitted to the atmosphere from incinerators thrtough the stack gases. Important points regarding some of these chemical emissions are given below.

• Dioxins

Extensive research has demonstrated that dioxins can cause a diverse array of toxic effects. They have become wide-spread contaminants throughout the globe and are present in the body tissues of human beings across the whole globe. Research suggests that, in industrialised countries, dioxins have now reached levels in tissues of the women which may cause subtle, adverse effects upon the immune system, and nervous system of their babies.

Incineration, particularly MSW incineration, was identified as a major source of dioxins during the 1980s and early 1990s. It has been estimated as accounting for between 40 and 80% of atmospheric dioxin emissions in various industrialised countries. The true figure may be even greater because there are several methodological flaws in nearly all of the dioxin inventories that estimate atmospheric emissions from incineration.

Considerable improvement in air pollution control technologies that have been installed in new or updated incinerators during the 1990s is thought to have led to substantial reductions in the quantity of dioxins released to the atmosphere from incinerator stacks. However, recent estimates suggest that MSW incinerators are still a main source of dioxins in the environment. In the UK, it was estimated that MSW incinerators were responsible for 30-56% of dioxin emissions while in Denmark a recent mass balance study identified MSW incineration as the dominant source of dioxins to atmosphere and a highly significant contributor (via ash residues) to landfill. Moreover, reduction of dioxins emitted in stack gases has most likely resulted in a corresponding increase in dioxins emitted as contaminants of ash residues.

While measurements taken from some new or modernised incinerators have shown that they comply with limits set by the new EC directive, others have not. Those not fulfilling the EC regulatory limit include incinerators that have recently been tested in Spain, Poland, Sweden, and Belgium. In Belgium, testing was carried out on an incinerator using the routine technique of taking "point measurements" which involves monitoring dioxin levels over a period of several hours. However, when testing was carried out by "continual monitoring", over a 2 week period, the results were substantially different. The point measurement technique underestimated dioxin emissions by a factor of 30 to 50. It is therefore of great concern that very few incinerators are tested using continual monitoring or tested under their normal operating conditions. Moreover, the new EC regulations do not stipulate that measurements should be taken using this technique, so current routine monitoring of incinerator stack gases, using point measurements, could be grossly inaccurate and underestimate dioxin emissions to air.

• Other Organic Compounds

For regulatory purposes, the EC has proposed a limit for total organic carbon emissions to atmosphere to regulate all the organic chemicals emitted. This regulation, however, fails to take into account the toxicity/health impacts of known organic chemicals that are emitted from incinerator stacks. Similarly it totally ignores unknown chemicals of unknown toxicity and the potential health effects they could cause.

• Heavy Metals

Heavy metals, including lead and cadmium, are emitted in stack gases from incinerators. Many heavy metals are persistent and exert a wide range of adverse impacts on health.

With the exception of mercury, the levels of heavy metals released in stack gases from incinerators have decreased considerably over the past decade due to improvement in air pollution abatement technologies. Nevertheless, the quantities in which they are still emitted from modern incinerators potentially add to current background levels in the environment and in humans. As is the case with dioxin emissions to the atmosphere, the reduction of levels of heavy metals emitted in stack gases causes a corresponding increase in levels in the ashes, which will, ultimately, result in contamination of the environment when these are disposed of.

• Particulate Matter

Incinerators of all types emit particulate matter into the atmosphere. The majority of this particulate matter is ultrafine in size. Current air pollution control devices on incinerators only prevent 5 to 30% of the "respirable" (<2.5 μm) sized particles from entering the atmosphere, and can do very little to prevent ultrafine (<0.1 μm) particulates from escaping. It is these respirable particles, and especially the ultrafine particles, which can reach the deepest regions of the lungs, and which are thought to be responsible for causing adverse impacts on human health. Incinerators therefore contribute to the type of particulate air pollution that is the most dangerous for human health. In addition, recent evidence suggests that particles containing heavy metals, such as those emitted from incinerators are

especially of concern with regard to health. Incinerators are, therefore, likely to produce particulate air pollution which is even more toxic than, for example, that emitted from a coal-fired power station.

The new EC Draft Directive does not set any limits for the release of fine particulate matter. Given the scale of the health impacts resulting from such particulate air pollution, this can be considered as an outstanding neglect of factors relevant to human health, and which requires rigid control and regulation.

• Ashes

Fly ashes from air filtration equipment on incinerators and the bottom ashes that remain after incineration contain numerous hazardous chemicals, such as dioxins and heavy metals. Despite the potential toxicity of ashes, there are no EC limits for levels of persistent organic chemicals and heavy metals in ashes.

Because of their contamination, disposal of incinerator ashes presents significant environmental problems. The majority of ash is landfilled. This can result in contamination of sub-soils and groundwater. In some cases, the contamination of groundwater by compounds that have leached from the waste, in particular, heavy metals like lead and cadmium from fly ash has been documented. In an attempt to reduce leaching, fly ash is sometimes stabilised in cement before disposal. Although this method reduces the immediate leaching of heavy metals and other toxic chemicals, weathering and erosion over time will ultimately cause their release back to the environment

There has been a recent tendency in some European countries to use bottom ashes and/or fly ashes for construction purposes, a practice that reduces the financial costs of "secure" ash disposal. Ash has been used in road and path construction. Again, however, the future releases of persistent toxic substances due to erosion over time could result in the release of substances back to the environment and, therefore, potentially to human exposure. This has recently been exemplified in Newcastle, UK where fly ash and bottom ash from a presently operating, modern incinerator, were used for path making and also spread over allotments as fertiliser between 1994 and 1999. Recent analysis of ash from the allotments found that it is contaminated with extremely high levels of heavy metals and dioxins. Clearly, the use of ashes from incinerators represents a potential threat to human health, but this practice is not being discouraged either by the EC or at a national level by the regulatory regimes proposed or currently in place.

• The Way Forward

A limited amount of epidemiological research has been directed at investigating the health impacts of incinerators. Despite this, scientific studies reveal that MSW and other incinerators have been associated with detrimental impacts on health.

The new EC draft directive on incinerators is not formulated to take human health impacts into account in relation to the regulation and control of these facilities. Rather, the regulatory limits that are set for the permissible release of substances are based on what is considered to be technically achievable. In any case, the draft EC directive on incinerators, not yet in force, can be regarded as already outdated. Many European countries have already committed themselves at the OSPAR Convention to phase out all releases of hazardous substances to the environment by 2020. In this context no emissions of hazardous chemicals would be allowable in stack gases or ashes. This is likely to prove impossible for incineration technology to ever achieve.

In addition, at the Fifth Intergovernmental Negotiating Committee Meeting (INC5) on the Elimination of Persistent Organic Pollutants (POPs), held in December 2000, a world-wide agreement was reached to reduce total dioxin releases, with the ultimate aim of their elimination. Incineration is listed as one of the main industrial source categories for dioxins, and requires the use of BAT (Best Available Techniques) for new installations and substantially modified existing facilities. It was also agreed to promote the development and, where deemed appropriate, require the use of substitute or modified materials, products and processes to prevent the formation and release of dioxins. In this context, incineration is acknowledged as a significant source of dioxins and, in the longer term these sources should be replaced by alternatives.

To comply with the provisions of the OSPAR agreement and of the emerging POPs Convention implies a radical rethink of industrial and manufacturing processes. Instead of waste-generating "dirty" technologies, which rely upon incineration and other environmentally dubious waste disposal techniques, OSPAR implies the need to develop and use "clean-production" technologies which eliminate toxic waste. The adoption of "zero-waste" as a central tenet of environmental regulation also implies that the Precautionary Principle of environmental protection will occupy an equally key position in the development of policy and regulatory frameworks. The precautionary principle requires that the burden of proof should not be laid upon the protectors of the environment to demonstrate conclusive harm, but rather on the prospective polluter to demonstrate no likelihood of harm. On this premise of precautionary regulation it can be argued that there is

already sufficient evidence of environmental contamination and adverse human health impacts to call for a complete phase out of incineration.

In the case of waste management, adoption of a zero releases strategy and the reduction of health impacts from waste management means a move towards an environmental management paradigm based upon the three axioms of reduce, re-use and recycle in relation to the generation of both municipal and industrial wastes.

GREENPEACE DEMANDS

A drive towards waste prevention, re-use and recycling, and therefore also towards lessening the adverse health impacts from waste management, should include the following measures:

- The phase out of all forms of industrial incineration by 2020, including MSW incineration. This is in line with the OSPAR Convention requirements for the phase out of emissions, discharge and losses of all hazardous substances by 2020.

- Financial and legal mechanisms to increase re-use of packaging (e.g. bottles, containers) and products (e.g. computer housings, electronic components).

- Financial mechanisms (such as the landfill tax) used directly to set up the necessary infrastructure for effective recycling.

- Stimulating markets for recycled materials by legal requirements for packaging and products, where appropriate, to contain specified amounts of recycled materials.

- Materials that cannot be safely recycled or composted at the end of their useful life (for example PVC plastic) must be phased out and replaced with more sustainable materials.

- In the short term, materials and products that add to the generation of hazardous substances in incinerators must be prevented from entering the the waste stream at the cost of the producer. Such products would include electronic equipment, metals and products containing metals such as batteries and florescent lighting and PVC plastics (vinyl flooring, PVC electrical cabling, PVC packaging, PVC-*u* window frames etc) and other products containing hazardous substances.

and more generally:

- Further the development of clean production technologies which are more efficient in terms of material and energy usage, produce cleaner products with less waste and which, ultimately can be designed to operate in a "closed loop" configuration in order to fulfil the needs of society in a more equitable and sustainable manner;

- Fully implement the Precautionary Principle, such that, in the future, problems are avoided before they occur. The continuation and further development of scientific research has a fundamental role to play in identification of potential problems and solutions. Nonetheless, we must be ready to take effective precautionary action to prevent environmental contamination and degradation in the face of the considerable and often irreducible uncertainties associated with determination of health and other environmental impacts from incineration.

1. INTRODUCTION TO HEALTH EFFECTS OF INCINERATORS

The impact of waste incinerators on health and their releases of hazardous combustion products, such as dioxins and PAHs are of great public concern (Ardevol *et al.* 1999). Research has identified numerous toxic compounds, which are emitted in stack gases and in ashes, as well as many unidentified substances of unknown toxicity (see section 5). Individuals who are exposed to the hazardous substances resulting from incineration, and whose health can, therefore, be potentially affected by such exposure, include workers connected with incinerator facilities and populations living within their local vicinity. Studies on exposure and health impacts of incinerators have focused entirely on these two groups of individuals.

Importantly, a recent publication by the National Research Council (NRC 2000), an arm of the National Academy of Sciences that was established to advise the U.S. government, concluded that it was not only the health of workers and local populations that could be affected by incinerators. The NRC reported that populations living more distantly from incinerators are also likely to be exposed to some incinerator pollutants. For example,

"*Persistent air pollutants, such as dioxins, furans and mercury, can be dispersed over large regions – well beyond the local areas and even the countries from which the sources first emanate.… Food contaminated near an incineration facility might be consumed by 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 markers. 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 a source incineration facility.*"

and,

"*The potential effects of metals and other pollutants that are very persistent in the environment may extend well beyond the area close to the incinerator. Persistent pollutants can be carried long distances from their emission sources, go through various chemical and physical transformations, and pass numerous times through soil, water, or food. Dioxins, furans, and mercury are examples of persistent pollutants for which incinerators have contributed a substantial portion of the total national emissions. Whereas one incinerator might contribute only a small fraction of the total environmental concentrations of these chemicals, the sum of the emissions of all the incineration facilities in a region can be considerable. The primary pathway of exposure to dioxins is consumption of contaminated food, which can expose a very broad population. In such a case, the incremental burden from all incinerators deserves serious consideration beyond a local level.*"

In the present report, published research on human exposure to pollutants from incinerators and health impact studies of workers and local populations is discussed. A broad range of health impacts has been documented in these two groups, including adverse effects on children living in local populations near to incinerators. However, whether the observed associations with pollutants from incinerators are causal is often difficult to confirm.

1.1 Types of Research Study

The impacts of incinerators on human health have been assessed primarily using three types of study. These are human exposure studies, epidemiological studies and finally, risk assessment studies. Exposure studies and epidemiological studies provide the most compelling evidence about the health impacts of incineration since they involve generating scientific data directly from the individuals under investigation. On the other hand, risk assessments are theoretical estimations of what health effects may occur based on mathematical calculation.

1.2 Exposure Studies

Exposure to compounds emitted from incinerators may occur, for example, by inhalation of contaminated air, or by consumption of local agricultural produce or soil that has been contaminated by deposition of airborne pollutants. In addition, workers at incinerator plants may also be exposed to contaminated ashes.

To assess possible health impacts resulting from exposure to incinerator releases, reliable methods of assessing exposure are required. One method to assess potential exposure is to monitor levels of contaminants in air from incinerators, and in soils, vegetation and agricultural produce (e.g. see section 4). However, such investigations do not permit the "internal exposure" in humans to be assessed directly (Ardevol *et al.* 1999). Evaluation of internal exposure requires the quantification of compounds in the human body. In recent years, with technological advances, it has become possible to monitor the level of certain toxic compounds from incinerators in the body tissues of humans. This involves the determination of contaminant concentrations in biological samples, for instance, in blood, urine, hair or breast milk.

Exposure studies analyse biological samples for:
1) chemical pollutants that are released from an incinerator, or, 2) metabolites (breakdown products) of these chemicals, or, 3) biomarkers of exposure, (which show biological effects of a toxic exposure). The results of the analyses are compared with a control group of individuals considered to be unexposed.

A number of studies have been conducted to assess exposure of incinerator workers (see section 2.1) and populations living near to incinerators (see section 3.1) using the analyses described above.

1.3 Epidemiological Studies

Epidemiological studies attempt to establish the incidence or prevalence of health effects that may be related to the intake of pollutants released from an incinerator. Information pertinent to the potentially contaminated people is used, for example, birth and death certificates, disease registries, physicians' reports, self-reported symptoms and illnesses. This is compared with similar information from potentially uncontaminated or less contaminated people. Some of the major challenges to establishing a cause-and-effect relationship through epidemiological studies are (NRC 2000):

- Identifying suitably exposed populations that are large enough to establish a useful degree of statistical significance.
- Identifying the many factors that modify the effect (e.g., age, sex, etc.) and/or potentially confounding factors (e.g., smoking, diet, etc.)
- Identifying biases (including reporting biases) in data collection.
- Measuring the frequency of occurrence and concentrations of specific pollutants within the affected population and a potentially unaffected control group.
- Measuring effects that are small, occur infrequently, take many years to appear, and/or occur not in the exposed individuals but in their offspring during infancy, childhood or adulthood.

Epidemiological studies have investigated a variety of health outcomes from exposure to incinerator releases in both workers and in populations living close to incinerators. In particular, cancer and respiratory effects have been analysed. Such human epidemiological studies are however limited in number given the widespread concern about potential health effects of incinerators. The rarity of these studies is possibly due to their expense and difficulty of performance. These studies are generally more valid than other health studies insofar as exposure to all pollutants emitted from incinerators are explicitly or implicitly accounted for, thus mirroring the "real" situation (Rowat 1999) although their power is determined by their design.

1.4 Risk Assessment

Risk assessment attempts to estimate exposure to a particular chemical from the releases in question and ultimately calculates the probability of health effects from the estimated exposure. Risk assessment is a step by step process which involves the use of mathematical equations to estimate pollutant releases, their transport and their transformation in the environment, together with human exposure and finally the likelihood of suffering health effects from this exposure. The use of risk assessment is largely for regulatory decision making.

The process of risk assessment itself is, however, fraught with uncertainties, is necessarily over-simplistic of environmental processes and warrants being viewed with deep scepticism on whether it can actually be protective of human health (e.g. Johnston *et al.* 1996). A fundamental problem of risk assessment is that the estimation of the health consequences of pollution is still a poorly understood science. Even for dioxin (TCDD), one of the most intensely studied chemicals, many unknowns remain and since risk assessment relies on toxicological data to estimate health effects, it is can only be as good as the data on which it is based. Indeed for many chemicals there is a substantial lack of toxicological information. This could obviously lead to imprecision in results generated by risk assessments. Moreover, in the case of the developing foetus and infant, there is a huge uncertainty in the toxicological significance of long-term low-dose exposure to pollutants. It is clear though that the developing stages of life are the most vulnerable to toxic insult. Risk assessments however are generally based on estimation of risk in adults and ignore the potential impacts on the foetus and developing young.

In estimating the probability of health impacts, many uncertainties appear at every stage of the risk assessment process. For instance, there is uncertainty in estimating the quantities of releases, in estimating the transport and transformation of pollutants in the environment and, from this calculation, estimating human exposure. It is indeed extremely difficult, if not impossible, to determine the actual doses involved in environmental exposures. In order to overcome uncertainties in estimations, risk assessors use "conservative" estimates and so assume that overestimating risks overcomes these problems and is therefore protective of public health. However, the notion of "conservative" is ill-defined and in practice raises significant questions concerning exactly how conservative a risk assessment should be and whether all uncertain parameters should be conservatively treated or just a selected few. In this way it becomes apparent that risk assessment not only contains many uncertainties, but it is also a subjective rather than a scientifically objective process. This again calls into question whether regulations derived from risk assessments can be truly protective of human health.

With regard to incineration and risk assessment, the National Research Council (NRC 2000), noted that the procedures used to perform risk assessment "*vary widely, from a snap judgment to the use of complex analytic models,*" and described other related difficulties:

"The committee's evaluation of waste incineration and public health was substantially impaired by the lack of available compilations of ambient concentrations of pollutants resulting from incinerator emissions. In addition, large variabilities and uncertainties associated with risk-assessment predictions often limit the ability to define risks posed by incinerators.... Emission data needed to fully characterize environmental concentrations for health-effects assessments are not readily available for most incineration facilities. Such information is lacking especially for dioxins and furans, heavy metals (such as lead, mercury, and cadmium), and particulate matter.... Generally, data are not collected during startup, shutdowns, and upset conditions – when the greatest emissions are expected to occur. Furthermore, such data are typically based on a few stack samples for each pollutant. Thus, the adequacy of such emissions data to characterize fully the contribution of incineration to ambient pollutant concentrations for health-effects assessments is uncertain."

A further point on the estimation of releases with regard to incinerators is that data are usually based on test burns that are carried out under optimal conditions. It is likely that such data underestimate releases under operational conditions (see further section 5.1.1), (Webster and Connett 1990).

Risk assessments on incinerators generally focus on only one or a few substances that are known to be emitted, in particular dioxins and selected heavy metals. However, in reality, releases from incinerators consist of complex mixtures of hundreds of chemicals including many unknown compounds with unknown toxicity. Risk assessment omits to take into account health impacts of many of the known chemicals and all the unknown chemicals (Johnston *et al.* 1996). In addition, in looking at just single chemicals, it does not address the issue of the combined toxicity of the chemical mixtures in stack emissions (Johnston *et al.* 1998). For instance, the combination of two or more chemicals together may cause an additive or even greater than additive (synergistic) effect or a less than additive (antagonistic) effect.

A further problem in risk assessment is that it is very difficult to determine which are the most appropriate and sensitive endpoints for detecting the toxicity of chemicals. An adverse effect on the immune system or respiratory system may, for instance, be more sensitive and be instigated at lower chemical concentrations than another sort of health impact. For health risk assessment on incinerators, toxicological endpoints can include both cancer and non-cancer effects on health. Whatever endpoint is chosen, it is thereby accepted as a key metric capable of being used to protect human health in an holistic manner. It is questionable, however, whether the correct endpoints are ever used in risk assessment. In addition, as discussed above, the developing young are likely to be more sensitive to some adverse chemical effects than adults.

In summary, there is a dauntingly wide spectrum of inadequacies and uncertainties inherent in the process of risk assessment, from the estimation of type and quantity of pollution, to estimates of exposure and health effects. Each of these problems alone can fatally compromise risk assessment procedures. It is particularly important that these limitations are recognised when risk assessment is applied in the formulation, implementation or enforcement of regulations. Risk assessment should be viewed with deep scepticism unless all the areas of uncertainty are explicitly defined (Johnston *et al.* 1998). Risk assessments that have been performed for incinerators are briefly discussed in section 3.3.

2. OCCUPATIONAL HEALTH IMPACTS

2.1 Exposure

Municipal incinerator workers have considerable exposure to incinerator ash and this raises the possibility that they might absorb significant quantities of dioxins, and other toxic substances present in ash. The greatest potential for exposure to the toxic components of ash occurs during ash cleaning operations (Schecter *et al*. 1991). Respiratory personal protective equipment is designed to give workers protection from pollutants although it is important to note that dioxins and many other contaminants are also absorbable through the skin.

According to the NRC (2000):

"*Incinerator operators and maintenance workers, and those involved in the collection, transport, and disposal of fly ash and emission control equipment residues, have the potential to be most exposed to toxic substances associated with incineration*."

Noting that "*incinerator workers have been exposed to high concentrations of dioxins and toxic metals, particularly lead, cadmium, and mercury,*" the NRC assigned its highest level of concern to incinerator workers, irrespective of the implementation of maximum achievable control technologies (NRC 2000).

Studies on exposure of incineration workers are limited in number and in their focus. The majority have investigated exposure to dioxins and a few heavy metals whilst two studies investigated other organic compounds. Nearly all the studies investigated MSW incinerators but not other types of incinerator.

2.1.1 Dioxins

Research has indicated that incinerator workers can be exposed to elevated levels of dioxins in workplace air. Studies published on incinerator workers themselves during the 1990s also implied that workers have suffered from exposure to dioxin levels in the workplace that were above background levels. Specifically, some studies reported elevated levels of dioxins (total TEQ) in workers' blood. Other studies did not find an increase in total dioxins but did find an increase in certain dioxin congeners. Overall, these studies demonstrated that workers at incinerators could be subjected to increased exposure to dioxins.

A study in the US at a refuse-derived fuel, coal co-fired incinerator showed that incinerator workers are exposed to higher than background levels of dioxins in air in the workplace (Pilspanen *et al.* 1992). Levels of dioxins in workplace areas were higher than in other ambient air concentrations from the region. The source of these pollutants was thought to be associated with particulate matter emitted through combustion backpressure or leakage from the boiler, and subsequently carried by air currents through the plant. Commenting on this study, Marty (1993) notes that it demonstrated that worker exposure to dioxins is considerable in the incinerator occupation compared with exposure of the general population. The National Institute for Occupational Safety and Health (NIOSH) investigated three MSW incinerators in New York in 1992. They determined that airborne concentrations of dioxins during a cleaning procedure (of the lower chamber) were high enough to exceed the protection capabilities of the protective respiration equipment worn by the workers during this operation (NIOSH 1995). The study concluded that cleaning operations at the incinerators poses a health hazard.

Kitamura *et al*. (2000) demonstrated that the average dioxin concentrations in the blood of workers was 3.7 times higher than the levels measured in nearby residents at an incinerator in Japan. The incinerator operated between 1988 until 1997 when it was closed due to high stack emissions of dioxins and contamination of local soil. The study measured dioxin concentrations in the blood of 94 workers. The level of dioxins in blood samples ranged from 13.3 to 805.8 parts per trillion (ppt) TEQ, with a mean value of 93.5 ppt TEQ (lipid basis). Workers who had cleaned the inside of incinerators had the highest dioxin levels. In addition, contact with fly ash was identified as an important factor for high dioxin levels in blood. Workers who had no direct contact with fly ash had lower blood levels (average 34.2 ppt TEQ). In comparison, the average concentration of dioxins determined in blood from residents living within 2 km of the incinerator was 25.3 ppt TEQ. Co-planar PCBs were also monitored in the study. Some workers showed high PCB levels (range 3.1 to 54.2 ppt TEQ) and the study noted that environmental measurement of PCBs might be necessary. Health impacts observed in workers from this study are discussed below in Section 2.2.2.

A study at a municipal waste incinerator in Germany was undertaken in which blood samples were taken from 56 male workers, and the level of dioxins was measured in a pooled (combined) blood sample (Schecter *et al.* 1991). The sample was compared to a pooled blood sample taken from a control group of males who had no known exposure to toxic materials. Results showed that the incinerator workers had a 30% higher level of dioxins (total TEQ) in blood compared to the control group. Dibenzofurans were particularly elevated (103 ppt in workers versus 47 ppt in controls). In addition, the congener profile (pattern of levels of the different dioxin congeners) in the workers blood was similar to the congener profile of incinerator ash. This implied, together with the elevated levels of dioxins in workers blood, that workers had been exposed to dioxins

in the workplace. These findings subsequently led to the implementation of more stringent worker protection methods at the plant (Schecter *et al.* 1994).

A study in Japan on two workers who had been employed for over eight years, at what is now an obsolete MSW incinerator, found blood levels of dioxins were still elevated several years after their employment ceased (Schecter *et al.* 1999). The concentrations of dioxins in the workers blood was extremely high (360 ppt TEQ and 278 ppt TEQ, lipid basis). By comparison, the average Japanese blood level is 24 ppt TEQ, making levels in the two men to be 15 and 11.5 times higher than the general population. Dibenzofurans accounted for most of the TEQ. The incinerator had burned household waste including PVC and other plastics. It is of note that elevation of dibenzofurans rather than of dibenzodioxins has been reported more frequently for such combustion processes. One of the workers wives also had elevated dioxins in her blood (98 ppt TEQ), but the wife of the other worker did not have elevated levels (18 ppt TEQ). It was proposed that the high level in one of the women might have resulted from her washing contaminated clothing brought home by her husband.

In contrast to the above studies, research at some incinerators has not always found elevated levels of total TEQ dioxins in workers blood, but has nevertheless found elevations in certain dioxin congeners, in particular, hexa- and hepta-dibenzofurans. A study of 10 workers from a MSW incinerator in Germany (Papke *et al*. 1993) found elevated levels of these congeners together with total dioxins in some of the workers. Another study on four workers from a MSW incinerator in the Netherlands reported elevated levels of these congeners as well as of hepta- and octa-dibenzodioxins as compared to the blood fat of five local residents (van den Hazel and Frankort 1996). The values reported show that the average concentration of hepta-dibenzodioxins was about three times higher in the workers; octa-dibenzodioxins, 1.7 times higher; hexa-dibenzofuran, almost two times higher; and hepta-dibenzofuran, 1.9 times higher.

A study on 31 workers from 3 chemical waste incinerators in Germany, by contrast, did not find elevated levels of dioxins in workers blood (Papke *et al.* 1994). Levels of dioxins in blood were within the normal range. However, two workers were identified who showed elevated levels of hepta- and hexa-dibenzofurans as described in the studies above.

2.1.2 Other Organic Compounds

A study was undertaken on workers at an incinerator in Germany regarded as having modern health and safety standards (Wribitzky *et al.* 1995). This investigated 45 workers who were in contact with the incinerator and others who were not, namely 54 periphery workers and 23 management workers. The biological exposure limits set in Germany were not exceeded for benzene, toluene or xylene, although levels of these substances were elevated above general population levels for some employees. Significantly higher toluene concentrations, however, were detected in blood of incinerator workers relative to other employees. In addition, higher levels of chlorophenols were found in incinerator workers compared to other employees, suggesting occupational exposure among these workers. The degree of elevation of both toluene and chlorophenols were nevertheless small and were not considered by the authors as relevant to occupational health.

NRC (2000) reviewed a study by Angerer (1992) which investigated levels of various organic chemicals in the blood and urine of 53 workers from a MSW incinerator in Germany compared to 431 control subjects. The study reported elevated levels of hexachlorobenzene in plasma and of chlorophenols in urine. Workers had significantly higher levels of 2,4-and 2,5-dichlorophenol, and 2,4,5-trichlorphenol, and of plasma hexachlorobenzene (HCB). These chemicals were analysed because they are dioxin precursors compounds. Of other chemicals detected in urine, 4-monochlorophenol and tetrachlorophenol, were higher in the control group and there were no significant differences between workers and the control group for levels of plasma PCBs, blood benzene and urinary 2,4,6-trichlorophenol and pentachlorophenol. The NRC review commented that due to the lack of consistent findings between the worker and control groups, no conclusion could be drawn from this study on the exposure to combustion precursors of dioxins and hence no inferences could be drawn concerning exposure to dioxins. However, it is possible that workers suffered increased exposure to PAHs based on evidence of a biomarker of exposure (see section 2.1.4).

2.1.3 Heavy Metals

Studies have been carried out in order to investigate whether workers may be exposed to elevated levels of some heavy metals in air in the workplace. Three studies on incinerator workers have suggested that workers had experienced increased exposure to certain heavy metals, but one study, by contrast, found little evidence of increased exposure.

NRC (2000) describe a study performed by the National Institute for Occupational Safety in 1992 at three MSW incinerators in New York that investigated levels of heavy metals in the workplace (NIOSH 1995). The airborne concentrations of aluminium, arsenic, cadmium, lead and nickel during some periods of the clean-out of the electrostatic precipitator were high enough to exceed the

protection capabilities of the air purifying respirators worn by the workers during these operations. This led to the conclusion that working during these clean-out operations at incinerators poses a health hazard.

In 1989, a study on incinerators in New York found that, in some cases, workers were exposed to high levels of lead in air. Following this report, a study was conducted on the levels of lead in workers blood at three New York incinerators (Malkin *et al.* 1992). Results showed that the average lead level was statistically significantly increased in workers compared to a control group of workers (mean 11.0 mg/dl versus 7.4 mg/dl), although it did not exceed the maximum US limit that is deemed acceptable in the workplace of 40 mg/dl. The study suggested that the presence of lead in incinerator ash is capable of increasing lead concentrations in workers blood. In contrast, a study on a German incinerator with very modern health and safety standards did not find that lead was elevated in workers blood (Wrbitzky *et al.* 1995).

A study by Bresnitz *et al.* (1992) was conducted on exposure to heavy metals and health impacts amongst 86 MSW incinerator workers in the US. This revealed that these substances were generally not elevated in the worker's blood and urine. Only 8 of the 471 tests for heavy metal exposure showed levels of heavy metals that were elevated above the expected levels for an unexposed population. These included zinc, mercury and lead. However, the elevated levels were not related to workers exposure categories and were not deemed to be clinically significant.

A study on a hazardous waste incinerator in Finland assessed levels of mercury in 11 workers in 1984 and again in 1994 (Kurttio *et al.* 1998). Results showed that during the ten-year period, mercury levels, measured in hair, increased in the workers from 0.62 to 0.98 mg/kg. However, mercury levels were not high in workers in comparison to unexposed population levels worldwide (0.5 – 4.0 mg/kg in hair). A study of workers at an incinerator in Germany that has notably high safety standards also did not find that background levels of mercury in workers were exceeded significantly. Nonetheless, it was found that concentrations of arsenic were elevated above background in several individuals from both the incinerator itself and in periphery and management workers. (Wrbitzky *et al.* 1995). Concentrations were highest in the incinerator workers. The study concluded that the elevation probably occurred as a result of exposure at work. Arsenic is highly carcinogenic and the study commented that the source must be identified so that exposure to workers could be reduced.

2.1.4 Biomarkers

Two studies used biomarkers to investigate exposure of workers to hazardous chemicals. One study indicated that workers might be exposed to increased levels of PAHs and another showed that they might be exposed to elevated levels of electrophilic compounds such as PAHs.

NRC (2000) reviewed a study by Angerer (1992) which investigated levels of various organic chemicals in the blood and urine of 53 workers from a MSW incinerator in Germany compared to 431 control subjects (see also above, section 2.1.2). The study reported elevated levels of hydroxypyrene in the urine of workers. Hydroxypyrene is known to be a good indicator of internal exposure to PAHs. The results showing higher hydroxypyrene in workers urine suggest, therefore, that workers had suffered higher exposure to PAHs.

A study at a chemical waste incinerator was conducted which examined thioether concentrations in the urine of workers (Van Doorn *et al.* 1981). Thioethers excreted in the urine are the end products of detoxification of electrophilic compounds in the body such as PAHs and benzene. They can therefore be used as a biomarker to indicate the extent to which an individual has been exposed to electrophilic compounds. The study tested the urine of 3 workers from the incinerator both before work and after work. Results were compared to urine samples taken at the same times from non-exposed men at the plant. The study revealed that the level of thioethers in the urine of the incineration workers was consistently higher following work compared to the start of the working day. This pattern of thioether excretion was not observed in the non-exposed control subjects. Moreover, the level of thioethers in urine in the incineration workers after work was consistently higher than thioethers in control subjects. From these results the study concluded that incineration workers were likely to inhale or absorb electrophilic compounds while at work, which were subsequently metabolised and excreted as thioethers in urine.

2.1.5 Mutagenic Compounds

Mutagenic compounds, also termed mutagens, are compounds that have the ability to damage DNA in cells of the body. Studies have shown that mutagenic compounds are present in incinerator emissions to air and in incinerator ashes. According to Ma *et al*. (1992), mutagenic compounds that are present in incinerator releases of gases, particulates and in ashes inevitably result in the exposure of incinerator workers to these compounds. One study on incinerator workers, discussed in this section, indicated that internal exposure to mutagenic compounds in the workplace might occur.

Laboratory studies have shown that incinerator emissions to air (e.g. Fomin and Hafner 1998, DeMarini *et al.* 1996), and fly ashes and bottom ashes (e.g. Shane *et al.* 1993), are mutagenic. Incinerator emissions to air consist of a complex mixture of organic chemicals and other elements. Within the organic fraction, it has been proposed that mutagenicity may be due to just one or a few chemical classes present in the chemical mixture. A recent study identified PAHs and nitroarenes (nitro-aromatic hydrocarbons) as being important contributors to the mutagenicity of incinerator emissions (DeMarini *et al.* 1996).

To investigate exposure to mutagens in workers, Scarlett *et al*. (1990) conducted a study to determine whether mutagenic compounds were present in the urine of incinerator workers. The study found that municipal waste incineration workers had a significantly increased frequency of mutagenic compounds in their urine compared to a control group of water treatment workers. Further research on workers from the same incineration plants was conducted to determine whether the quantity of mutagens in urine was consistently elevated in the workers or varied with time (Ma *et al.* 1992). The first in a series of tests again showed significantly increased mutagens in the urine of incinerator workers compared to water treatment workers. However, further tests showed lower frequencies of mutagens in urine of the incinerator workers. At the same time, the quantity of mutagens in the urine of water workers remained consistent. The study noted that likely explanations for lower amounts of mutagens in the urine of incineration workers on second and third testing was that exposure to mutagens in the incinerator workplace is highly variable. Alternatively, or in addition to this, the workers who were being investigated may have taken extra measures to reduce their exposure by wearing protective clothing and masks after they suspected that they were being exposed to toxicants. Although mutations play a role in the process of carcinogenesis, the study noted that the presence of mutagens in the urine of incinerator workers did not establish per se, that mutations were taking place. Hence, it was not possible to relate these findings to an evaluation of the likelihood of cancer or other adverse health outcome through this particular study although the findings are suggestive.

2.2 Health Impacts

Studies on mortality and morbidity (illness) among incinerator workers are very limited innumber. Research has reported a broad range of health impacts associated with working at incinerators including death from heart disease and certain cancers and hyperlipidemia, allergy and hypertension. One study reported chloracne in a highly exposed worker, a condition specifically associated with dioxin exposure.

2.2.1 Mortality

Gustavsson (1989) investigated mortality among 176 incinerator workers who were employed for one year or more between 1920 and 1985 at a municipal waste incinerator in Sweden. The study noted that the working environment at this incinerator was more contaminated than would be expected at modern incineration plants. Results revealed an excess of ischemic heart disease. It was calculated that the excess was caused by occupational factors. The excess was found to be highest and (statistically significant) in workers with more than 40 years exposure.

An excess of deaths from lung cancer was also found in the study. Compared with national rates of lung cancer in Sweden, the workers had a 3.5-fold higher probability of dying from lung cancer. Compared with local lung cancer rates they had a 2-fold higher likelihood of dying from the disease. The small sample size in the study precluded fully conclusive statements with respect to the statistical significance of the rate of lung cancer among workers (Marty 1993). Gustavsson *et al.* (1993) point out, however, that analysis of the exposure time and latency period suggested that it was improbable that the high rates of lung cancer among workers were due to smoking. Additionally, according to calculations from previous studies, only very excessive smoking habits (100% smokers) could produce the size of cancer excess seen in this study. It was noted that exposure of workers to polycyclic organic compounds, especially PAHs, may have been be an important factor in the lung cancer excess.

The study also found a 1.5-fold increased probability of death from oesophageal cancer. Taken in the context of the study on its own, the evidence for an occupational origin of oesophageal cancer was weak. However, other studies on workers in Sweden exposed to combustion products, for example, gas workers, chimney sweeps and bus garage workers have also reported an excess of oesophageal cancer. Considering the results of excess oesophageal cancer among incinerator workers in the context of these other studies supports the existence of an increased health threat due to occupational exposure. It appears that this increased threat cannot be attributed to cigarette smoking and alcohol consumption which are both known contributory factors (Gustavsson *et al.* 1993).

In contrast to the above study by Gustavsson (1989), a study on 532 workers employed at two municipal waste incinerators in Rome, Italy, between 1962 and 1992 did not find an excess of lung cancer (Rapiti *et al.* 1997). Mortality from lung cancer was reduced in comparison to the general population and cancer mortality from all cancers combined was similar to that of the general population. However, there was a 2.79-fold increased likelihood of dying from

gastric cancer among the workers. This excess probability was evident for workers who had more than 10 years latency since first employment. Increased gastric cancer has also been observed among sewage workers and, to some extent, incinerator workers have similar occupational exposures such as to inhalation of volatile pathogens, bacterial toxins and organic dust. There are also other contributory factors for gastric cancer including alcohol consumption, deficient intake of fruit and vegetables and lower socio-economic status. Such factors could have been present in the incinerator workers in this study and may explain the excess of gastric cancers to some extent. The study concluded that incinerator workers deserve increased surveillance by means of epidemiological studies and the role of dust and bacterial toxins in waste management requires further investigation.

2.2.2 Morbidity

Kitamura *et al.* (2000) investigated morbidity in 94 municipal incinerator workers who had worked at an incinerator in Japan. The incinerator had operated between 1988 and 1997 when it was closed due to high stack emissions of dioxins which resulted in contamination of local soil. The study found elevated dioxin levels in the blood of workers (range 13.3 to 805.8 ppt TEQ, mean 93.5 ppt TEQ (lipid basis) (see also section 2.1.1). At blood dioxin levels above 100 ppt TEQ, a statistically significant relationship with hyperlipidemia was found. In addition, there was a marginal correlation between dioxin levels and allergy. However, the study noted that these health conditions were self-reported by the workers and that confirmation of the diagnosis may be necessary because there was no association between dioxin levels and plasma lipid levels.

Investigations of blood biochemistry did not find any significant associations with dioxin levels in the blood of workers although some decreased liver function was noted. Tests for the immune system revealed significant associations between dioxin blood levels and natural killer (NK) cell activity and PHA stimulation. Dioxin has previously been associated with effects on the immune system and the authors noted that consequently a follow-up study was necessary.

The study also investigated the sex ratio of children born to workers. Theoretically the number of female and male children born should be equal, but in reality there is a very slight male excess (see discussion section 3.2.3). In this study, when the workers were divided into high (greater than 49 ppt TEQ dioxins in blood) and low (less than 49 ppt TEQ) exposure groups, there were 16 boys and 17 girls born to the low exposure workers. This compared to 2 boys and 5 girls among the high exposure group. The slight excess in the number of females born in the high exposure group was not, however, considered to be statistically significant.

A study by Bresnitz *et al.* (1992) was conducted on morbidity amongst 86 MSW incinerator workers in the US. The study investigated several different health parameters. It divided the workers into two hypothetical groups, those considered to experience high exposure in the workplace and those with low exposure. Results revealed an excess of workers (31%) who had urinary abnormalities, namely significant proteinuria (protein in the urine). For this effect, however, no difference was apparent between the high and the low exposure groups. The prevalence of hypertension was also above normal among the workers and it was suggested that the hypertension might explain the increased prevalence of proteinuria. Tests on lung function in the workers showed that this parameter was affected by smoking status. Tests also suggested an increased potential for small airway obstruction of the lungs although the diagnosis was not confirmed. The high exposure group had a 19% increased likelihood of possible small airway obstruction of the lungs compared to the low exposure group. Among workers who had never smoked in the groups, there was an 85% increased potential for small airway obstruction in the high exposure group. The study concluded that additional studies were needed to assess the potential health effects of MSW incinerator by-products. The authors also suggested that increased efforts in reducing personal risk factors and potential occupational exposures were needed in order to reduce morbidity among incinerator workers.

Schecter *et al.* (1999) found particularly high dioxin levels in the blood of two men several years after they had worked at an old Japanese waste incinerator (see also section 2.1.1). One worker, who had a dioxin level of 360 ppt TEQ, had chloracne, a skin condition caused by exposure to dioxin. The other worker, who had a dioxin level of 278 ppt TEQ, did not have chloracne. At the time of the investigation he was recovering from two episodes of gastrointestinal cancer of unknown aetiology (medical cause).

3. HEALTH IMPACTS ON POPULATIONS LIVING NEAR TO INCINERATORS

Hens *et al.* (2000) note that incinerator release of pollutants to air and to water is effectively a dilution and dispersal of pollutants over space and time. This causes a slow, but gradual accumulation of pollutants in the food chain and the human body, such that health effects may often only become visible and measurable after a long latency period.

3.1 Exposure Studies

A limited number of studies have been conducted to determine whether individuals residing near to incinerators have been exposed to pollutants. Studies are restricted to investigations of exposure to dioxins and heavy metals. Results of these studies are mixed. Some reported elevated exposure among nearby residents while others found no evidence of increased exposure.

3.1.1 Dioxins and PCBs

Three studies have reported increases in dioxin levels in residents living near to incinerators, while two studies have found no evidence of increased exposure. One study also reported that certain PCB congeners were possibly increased in the blood of child residents.

Gonzalez *et al.* (2000) investigated exposure in residents living in the vicinity of a newly built incinerator in Mataró, Spain, both before and two years after the plant started running. The study determined the level of dioxins in 1995 and again in 1997 in pooled blood samples from 104 individuals living between 0.5 and 1.5 km from the incinerator and from 97 individuals living between 3.5 to 4.0 km away. In 1995, prior to start up of the incinerator, dioxin levels in blood of those living near and those living farther away were 13.5 ppt TEQ and 13.4 ppt TEQ, respectively. In 1997, after 2 years of operation of the incinerator, dioxin levels had increased in both groups of people by about 25% and PCBs increased by about 12%. When further repeated analyses were conducted, the increase in dioxins among residents was of the order of 10 to 15%, rather than 25%. The increase in dioxins was not different in the residents living near to the incinerator and those living further away and the authors commented that the increase in dioxin blood levels was unlikely to be attributable to the incinerator. The incinerator's dioxin stack emissions were reported as 0.98-2.5ng TEQ/m^3.

A study in Japan was undertaken in an area close to a MSW incinerator for which high local dioxins in soil (see section 4.2.1), and an unusually high rate of cancer (2-fold excess) among residents, had been reported (Miyata *et al.* 1998). The study tested blood samples from 13 women and 5 men living within 2 km of the incinerator. Levels of dioxins were raised considerably in the residents compared to background levels found in the general population. For instance, women had an average blood level of 149 pg TEQ/g lipid and men 81 pg TEQ/g lipid, whereas the background level for the general population is in the range of 15 to 29 pg TEQ/g lipid. The authors commented that increased exposure in the residents was considered to be due to direct inhalation of dioxins from the stack gas of the incinerator and by intake of local vegetables contaminated by stack gas.

Following reports of high levels of dioxins in cow's milk at farms near to Coalite chemicals, in Derbyshire, UK where an incinerator was operating prior to 1991 (see also section 4.2.2), a study was undertaken on levels of dioxins in the blood of 10 residents from the farms (Startin *et al.* 1994). Results of the study revealed elevated levels of dioxins in blood of all the residents. Their blood levels were compared to available data on background dioxin levels for the German population since no relevant UK data were available. Three of the residents had blood levels (49, 85 and 95 pg TEQ/g lipid) which were just above or at the upper end of background levels, and the other 7 residents had levels (137-291 pg TEQ/g lipid) that were unmistakably higher than background levels.

Holdke *et al.* (1998) analysed levels of PCBs in the blood of 348 children between the ages of seven and ten years who lived near a hazardous waste incinerator in Germany. The results were compared to a control group of children who lived in a region with similar industrial pollution and with a second control group of children who lived in an area with lower industrial pollution. Among those who lived in the vicinity of the hazardous waste incinerator, PCB 170 and PCB 180 were present at statistically significant higher concentrations, while PCB 183 and PCB 187 were detected with greater frequency than among the control children from the area with lower industrial pollution. According to the study, while the results can only be viewed as a regional comparison of the three groups and the effects small, the statistically significant results do indicate a regionally plausible pattern.

Two other studies in Europe have found no increase in levels of dioxins of individuals residing close to incinerators. Deml *et al.* (1996) sampled blood from 39 persons and breast milk from 7 persons in 1993 who lived near to a MSW incinerator in Germany. The study reported that there was no indication of increased blood levels of dioxins in the residents. Levels of dioxins in the blood (mean 17.0 ppt TEQ lipid, range 5.2 to 34.5 ppt TEQ lipid) and breast milk (mean 12.4 ppt TEQ lipid, range 6 to 19 ppt TEQ lipid) of residents were not significantly different from background levels in the German population (range 10 to 48 ppt TEQ lipid in blood and mean 30 ppt TEQ lipid in breast milk).

Similarly, a study on exposure of a limited number of residents (five) living near to an incinerator in Duiven, The

Netherlands, did not find increased dioxin levels in their blood (van den Hazel and Frankort 1996). This study was undertaken specifically to test whether residents had elevated levels of dioxin congeners in their bodies because of their potential exposure to fly ash blown away from the storage site near the waste incinerator. Levels of dioxins in blood of the residents (mean 31.4 ppt TEQ lipid) were similar to levels in the control group of 5 individuals from the Dutch general population (mean 33.8 ppt TEQ lipid). In addition, the study did not find increases in the levels of any particular dioxin congeners amongst the residents.

3.1.2 Heavy Metals

Only one study was located in the scientific literature on exposure to heavy metals in individuals residing near to incinerators. Kurttio *et al.* (1998) investigated changes in the level of mercury between 1984 and 1994 in the hair of 113 individuals who lived near to a hazardous waste incinerator in Finland. Mercury concentrations were found to increase in workers at the plant (see section 2.1.3) and in residents such that levels increased with decreasing distance from the incinerator.

For example, levels increased by 0.16 mg/kg in residents living 1.5-2 km (high exposure group) from the plant, 0.13 mg/kg at 2.5-3.7 km (medium exposure group) and 0.03 mg/kg at about 5km (low exposure group). The results indicated that the incinerator was the likely source of exposure among residents. Exposure was most likely mainly due to inhalation and possibly via ingestion of local well water and vegetables. The authors concluded that the increase in mercury concentrations in the residents over time was small and, on the basis of current knowledge, did not pose a health threat.

3.1.3 Biomarkers

The theory behind using biomarkers in epidemiology studies relies on early biological effects of a toxic exposure, (i.e. the biomarker), being more prevalent and easier to detect in a potentially exposed population than clinical disease.

A study conducted at a recently built incinerator in Spain, compared children living in it's vicinity to children living outside the zone of influence of the incinerator using urinary thioethers as a biomarker (Ardevol *et al.* 1999). The use of urinary thioethers as a biomarker relies on the fact that when electrophilic compounds such as PAHs are detoxified in the body, the metabolic end products can be detected as thioethers in the urine. Electrophilic compounds are generally potent mutagenic and carcinogenic compounds.

The study assessed the possible contribution of incinerator releases to urinary thioethers in children aged 7-10 during 1997. Research on children, rather than on adults, eliminates other potential effects on health that may interfere with study results, including smoking, occupational influences or other lifestyle toxicants. The study reported that there was a greater quantity of urinary thioethers in the urine of children living near to the incinerator than the control group of children living further away, although the result was not statistically significant.

The study also found that parental smoking of tobacco predicted a statistically significantly greater quantity of urinary thioethers in children of both groups. In addition, among children who were exposed to smoking by both parents in the home, there was a significantly greater quantity of thioethers in urine from the incinerator group compared to the control group. It is possible that this effect was caused by a greater extent of exposure to tobacco smoke in these children. Alternatively, it may have been caused by the addition of exposure to the tobacco smoke and exposure to incinerator releases. In the case of releases from incinerators, the higher amounts of thioethers in urine of the children may have been caused by exposure to PAHs and possibly dioxins.

3.2 Health Effects - Epidemiological Studies

The majority of epidemiological studies on the health of populations residing near to incinerators have focused either on incidence of cancer or respiratory symptoms. Additionally, some research has investigated other potential effects including congenital abnormalities and changes in the sex ratio. Considering the widespread use of incinerators on a global scale, the number of studies that have investigated health effects in residents near to these facilities is sparse.

3.2.1 Cancer

Some of the substances emitted from incinerator stacks, including cadmium, PAHs and dioxin (TCDD), have been classified as human carcinogens or likely/possible human carcinogens by the International Agency for Research on Cancer (McGregor *et al.* 1998, see Elliot *et al.* 1996). A number of studies have been undertaken on cancer incidence on populations living near to incinerators or other industrial sites. The majority of these studies have found an association between elevated rates of cancers and living close to incinerators or other industrial sites, including childhood cancer. Most research in this field necessitates consideration of exposure to material released from incinerators over a number of years because the time taken for cancer to develop (the latency period) is long for many cancers.

Soft Tissue Sarcoma and Non-Hodgkin's Lymphoma

A study in the area of Doubs, eastern France, was

conducted to investigate clustering of two types of cancer, soft tissue sarcoma and non-Hodgkin's lymphoma, near to a MSW (Viel *et al.* 2000). The study was undertaken following a report of high dioxin emissions from the incinerator. The study found highly significant clusters of both cancers in areas close to the incinerator but not in other surrounding regions.

In a press release in 1998, the French Ministry of the Environment revealed that 71 MSW incinerators had dioxin emissions to atmosphere above 10 ng I-TEQ/m^3. One of the incinerators, Bescançon, had a reported dioxin air emission of 16.3 ng I-TEQ/m^3. A general cancer registry covered the area local to this incinerator and this provided an opportunity for researchers to study the incidence of cancer in the region. Soft tissue sarcoma and non-Hodgkin's lymphoma were selected for investigation because previous work has suggested that dioxins increase the probability of contracting these cancers. The incinerator had operated from 1971 onwards.

The study divided the region of Doubs into 26 areas (statistical units) for the purpose of the analysis. During the period 1980 to 1995, 110 cases of soft tissue sarcoma and 803 cases of non-Hodgkin's lymphoma were recorded. Analysis showed that statistically significant clusters of both cancers were present in 2 of the 26 areas, Bescançon and Audeux that were closest to the incinerator. There was a 44% increased incidence of soft tissue sarcoma and a 27% increased incidence in non-Hodgkin's lymphoma. No other clusters were found in the remaining 24 areas. Possible confounding factors of socio-economic status and urbanisation were discussed as unlikely to bias results. Furthermore, to make sure that distance to health centres did not confound the results (that is, due to closer residence and the consequent wider access to specialised care leading to more frequent cancer diagnoses), the study also considered the frequency of Hodgkin's disease as a control cancer. Hodgkin's disease is a cancer that is not associated with dioxin exposure. The study found no clusters of Hodgkin's lymphoma within the entire study area. The authors concluded from this that the clusters of soft tissue sarcoma and non-Hodgkin's lymphoma near the incinerator were not attributable to the presence of University Hospital in the Bescançon -Audeux area resulting in more reliable diagnosis of the diseases.

In conclusion, the authors stated that the consistency of the findings for clustering of soft tissue sarcoma and non-Hodgkin's lymphoma around the incinerator was remarkable. However, they cautioned that before clusters of both these cancers are attributed to dioxin release from the incinerator, the findings should be confirmed by further investigation. If dioxin is involved, the route of exposure among residents remains to be determined.

Lung Cancer

A study in Trieste, an industrialised city in northeast Italy, was undertaken to investigate the impact of air pollution from a number of sources (ship yard, iron foundry, incinerator and city centre) on lung cancer (Biggeri *et al.* 1996). It found that the prevalence of all types of lung cancer was increased both by residence close to the incinerator and to the city centre.

The method used in this study involved the identification of individual subjects who had died from lung cancer in the region, and subsequently, the identification of matched control individuals who had died at a similar time but not from cancer or lung disease. A total of 755 male individuals who had died from lung cancer between 1979 and 1981 or between 1985 and 1986 were identified. The two enrolment periods were selected to cover an extended period of time bearing in mind the costs of the study. Analysis of results accounted for confounding factors including smoking habits, age, likelihood of exposure to carcinogens in the workplace, and approximate levels of air particulate.

Results of the study showed there was a statistically significant increased probability of dying from all types of lung cancer among people living by the incinerator. The likelihood of dying from lung cancer for individuals living within a short distance of the incinerator was 6.7 times greater than for those living in other areas. Independently from this, living close to the city centre was also associated with a higher probability of death from lung cancer (2.2-fold higher at the city centre). This study confirmed the findings of a previous study in Trieste, which had also identified an increased likelihood of lung cancer in the neighbourhood of the incinerator (Babone *et al.* 1994). Some possible confounding of results through factors such as length of time at the death address, (i.e. change of residence), could not be excluded. The study concluded that the results provided further evidence that air pollution is a moderate contributory factor for lung cancer and that this was consistent with the hypothesis of an independent impact on health of residing close to the incinerator and to the city centre.

Cancer of the larynx

An investigation of cancer rates around an incinerator of waste solvents and oils at Charnock Richard, Lancashire, UK was undertaken during the late 1980's by the local council. Statistical analysis of the results identified a significant excess of cancer of the larynx close to the incinerator, which decreased as distance from the incinerator increased (Diggle *et al.* 1990). Following this report, another study was undertaken to investigate incidence of laryngeal cancer at this incinerator and at nine other similar incinerators in the UK which began operation before 1979 (Elliot *et al.*

1992). This study found no excess of larynx or lung cancers up to 10 km from the incinerator sites when lag periods (between incinerator start up and cancer incidence) of 5 and 10 years were used. Consequently, the study concluded that the apparent cluster of cases of cancer of the larynx at Charnock Richard, Lancashire, was unlikely to be due to the incinerator. However, there were several recognised limitations in the data used in this study. For instance, the lag time of 5 and 10 years for the development of laryngeal cancer is short considering the epidemiology of solid tumours. A study on mustard gas workers, for example, showed that cancer of the larynx was evident only after a follow-up of at least 10 years since first employment and another study showed excess mortality from larynx cancer in workers exposed to dioxin was only apparent after 20 years.

A more recent study on the incidence of various cancers in a population living within the vicinity of an incinerator, a waste disposal site, and an oil refinery plant in Rome which had operated since the early 1960s, did find an increased probability of mortality from cancer of the larynx (Michelozzi *et al.* 1998). The investigation was undertaken following concerns about pollution from the industries affecting the resident population. No excesses were found for liver, lung and lymphohaematopoietic cancers. However, an increased likelihood of cancer of the larynx was evident at 0-3 and 3-8 km distance from the industries, although the result was not statistically significant. Nevertheless, the authors hypothesised a possible link between emissions from the industries and larynx cancer since there was a statistically significant decline in this cancer among men with increasing distance from the industries. The study noted that this was interesting because results from other studies on cancer incidence in the vicinity of these industries were conflicting. It concluded that the results for laryngeal cancer were based on a limited number of cases, and further studies would be necessary to determine whether the presence of refineries or incinerators actually does represent a factor contributing to an increased probability of contracting this disease in resident populations.

Liver Cancer and Other Cancers

A study was undertaken on cancer incidence in individuals living near to incinerators in Great Britain as a consequence of concerns about possible health effects of residing close to these facilities (Elliot *et al.* 1996). The research showed a statistically significant excess of liver cancer among residents.

The study investigated cancer incidence among over 14 million people living within 7.5 km of 72 MSW incinerators. Data on cancer incidence among residents from 1974-1987 was compiled using the national cancer registration scheme. Cancer incidence rates for populations living close to incinerators were compared with national cancer rates to determine whether there was an excess of observed numbers of cancer cases versus expected numbers. Results showed that there were statistically significant excesses of all cancers combined and of stomach, colorectal, liver and lung cancer for populations living within 7.5 km of incinerators. The incidence of cancer decreased with increasing distance from the incinerators. The greatest increased probability of occurrence was found for liver cancer, for which there was 37% excess from 0 to 1 km distance from incinerators compared to national rates. However, further analysis of the data indicated that the excesses of all cancers combined and of stomach and lung cancer were likely to be due to confounding, in this case social deprivation. Social deprivation tends to be high in polluted areas and it is strongly predictive of disease occurrence. With regard to liver cancer it was noted that social deprivation could account for at least part of the increased likelihood of contracting this disease as observed in the study. It was also noted that there was some misdiagnosis of primary liver cancer due to secondary liver tumours (i.e. tumours arising subsequently to, and as a result of, other types of primary tumours). The study concluded that further research would be needed to confirm whether or not there was an excess of primary liver cancer in the vicinity of incinerators. Further work on the diagnosis of liver cancer in this study was subsequently carried out (Elliot *et al.* 2000). It also indicated an increase in the rate of liver cancer in residents living near to incinerators.

The initial study (Elliot *et al.* 1996) utilised information recorded on death certificates. For further analysis of the data, the second study, (Elliot *et al.* 2000), included a review of histology slides and reports and medical records in order to clarify whether liver cancers were primary cancers or secondary cancers. Out of the 235 original cases of liver cancer recorded on death certificates a review of 119 cases (51%) was performed. Primary liver cancer was confirmed in 55% of these cases and secondary cancers in 18%. If these figures are used to re-calculate the incidence of liver cancer in the first study, the excess of 37% liver cancers (23 cases) reported in the first study is reduced to 12.6 cases, and 18.8 cases when only definite secondary cancers are excluded. This translates to 0.53 and 0.78 excess cases per 1,000,000 cases per year (or an increased probability of contracting liver cancer of 20 and 30% within 1 km distance of MSW incinerators). The study concluded that the true excess would be expected to lie somewhere between these two figures. The study could not rule out the possibility of confounding on the results from social deprivation. Elliot *et al.* (2000) commented that if the findings of excess liver cancers in this and the

previous study were caused by residence near to MSW incinerators, then the results related to historical exposure patterns around these installations.

Childhood Cancer

An analysis by Knox (2000) has recently been published that used data on MSW incinerators from the initial study by Elliot *et al.* (1996), (see above), to determine whether the likelihood of contracting childhood cancer is increased near to incinerators. The study considered childhood cancer around 70 MSW incinerators between 1974-1987, and 307 hospital waste incinerators between 1953-1980. Latent intervals for childhood cancers are short and this mitigates problems of the often-longer latency periods for cancer in adults encountered in the "all ages" study by Elliot *et al.* (1996). The analysis used a newly developed, sensitive method that could consider the distance of the birth address of each child from an incinerator and also the death address, if this was different. In this regard, the "migration method" used for this analysis could compare distances from incinerators to the birth addresses and to death addresses of children with cancer who had moved house. The study identified an increased incidence of child cancer in children who were born near to incinerators.

The developmental stages of life are generally the most vulnerable to toxic insult. Thus, exposure of the developing foetus in the womb and during the early stages of life to toxic substances may lead to a greater potential for adverse effects on health, such as cancer, than exposure in later years. In the study by Knox (2000), if exposures to toxic substances from living near to an incinerator during the early stages of life predicted an increase in cancer incidence, then there would be closer associations with the birth address of children rather than with the death address.

Results of the analysis indeed showed a highly statistically significant excess of migration away from birthplaces close to incinerators within 5 km of the sites. Thus, exposure to incinerators at the birth address, and hence during the early developmental stages of life, was associated with a higher probability of contracting cancer than exposure at the death address or exposure in later years. For children who were born within a distance of 5 km of MSW incinerators, there was a 2-fold increase in the probability of dying from cancer.

These results are also in agreement with previous research which showed an increased likelihood of childhood cancer in children who were born within a short distance of hospital waste incinerators, large-scale high temperature combustion sources or installations emitting VOCs (Knox and Gilman 1998). The excess numbers of leukaemias and solid tumours of all types were similar to those found in the study on childhood cancer in the vicinity of MSW incinerators by Knox (2000). This phenomenon was also observed in previous studies on proximity of childhood cancer to industrial sites and exposures to pre-natal medical radiation. Such a result would be expected for agents/chemicals that have systemic access (i.e. access via the circulation) to the DNA/RNA in all types of foetal cells (Knox 2000).

The study conducted by Knox and Gilman (1998) on the level of childhood cancers near to many different industries concluded that increased cancer rates were apparent in children born near to hospital incinerators, other combustion sources and industries emitting VOCs. From these results it was concluded that multiple toxic sources are responsible for many birth–time or pre-birth (foetal) initiations of cancer. This effect on the developing young is likely to be mediated through various VOCs and products of combustion. With regard to waste incineration itself, concordance of results on childhood cancer from MSW incinerators (Knox 2000) and hospital waste incinerators (Knox and Gilman 1998) suggests a common direct effect of being born near to incinerators and childhood cancer. Knox (2000) noted, however, that it is difficult to say whether the apparent cancer-related threats to health near incinerators might also stem from other hazards in the nearby environments. In this regard, the most "toxic" incinerators in the study were close to industrial sources of kinds implicated in previous studies. For this reason, the present overall conclusion of the study was that the increased probability of childhood cancer stems from residence near to large-scale combustion processes as a whole, of which incinerators are one component (Knox 2000).

3.2.2 Respiratory Effects

Incinerators, in particular cement kilns, emit considerable quantities of SO_2 and NO_2. Long term exposure to these substances is known to have negative impacts on respiratory health (see e.g. Ayres 1998). Similarly, incinerators emit fine particulate matter and many studies have reported that long term exposure to particulate matter is associated with adverse effects on respiratory symptoms (see further appendix A). Despite the potential negative impacts on respiratory health from substances known to be emitted from incinerators, there have been only a limited number of epidemiological studies on respiratory effects in individuals who live near to incinerators. Of the research that has been undertaken, some studies have suggested negative impacts on respiratory health whilst others have found no effect.

An early study by Zmirou (1984) suggested there was an increased use of medication for respiratory illnesses among residents living near to a MSW incinerator in a French village. The study found that medicines for respiratory

problems such as bronchodilators, expectorants and cough medicines were purchased significantly more often at distances closer to the incinerator. The investigators pointed out that it is not possible to conclude a cause-effect relationship from this study, but stated that the observation made by the study is consistent with the hypothesis that pollution generated by the incinerator may exacerbate respiratory problems (see: Marty 1993).

At a hazardous waste incinerator in western North Carolina, US, an investigation of the health of nearby residents was conducted following reports of illness and neurological symptoms in workers employed at the plant (ATSDR 1993). After adjustment for confounding factors such as age, sex and cigarette smoking, the study found significant increases in the prevalence of self-reported respiratory symptoms. For instance, residents living near to the incinerator were almost nine times more likely to report recurrent wheezing or cough compared to residents living further from the site, and they were almost two times more likely to report other respiratory symptoms. In addition, chest pain, poor co-ordination, dizziness and irritative symptoms were also significantly elevated. However, no difference between the two groups was reported for the prevalence of physician-diagnosed diseases and hospital admissions for these diseases. Although this study found an increase in respiratory symptoms among residents living near to an incinerator according to a review of this study by NRC (2000), there are several concerns surrounding the study, which limit the interpretation of the findings. For instance, there are concerns regarding the retrospective nature of the study (incinerator operated from 1977 to 1988 and the study was not conducted until 1991), and regarding adverse publicity before the incinerator shutdown. The NRC commented that the study was of limited utility in evaluating the effect of incinerator exposures.

A study in Taiwan investigated the respiratory health of children living near a wire-reclamation incinerator and reported associated adverse effects on lung function (Wang *et al.* 1992). The study tested 86 primary school children and compared the results to a control group of 92 children from a "non-polluted" city. Air pollution in the incinerator district as measured by SO_2 and NO_2, was notably greater than in the comparison city. Questionnaires administered to the children revealed no differences in respiratory symptoms. However, abnormal forced expiratory volume in 1 second (FEV1), a measure of lung function, was significantly greater in the incinerator group (17.5%) than the control group (3.2%). Further testing of lung function of 26 children from each group revealed a positive methacholine-challenge test in 9 of the incinerator group but only 1 of the control group. From these results, the authors concluded that the high level of air pollution to which the children living near to the incinerator were exposed was associated with a detrimental effect on lung function. A review of this study by NRC (2000) noted that the study appears to demonstrate that higher concentrations of air pollutants alter lung function in children, but does not directly allow any inference about the contribution of incinerators as opposed to other pollutant sources to the observed health effects.

In the US, it has become common practice for hazardous wastes to be used as part of the fuel to achieve high temperatures during cement kiln operation. A study conducted on cement kilns operating in Midlothian, Texas, documented statistically significant increased self-reported respiratory symptoms among a sample of nearby residents as compared to residents living further away (Legator *et al.* 1998). Risk assessments based on measurements of incinerator emissions recorded in 1997/8 in the area had reported that there was no threat posed to human health from the cement kilns (see Legator *et al.* 1998). In addition, a study by the Department of Health in the region in 1992 concluded that there

"*did not appear to be any consistent patterns of illness or symptoms that might be an indication of …a common-source health problem among the study respondents*".

However, subsequent analysis of both the above pieces of research concluded that there were deficiencies, flaws and inadequacies in the methodology of the studies. Since then, Legator *et al.* (1998) have conducted research aimed at identifying whether exposure of nearby residents to pollutants from the cement kilns resulted in adverse health effects.

A randomised sample of 58 individuals living near to the incinerators was selected and requested to undergo an interview based on an extensive health questionnaire. The results were compared to a control group of 54 individuals who lived further away from the incinerators. The study showed that no areas of health appeared to be significantly adversely affected with the exception of the respiratory category. The population living close to the incinerators reported significantly higher frequencies of respiratory symptoms ($p = 0.002$) than the control group. All of the respiratory symptoms on the questionnaire, including lung disease, wheezing, emphysema, persistent cough and bronchitis were elevated, the only exception being pneumonia. The study was not subject to some of the limitations that can hinder studies of this nature such as biases on questionnaire reporting. In addition, the control population was older than the incinerator-exposed population and since older individuals are more sensitive to the effects of chemicals, it is likely that impacts on the incinerator-exposed population were underestimated. The

study concluded that the results add to the growing body of information that persons living next to incinerator-generated air pollution experience increased prevalence of respiratory symptoms.

Gray *et al.* (1994) conducted a study on the incidence of asthma among two groups of children living near sewage sludge incinerators in Sydney, Australia. Respiratory illness was monitored by way of questionnaire, and various physiological tests, including tests on lung function. The study found no adverse effect on either the prevalence or the severity of asthma in the children compared to a control comparison group of children living in a different region of Sydney. In addition, no differences were apparent in lung function. Measurements of SO_x, NO_x, hydrogen sulphide, ozone (O_3) and particulate matter did not detect any statistically significant differences between the incinerator and comparison areas. The study concluded that releases from high-temperature sewage-sludge incinerators appeared to have no adverse effect on the prevalence or severity of childhood asthma.

A study in the US reported no significant difference in levels of particulate air pollution or respiratory health in communities residing near to three waste incinerators from 1992 to 1994 (Shy *et al.* 1995). The study was conducted on three communities living near to a municipal, hazardous and medical waste incinerator in North Carolina US and three comparison communities that were more than 3 km upwind of incinerators. The study simultaneously monitored air quality in the communities and respiratory health effects in individuals.

In all, 6963 individuals participated in a telephone survey of respiratory health over the course of 3 years and 100-144 individuals per community per year participated in having tests on lung function. Shy *et al.* (1995) reported results for the first year of the three-year study. The study found no significant difference in the concentration of particulate matter (PM10) in the incinerator communities relative to the comparison communities. Incinerators were calculated to contribute to less than 3% of particulate matter measured in the communities, the remainder coming from other sources around the region. However, consistently higher levels of particulate zinc, lead, and chloride were found in incinerator communities when winds were coming from the direction of the medical and MSW incinerators. The study noted that if a chemical component of incinerator releases can cause respiratory effects in an exposed community, standard measures of air pollution may fail to detect the relevant differences in human exposure.

With regard to respiratory health in the study, no significant differences were found in respiratory symptoms recorded by telephone survey between the incinerator and comparison communities. In addition, results of lung function from this study for 1992/1993, plus a subsequent more in depth analysis of lung function (Lee and Shy 1999) did not find any relationship between particulate levels (PM10) in the communities and lung function. Furthermore, there were no apparent differences in lung function between the incinerator and comparison communities. This is in contrast to previous studies which have reported increased respiratory effects associated with increases in PM10 (see appendix A). The different results of this study may be because the particulate levels were relatively low by comparison with previous studies and therefore effects on lung function might not be possible to detect even if they existed (Shy *et al*. 1995).

Although the above study (Shy *et al.* 1995) did not find an association between living near to the three particular incinerators in the study and an increase in acute or chronic respiratory symptoms, it was emphasised that the study did have several limitations. For instance, there was more cigarette smoking and greater use of kerosene heaters in the homes of the comparison communities and this would tend to mask any moderate-sized respiratory effects in the incinerator communities. Also, a major problem was the possible significant misclassification of exposure to pollutants from the incinerators because different sections of the community were likely to experience different air pollution levels due to the prevailing wind direction. It was reported that this would tend to lead towards the result of no effect upon respiratory health. Further analysis of lung function results by Lee and Shy (1999) also noted that the study was limited by a lack of information on individual exposures to releases. The authors commented that the lack of association between PM10 and respiratory health in the study must be interpreted cautiously because exposure estimation based on monitoring of ambient air likely resulted in misclassification of true exposure levels.

3.2.3 Sex Ratio

In humans, the ratio of males to females born should theoretically be 1:1. But in reality there is a slight excess of males at birth. This may be attributable to a number of different factors including age of parents and time of insemination within the cycle (Moller 1996).

The sex ratio has been found to vary somewhat between different countries. Abnormal sex ratios have been associated with some occupational environments, for instance, an excess of male births was reported for tax experts and chartered accountants and an excess of female births was reported for librarians and quantity surveyors (see Williams *et al.* 1992). The mechanisms which cause

sex ratio to vary have not been clarified but a hormonal influence has been implicated in having an effect.

Recent research has indicated a decreased proportion of male births in the general population of Denmark, Netherlands, USA, Canada, and in sawmill workers who were exposed to trichlorophenate that was contaminated with dioxins. It has been hypothesised that these changes in sex ratio may be caused by exposure to chemical pollution. There is some evidence for this from studies on populations who have been exposed to dioxins. For example, a study on the population at Seveso, Italy, who were exposed to high levels of dioxin (TCDD) following an explosion at a chemical herbicide plant in 1976 has investigated sex ratio (Mocarelli *et al.* 2000). Individuals were included in the study who were exposed at the time of the accident, whether as a child or as an adult, and have subsequently had children of their own. Exposure was assessed by analysing blood samples taken around the time of the accident and kept frozen in storage. The results showed that increased levels of dioxin in the blood of the fathers increased the probability of siring female children. The concentration in the blood of the fathers at the time of the accident was about 20 times the estimated average concentration of TCDD currently found in humans in industrialised countries, although the levels in blood had fallen in some cases by the time children were conceived. The study showed that male exposure to TCDD before and during puberty is linked to this sex-ratio effect of siring more females. This indicates that the time before and during puberty may be a very sensitive time for dioxin toxicity in men. Men in adulthood at the time of exposure were also affected. Overall, the data showed that exposure of men to TCDD is linked to a lowered male/female sex ratio in their offspring, which may persist for years after exposure.

One study has investigated sex ratios in populations living near to incinerators. This study was carried out on residents living in the vicinity of two incinerators in Scotland, UK (Williams *et al.* 1992). For the purpose of the study, the area was hypothetically divided up into 16 different districts (by postcodes) including 6 districts further away from the incinerators, which were used as a comparison area in the analysis of results. No difference was found in the sex ratios of births among residents between the potentially exposed area (comprising 3 districts) and the comparison area. However, when districts were considered individually, the area identified as being most vulnerable to air pollution from incinerators had a statistically significant excess of female births. Another district considered to be potentially vulnerable also had an excess of female births and another had an excess of male births although these were not statistically significant. The study proposed that incinerator releases such as polychlorinated hydrocarbons, dioxins and pesticides, if present, might have affected the sex ratios. However, the authors noted that it is not possible to attribute causality of increased female births to materials released by incinerators this study alone and proposed that more research was needed.

3.2.4 Congenital Abnormalities

Research on populations living near to incinerators has reported an increased incidence of congenital abnormalities. One study in Amsterdam found increased numbers of orofacial clefts and other midline defects in a community living near to an incinerator site used for the open burning of chemicals. Another study near to an incinerator in Belgium found an increased prevalence of congenital abnormalities. Other research on congenital eye malformations has not detected an increased prevalence near to incinerators.

Between 1961 and 1969, a poorly regulated incinerator carried out open-burning of waste chemicals in Zeeburg, Amsterdam. A recent analysis of data from the region has revealed a dramatic increase in the incidence of orofacial clefts in babies born after incineration began (ten Tusscher *et al.* 2000). In comparison, no increase in orofacial clefts during the same time period was observed in births from another area that was unaffected by the waste burning. For instance, the average incidence of orofacial clefts in Zeeburg between 1961 and 1969 was 2.5 per 1000 births compared to 1.2 per 1000 births in the comparison area. In particular, for the years 1963 and 1964, the incidence of congenital abnormalities at Zeeburg was respectively 5.1 and 7.1 per 1000 births. This result for 1963/4 was statistically significantly different to the comparison area.

The residential location of many of the women who gave birth to babies who had orofacial clefts was found to be situated in an area along a corridor of wind-flow from the incinerator. It is known that chemical exposure can be a cause of orofacial clefts and it was noted that dioxin (TCDD) is known to cause cleft palate in mice. The authors concluded that although a cause-effect relationship cannot be proven in this case, it seems very likely that there is a link between the open incineration of chemicals and the increased incidence of orofacial clefts in Zeeburg, Amsterdam for the years 1960 to 1969. Furthermore, as well as orofacial clefts, the majority of babies born in Zeeburg with some other midline defects were born in the area corresponding to wind-flow from the incinerator. These conditions included central nervous system defects (mainly spina bifida) and genital defects (mainly hypospadias).

A cluster of congenital abnormalities was discovered among inhabitants of the Neerland neighbourhood in the Wilrijk region, Belgium. This stirred up unrest in the local community. The area is situated between two municipal

waste incinerators, one at a distance of 1200 meters and the other at a distance of 800 meters. Research had previously shown that the area around Wilrijk was among the regions in Flanders that received the highest dioxin deposition. This was due to the incineration of municipal wastes between 1980 and 1996. Following concern by residents about the cluster of congenital abnormalities, two health studies were ordered by the government and took place between 1997-1998. The first (Verschaeve and Schoeters 1998) investigated genetic damage in chromosomes in certain types of blood cells (peripheral lymphocytes), and the second investigated children's health (Aelvo *et al*. 1998). Van Larebeke (2000) recently reviewed these studies.

The first study, on chromosomal damage, compared 24 children from the area with a control group of 20 children from another neighbourhood in the Antwerp region. The study did not detect any differences in chromosomal aberrations between the two groups. However, van Larebeke (2000) commented that if genetic effects were present, they would be expected to be of low intensity. The study did not have the statistical sensitivity to be able to detect such effects at low intensity. Therefore it is possible that such effects could be present but would not have been found by the study. If present, such genetic effects could have a significant impact on health.

The second study assessed health problems of children from Neerland. The study found an increased incidence of congenital malformations in babies from Neerland compared with incidence within Belgium as a whole, although the result was not statistically significant. The probability of giving birth to a baby with congenital malformation was 1.26 times greater for Neerland women than for Flemish women in general. There was also an increased incidence in congenitally malformed babies born in Neerland compared to babies who were born in the same clinics but whose families resided elsewhere. The increased probability of having a congenitally malformed baby appeared to be confined to children born to parents who had not resided for a long time in Neerland.

In addition to congenital abnormalities, the second study also investigated performance and health of children from Neerland at school compared to other children from a nearby area and to Flemish children in general. No differences were apparent in failure at school. However, increased allergies and repeated episodes of the common cold were significantly increased in Neerland children in the third class of maternal school, and increased complaints on health in general were also more frequent. At the age of 9, in third class of primary school, there was a significant increase in allergies and "use of medication". Use of medication is considered to be an indirect measure of ill health caused by pollution.

Van Larebeke (2000) concluded in a review of the studies that a more detailed in-depth analysis of the health status of children from the Neerland neighbourhood might reveal other health effects possibly related to pollution. For instance, data on individual exposure determined as blood levels of dioxins were lacking in the study as were data on early (pre-symptomatic) biological effects. The present results were considered to be sufficiently indicative to warrant further study to include investigation of these points. Both incinerators were shut down in November 1997 due to their exceeding the dioxin air emission standards and consequent considerations of public health (Nouwen *et al.* 1999).

In a review of incineration and health, Gatrell and Lovett (1989) discuss findings on congenital eye malformations in children born in the vicinity of incinerators. At two chemical waste incinerators in Scotland, UK, (owned by ReChem), there were reports in national newspapers of congenital eye malformations in children born near to the incinerators. However, government studies found no evidence of increased congenital eye malformations in children born in the vicinity of these plants, or another Rechem chemical waste incinerator in Wales. The government studies have, however, been questioned on accuracy because the database of eye malformations that was used has a voluntary rather than obligatory notification system and therefore some genuine cases may be missed out. Further research on this subject by Gatrell and Lovett (1989) investigated whether there was any evidence of clustering of eye malformations around incinerator sites in areas across England and Wales, but found no evidence of a link. Again, this study was limited by the database of registered congenital eye malformations.

3.2.5 Multiple Pregnancy

There are inconsistent results reported in the scientific literature about a possible increase in multiple pregnancies near to incinerators. An initial study (Lloyd *et al.* 1988) investigated twinning rates around two chemical waste incinerators in Scotland, UK, between 1976 and 1983, following anecdotal reports of increased twinning in cattle in the area. For the years 1980 to 1983, the study found the highest values of twinning were apparent in the areas considered to be most vulnerable to incinerator releases. Values for 1980 were statistically significant. Comparatively high values were also observed in areas designated as less vulnerable in the period for 1976-9. Analysis of results indicated that the late 1970s and early 1980s was the period when spatial clustering of excess twinning became evident.

During the late 1970s to the early 80's, the rate of twinning in cattle in the area was also seen to increase dramatically. Lloyd *et al.* (1988) proposed that the increased rate of twinning in cattle and in humans in the area was consistent with the hypothesis that environmental air pollution may have affected obstetric parameters of the local populations of people and animals. However, not all confounding factors could be ruled out and based on the results, the authors commented that it would be premature to attribute a causal link between the pollution from incinerators and twinning.

In addition to increased twinning in cattle, farmers in the area had reported other effects in cattle including increased abnormalities and stillbirths and unexpected deaths. A subsequent study, (Lenihan Inquiry Report) however did not find a link between the incinerator releases and problems in the cattle (cited in Petts 1992, Gatrell and Lovett 1989).

Van Larebeke (2000) noted that data from a Belgian study on incineration and health effects (discussed above, section 4.2.5), showed that there was a statistically significant increased (2.6-fold) probability of having multiple pregnancies in a population living in the neighbourhood of two MSW incinerators. In another study however, data of twin deliveries in Sweden between 1973 and 1990 did not show evidence of clusters of twin births in the vicinity of incinerators (Rydhstroem 1998). The study used a method that could compare the number of twin deliveries both before and after the commissioning of an incinerator.

3.2.6 Hormonal Effects

Thyroid hormones in the blood of children living in industrial/agricultural municipalities close to the Beibesheim incinerator in Germany were compared to those of children living in an industrial/agricultural area without an incinerator and in a second comparison area (Osius & Karmaus, 1998). The incinerator was licenced to burn PCB contaminated materials (Osius *et al.* 1999). The initial 1998 study determined thyroid hormones (free thyroxine and free triiodothyronine) in blood samples from 671 children, aged 7-10 years. Blood serum levels of free thyroxine (FT4) and, to a lesser extent, free triiodothyronine (FT4) were statistically significantly lower in children living in the area in which the incinerator was operating. In this group, it was also found that there was a higher prevalence of FT3 values below clinical references. Mean levels of thyrotropin stimulating hormone (TSH), however, were only marginally different. The authors concluded that their results, considered with those of Holdke *et al.* (1998), (see: Section 3.1.1), suggested that children exposed to toxic waste incineration in the studied area had lower free blood thyroid hormone levels.

In the later 1999 study, the authors attempted to correlate blood contaminant levels with the highly complex thyroid hormone system, which regulates the development of brain function and cell growth. It was found that increased concentrations of the mon-ortho congener PCB 118 in the blood were statistically significantly associated with increased levels of TSH. Elevated levels of PCB congeners 138, 153, 180, 183 and 187 were associated with reduced blood FT3 levels. No associations were found for PCB congeners and FT4 although elevated blood cadmium concentrations were associated with raised TSH levels and diminished FT4 levels. The authors concluded that the study supported the hypothesis that cadmium and PCBs could have a detrimental effect upon levels of thyroid hormones. Given the importance of the thyroid hormone system in the growth and development of children the authors suggested that future studies should be made of impacts of these contaminants upon thyroid hormones in different age groups and to consider neurological development as a component of these studies.

3.3 Risk Assessment

The present regulatory system aims to set quantities or rates at which chemicals can be legally released into the environment. In Europe, limits are generally based on the process of hazard assessment but in recent years the process of risk assessment has been increasingly implemented.

A risk assessment on health effects attempts to estimate exposure to a particular chemical from the pollutant releases in question and finally calculates the probability of health effects from the estimated exposure. Many risk assessments have been reported on health effects expected to arise from exposure to incinerator releases, particularly cancer risk. Nearly all such risk assessments from the 1980s through to the 1990s have concluded that contaminants from incinerators do not pose a significant health risk to populations living within their vicinity. This is in direct contrast to human epidemiological studies, some of which have found evidence of health impacts.

For example, a review of risk assessment data for hazardous waste incinerators in the 1980s by Oppelt (1990) points to a conclusion that stack emissions from burning hazardous waste pose little risk to human health. Data on which many of these assessment were based has however been criticised by the US EPA Science Advisory Board as being insufficient. Dempsey and Oppelt (1993) discuss risk assessments conducted for cement kilns burning hazardous waste. These concluded that no adverse health effects would be expected due to emissions. A US health risk assessment on MSW incinerator air emissions estimated that carcinogenic and non-carcinogenic risks from exposure via inhalation were within acceptable limits (Roffman and

Roffman 1991). Similarly, a study in Germany estimated that cancer risk caused by inhalation of selected heavy metals and dioxins emitted from modern municipal waste incinerators would not endanger health. (Eikman 1994). A study on a MSW incinerator in Mokdong, Seoul, Korea, also reported that cancer risk from inhalation was less than the acceptable risk value of one cancer case in a million (Lee *et al.* 1997).

One of the criticisms of many health risk assessments on incinerators is that they only account for exposure via inhalation and do not consider other routes of potential exposure such as ingestion of soil and vegetation and absorption through the skin (Webster and Connett 1990). This criticism applies to many of the above mentioned studies. Given that ingestion of food is the dominant route of exposure for incinerators sited in or near to agricultural areas this brings all the above risk assessments into substantial question (see e.g. Meneses *et al.* 1999, Webster and Connett 1990).

A recent risk assessment of a MSW incinerator in Montcada, Catalonia, Spain, did take into account all of the known potential exposure routes for dioxins. The risk assessment estimated exposure of local residents to incinerator air emissions of dioxins via inhalation of air and particulate matter, and via soil and vegetable intake from the area and via absorption of soil through the skin (Meneses *et al.* 1999). It compared the intake of dioxins through these routes with intake through a normal diet. Results showed that incinerator air emissions accounted for less than 6% of the total dioxin intake for the population, while diet accounted for greater than 94%. The study concluded that according to the WHO standard for tolerable daily intake (TDI) of dioxins, (i.e. the daily intake of dioxins per person proposed as safe based on current knowledge), intake of dioxins from the incinerator would not imply health risks for the general population of the area. However, the study failed to mention that the incinerator emissions would nevertheless add to levels of dioxins already stored in the tissues of the nearby population as well as those already present in the food supply.

Interestingly, a recent risk assessment was published which did indicate an increased health risk from exposure to dioxins for some child members of a population living near to incinerators (Nouwen *et al.* 1999). The risk assessment was conducted for the population living within the vicinity of the two incinerators in Neerland, Wilrijk, Belgium. Epidemiological studies in this region indicated that an increased probability of congenital malformations at birth and some impacts on respiratory health of children as discussed in section 4.2.5 above (van Larebeke 2000). The risk assessment considered 3 possible exposure scenarios. These were, firstly, a worst case scenario in which individuals lived exclusively from foods (meat, milk and vegetables) produced in the vicinity of the incinerator. Secondly, a scenario which assumed people consumed a mixture of commercial produce and produce from the area (25% crops and 50% meat) was considered. Thirdly, a scenario was proposed in which individuals consumed only commercial produce containing background concentrations of dioxins. This latter scenario was assumed to be the exposure situation for the majority of the residents at Neerland. Exposures were estimated based on dietary exposure, inhalation and dermal exposure. The present tolerable daily intake set by WHO for dioxins is 1-4 pg TEQ/kg bw/day. The risk assessment estimated that children in the first, high exposure scenario, would have exceeded this limit in 1980 by a factor in excess of 4 (16.62 pg TEQ/kg bw/day). Children with an estimated medium exposure in the second scenario, also exceeded the WHO TDI in 1997 by a 2-fold margin (8.17 pg TEQ/kg bw /day). The study considered that this would be the situation for a relatively few families.

4. ENVIRONMENTAL CONTAMINATION

4.1 Deliberate and Fugitive Releases from Incinerators.

Incinerator wastes in the form of stack gas, fly ash, bottom ash/slag, scrubber water, scrubber water filter cake, etc. are deliberately dispersed or otherwise released to the environment, carrying with them the diversity of pollutants formed or redistributed during the incineration process. Some of the incinerator wastes, as well as the wastes actually burned, are also released unintentionally as fugitive emissions.

One important difference between the two types of releases, deliberate and fugitive emissions, is the extent to which they are subject to regulatory oversight and control. Thus far, stack gas would appear to be the most highly regulated of the deliberate releases from incinerators. Characterisation and oversight of the other deliberately released incinerator wastes is sparse at best.

Fugitive emissions are vapours or particles that escape during waste tipping, waste feeding, incineration, and ash handling. For example, fugitive dusts can be released from bottom-ash pits and fly-ash hoppers as well as during the process of transferring these ashes to transport vehicles and during the transfer from transport vehicles to the final repositories, such as a landfill. These dusts, particularly fly ash dusts from particulate air pollution control devices, are enriched in toxic metals and contain condensed organic matter (NRC 2000).

At hazardous waste incineration facilities, fugitive emissions are, in the case of liquid wastes, released as vapours from liquid waste tank vents, pump seals, and valves. For solid wastes, fugitive emissions escape as dust from solid-materials handling and during the handling and transport of fly ash captured by air pollution control devices. Also, the high-temperature seals on rotary-kiln incinerators are a potential source of vapour and dust emissions generated by such incineration facilities (NRC 2000).

Fugitive emissions can be minimised by designing buildings to be under negative pressure so that air is drawn from the areas where both the incinerator ashes and the wastes to be burned are handled and stored. The National Resource Council noted,

"*Although some facilities have partially closed ash-removal systems, few have completely enclosed ash-handling systems throughout the plant.*"

Fugitive emissions that are released near ground level may well have a greater impact on the nearby environment than those released into the air from the incinerator stack. The pattern of dispersal of both fugitive emissions and stack releases depends on a potentially infinite number of variables, for example, type of terrain, presence of nearby structures or trees, wind direction and velocity, weather conditions and relative humidity and the interactions between them.

4.2 Studies on Environmental Contamination

Pollutants that are emitted into the atmosphere from an incinerator stack, as well as fugitive emissions, may be deposited on the ground near to the incinerator and so contaminate the local environment. Some pollutants, including PM10 particulate matter and volatile and semivolatile organic compounds, such as dioxins and PCBs, may also be transported great distances on air currents. For example, Lorber *et al.* (1998) estimated that only around 2% of the dioxin emissions to air are deposited in soil near to an incinerator while the remainder is much more widely dispersed.

Most research on environmental contamination in the vicinity of incinerators has focused on dioxins and heavy metals, ignoring most other pollutants. Studies show that soil and vegetation close to incinerators may become contaminated with incinerator releases of dioxins and heavy metals to levels above normal background concentrations. As a consequence, there is a possibility of agricultural produce, such as crops, becoming contaminated. Livestock may also take in pollutants, largely through ingestion of contaminated vegetation and soil. In some instances this has led to cow's milk being banned from sale due to unacceptably high levels of dioxins, and recommendations to avoid the consumption of eggs and poultry.

This section discusses studies on levels of dioxins and heavy metals found in soil and vegetation in the vicinity of incinerators, both in the past, and more recently. Levels found in cow's milk are also discussed. Considering the potential for contamination of agricultural produce close to incinerators of all types, the research in this area is very limited.

4.2.1 Soil and Vegetation

Research has shown that soil and vegetation can be used as suitable media for monitoring contamination from atmospheric deposition of dioxins and heavy metals (see Schuhmacher *et al.* 1999a, Schuhmacher *et al.* (1997a), Gutenman *et al.* (1992).

Levels of dioxins in soils have been widely used to describe long-term exposure to these chemicals. On the other hand, vegetation is a more representative index of short-term exposure to dioxins (Schuhmacher *et al.* 1999b).

With regard to vegetation, dioxins and heavy metals may simply be deposited on the surface of leaves or be present in soil particles on the plants. In addition, metals may enter the leaves through the small pores on the leaf surfaces (stomata) and be taken up by the roots in woody plants (see Bache *et al.* 1992). Dioxins are, however, apparently not taken up by the root system of plants (Hulster and Marschner 1992).

Dioxins

There are many sources of dioxins in urban areas besides incinerators and consequently, in urban/industrial areas, it is difficult to clarify whether dioxin contamination is coming from an incinerator as opposed to other sources. Nevertheless, studies have shown that high levels of dioxins are present in soils near to some incinerators. In many instances, they have also shown that the level of dioxin found in soil and vegetation is dependent upon the distance from the incinerator, a phenomenon which implicates incinerators as a primary source of the contamination.

For instance, a study which took soil samples from the surroundings of a clinical waste incinerator in Spain, found the highest levels were located at distances nearer to the incinerator compared to further away (Jimenez *et al.* 1996). Levels in soils close to the incinerator were 2.1 to 7.5 times higher than usual background levels of dioxins in soils. In another study, extremely high dioxin levels (e.g. 252 and 211 ppt TEQ) were found in soils on the leeward side of a Japanese MSW incinerator (Ohta *et al.* 1997). These levels are exceedingly high compared to background levels found in soils of industrialised countries (e.g. 3.6 ppt TEQ for rural soils and 11.9 ppt TEQ for urban soils of North America, and similar levels for Europe), (USEPA 2000). This incinerator has generated considerable controversy due to the high number of cancer deaths recorded nearby. The study showed that the high soil levels correlated with the area of high cancer incidence.

In 1993, exceptionally high levels of dioxins and PCBs were reported in soils near to the Shanks hazardous waste incinerator (formerly the Rechem incinerator) in Wales, UK (see ENDS 2000b). The highest level of dioxins was 1740 ng I-TEQ/kg (Foxall and Lovett 1994). The study indicated that releases other than those from the incinerator stack could have been substantially responsible for the high levels, including fugitive emissions during ash disposal operations from waste storage areas and the transformer handling facility (Foxall and Lovett 1994). The plant has since been upgraded and recent data suggest that levels of dioxins in soils have now fallen to around two-thirds of the 1993 values. The decline in PCBs is however less marked. Moreover, average air emissions of PCBs (2 ng/m^3) from the plant are well above levels in urban areas of Britain which rarely exceed 1ng/m^3 and are usually well below 0.5 ng/m^3. Even so, despite being previously challenged by the Environment Agency, the company now has authorisation to import another 200 transformer carcasses, a known source of PCBs, from Italy (ENDS 2000b).

A 1998 study on an old MSW incinerator in Montcada, Barcelona, reported dioxin levels in soils ranging from 0.06 to 127.0 ng I-TEQ/kg (ppt) with a mean concentration of 9.95 ng I-TEQ/kg (ppt), (Granero *et al.* 1999). The study found that levels had increased at all sites that were monitored between 1996 and 1997 and again between 1997 and 1998. However, the increases were not statistically significant. The authors noted that although the conditions of incineration remained constant during 1996 to 1998, it is possible that the potential accumulation of dioxins in soils that may be expected from incineration could be counteracted by a decrease in emissions from other sources in the area.

Some studies have shown that incineration is not always associated with high levels of dioxins in local soils. For instance, a study in Spain showed that levels in the vicinity of an old incinerator in Catalonia in 1997 (range 0.11 to 3.88 mean 1.17 ng I-TEQ/kg (ppt) were not unduly high and indeed were consistent with levels found at MSW incinerator sites in other studies in the US and The Netherlands (Schuhmacher *et al.* 1999a). The levels found in 1997 had, however, increased slightly (8.3%), but not significantly, from levels previously determined in 1996.

Studies on vegetation near a MSW incinerator in Catalonia, Spain in 1996/7 showed that the incinerator contributed to dioxins in vegetation because higher levels were apparent closest to the incinerator with lower levels recorded further away (Domingo *et al.* 1998). However, between 1996 and 1997, unlike levels in soils at the incinerator which remained relatively constant, (see above: Schuhmacher *et al.* 1999a), in some areas there was a reduction in the levels of dioxins in vegetation. Since vegetation largely reflects short-term changes in dioxin exposure, whereas soil reflects longer-term exposure, the study noted that the reduction in dioxin levels in vegetation might be due to better pollution control at the incinerator. It could also, however, be reflective of a reduction of dioxin emissions from other sources in the area in general. Similarly, another study in Spain at an old MSW incinerator in Montcarda, Barcelona, found a decrease in levels of dioxin between 1997 and 1998. The study proposed that the reduction in dioxin levels was probably due to general abatement actions to reduce dioxin air emissions (Schuhmacher *et al.* 1999b).

Heavy Metals

Heavy metals released into the environment from incinerators can contaminate soils and build up

(bioaccumulate) in plants and animals. In this way they eventually make their way to humans via the food chain or through contamination of drinking water. In addition, for people living in the vicinity of incinerators, and especially for children, exposure to heavy metals may also occur due to the consumption of dirt or dust originating from contaminated soil. Other routes of heavy metal intake include inhalation and absorption via the skin (Schuhmacher *et al.* 1997b).

Data on levels of heavy metals in soils near to incinerators are very limited. A study on soils near to an industrial incinerator in Italy found lead pollution in soils was increased by some 600% (Zanini and Bonifacio 1991). A more recent investigation into levels of cadmium and lead in soils surrounding the Baldovie MSW incinerator in Scotland determined that the incinerator was responsible for the long-term distribution of the metals in soils within a 5 km vicinity of the incinerator (Collett *et al.* 1998). It was found that long-term concentrations of cadmium and lead in emissions to air from the incinerator were related to levels found in local soils. Although the levels of cadmium and lead were linked to incinerator air emissions, the study also reported that levels of cadmium and lead were within their normal background ranges in soils. A study on a sewage sludge incinerator near to Birmingham, UK, found evidence of lead and cadmium contamination in surface dusts close to the incinerator (Feng and Barratt 1999).

A recent study in Spain on the old MSW incinerator in Montcada, Barcelona did not find levels of heavy metals that were considered to be high (Schuhmacher *et al.* 1997b). For cadmium and lead, levels were similar to concentrations reported for uncontaminated soils.

There are only a few studies in the scientific literature on levels of heavy metals in vegetation in the vicinity of incinerators. Bache *et al.* (1991) reported contamination of vegetation surrounding a MSW incinerator (without air emission controls) in the US by several heavy metals. Another study on a MSW incinerator in the US, this time fitted with pollution control equipment, found that levels of cadmium and lead in tree foliage were linked to distance from the incinerator (Bache *et al.* 1992). The study concluded that even a MSW incinerator with pollution control equipment (electrostatic precipitator) could result in the significant deposition of metals such as cadmium and lead in surrounding areas. A study on a MSW incinerator in New Jersey reported that levels of mercury in vegetation (moss placed at specific sites around the incinerator) was related to distance from the incinerator (Carpi and Weinstein 1994). The highest concentrations of mercury were located closest to the incinerator.

One study has been documented in the scientific literature, which did not find a link between incineration and heavy metals in surrounding vegetation. The study reported that contamination of vegetation with cadmium and lead was not apparently related to distance from a MSW incinerator (Gutenman *et al.* 1992).

4.2.2 Cow's Milk

Cattle that graze in areas subject to air deposition of pollutants, such as dioxins, can ingest the pollutants that have been deposited on vegetation and soils. Dioxins can subsequently be passed to their milk, and hence, ultimately to humans. This is because elimination via milk is a major route of excretion of dioxins in cow's (Baldassarri *et al.* 1994). Research conducted in several countries during the 1990's has demonstrated elevated levels of dioxins in cow's milk from farms located near to incinerators.

A decade ago, a study in the Netherlands reported high concentrations of dioxins in cow's milk (up to 13.5 pg I-TEQ/g fat [ppt]). This led to a decision by the Dutch government to instigate an upper limit for dioxin levels in milk and milk products of 6 pg I-TEQ/g (fat), (Liem *et al.* 1990). Some other European countries including Germany, Holland and Austria (Ramos *et al.* 1997) later also adopted this limit-value. A study in Austria reported high dioxin levels in cow's milk obtained from farms sited close to incinerators (up to 8.6 pg I-TEQ/g fat), (MAFF 1997a). In the UK, exceptionally high levels of dioxins (up to 1.9 pg TEQ/g whole milk, equivalent to 48 pg TEQ/g fat) were reported in milk from farms located near to a chemical waste incinerator at the Coalite chemicals plant in Derbyshire (MAFF 1992, EA 1997, Sandells *et al.* 1997). The incinerator was subsequently closed down in November 1991.

More recent studies have also found evidence of increased levels of dioxins in cow's milk from farms situated near to incinerators. For instance, a UK study on milk from farms located within the vicinity of potential dioxin sources in the UK, found that farms near to two out of eight MSW incinerator sites investigated had milk with levels of dioxins which exceeded the Dutch 6 pg I-TEQ/g fat limit in 1993-5 (MAFF 1997b). In 1995, milk from a farm sited near a MSW incinerator in Bristol had a level of 6.1 pg TEQ/g fat whilst milk from farms near to an incinerator in West Yorkshire had levels ranging from 3.1 to 11 pg I-TEQ/g fat. A retest at farms in the latter area in 1996 showed that high levels remained in milk (1.9-8.6 pg I-TEQ/g fat). The incinerator was closed later in the year for failing to meet newly imposed pollution control standards.

A study in Switzerland of cow's milk from farms located in both rural and more industrial areas reported that local influence of incinerators on dioxin levels in milk was clearly

detectable (Schmid and Schlatter 1992). Similarly, a more recent study in Spain found that milk from rural areas had levels of dioxins (1.3-2.47 pg I-TEQ/g fat) that were lower than levels in milk from a farm located in the vicinity of potential dioxin sources. Of the dioxin sources, it was determined that a waste incinerator had the greatest effect such that the highest levels (3.32 pg I-TEQ/g fat) in milk were obtained for a farm situated close by (Ramos *et al.* 1997).

5. INCINERATOR RELEASES

All waste incinerators are also waste generators - incineration of waste results in output of waste products. This is because physical matter cannot actually be destroyed, but can only be transformed into new forms. Thus when things are burned, they do not disappear as is the common perception, but merely change their form. Waste products resulting from incineration take the form of stack gas emissions to the atmosphere, bottom ashes (slag) and fly ashes (caught in filters in the incinerator stack) which ultimately are disposed of to landfill sites. Where water is used for cleaning processes in an incinerator, there are also releases of waste products to water.

It is a popular misconception that the weight and volume of the original raw waste are reduced during incineration. Although it is often stated that the solid residues (ashes) remaining equate to about one third of the initial weight of the raw waste (Pluss and Ferrell 1991), and volume reduction of about 90% is achieved (Williams 1990) neither of these statistics stand up to scrutiny. If all the waste outputs from an incinerator are summed, then the output will exceed the original waste input. The gases present in the flue stack result from the combination of carbon-based materials with oxygen and are usually ignored in calculating the mass of residues, but the combination with oxygen to form CO2 increases actual weight. Residues from wet gas cleaning systems can generate appreciable volumes of contaminated water and solids. In the case of the statistic concerning volume reduction, this is usually generated by reference to the volume of uncompacted wastes. Landfilled MSW, however, is generally compacted to increase stability and prevent water infiltration as well as reduce the volume of the wastes. Comparing unburned waste and incinerator ash, the actual volume reduction achievable is closer to 45% (DoE 1995).

Numerous chemicals are released into the wastes generated by incineration, including hazardous chemicals. For instance, MSW incinerators are typically fed a mixed waste stream and the combustion of such waste leads to hazardous substances originally present within the waste being mobilised into releases from the incineration plant. While some chemicals remain in their original form, others are changed into new chemical species. For example, heavy metals are not destroyed by incineration but are simply concentrated in the remaining wastes. They can remain in their original form during incineration or may react to form new compounds such as metal oxides, chlorides or fluorides (Dempsey and Oppelt 1993).

The exact nature of the substances released during incineration depends on the composition of the waste that is incinerated. For instance, incineration of chlorinated organic compounds will cause the formation of hydrogen chloride (HCl) and this in turn can contribute to the formation of dioxins. Technical standards that are applied both to the incineration process and to pollution control equipment will also influence the final products of incineration (EEA 2000). However, whatever control technology is applied, all types of incineration result in releases of toxic substances in ashes and in the form of gases/particulate matter to air. These substances include heavy metals, numerous organic compounds, such as dioxins, and gases, such as nitrogen oxides, sulphur oxides, hydrogen chloride, hydrogen fluoride, together with carbon dioxide. According to the NRC (2000):

" … *the products of primary concern, owing to their potential effects on human health and the environment, are compounds that contain sulfur, nitrogen, halogens (such as chlorine), and toxic metals. Specific compounds of concern include CO, NO_x, SO_x, HCl, cadmium, lead, mercury, chromium, arsenic, beryllium, dioxins and furans, PCBs, and polycyclic aromatic hydrocarbons.* …"

In many countries over the past few years, new regulatory air emission standards have forced the closure or updating of many old incinerators or the building of new ones. Upgraded plants (together with new ones) may be fitted with modern, improved air pollution control technology. For example, out of the 780 incinerators in operation in the UK in the early 1990s (30 for municipal waste, 700 for clinical waste, 40 attached to chemical companies, 6 for sewage sludge and 4 for hazardous waste), only 110 remained after the tightening of standards (Murray 1999). Presently there are 12 MSW operating incinerators in the UK. The closure or updating of old incinerators is considered to have led to a substantial reduction of emissions of toxic substances to air.

One study in the Netherlands has also estimated that dioxin emissions to air have been significantly reduced (Born 1996). Murray (1999) states that the most sophisticated German technology developed during the early 1990's has cut atmospheric emissions broadly by a factor of ten. Although this is a significant improvement, the problem of toxic waste products from incineration has not disappeared. In fact, the problem has shifted so that more of the dioxins and other toxic substances generated now appear in the ashes, thereby creating new disposal and pollution problems. The European Environment Agency (EEA 2000) has warned that even if total air emissions from incineration are reduced in the future as standards improve "this might be offset with increased incineration capacity". In this regard, it is of great concern that an increase in the use of incineration is being proposed in some European countries. In the UK for instance, following the closure of numerous old incinerators, up to 177 possible new ones have been proposed by the government (ENDS 1999).

On the regulatory front, among the various incinerator outputs, stack gas has received the greatest share of attention and is the most highly regulated since the gas and its toxic components are dispersed directly into the open air. However, the other incinerator wastes also contain toxic pollutants and, consequently, pose threats to public health that may be less obvious and/or immediate but are no less real.

The Commission of the European Communities (CEC) has been drafting a proposal for a new waste incineration directive since 1998 (EC 1998, EC 1999). The proposed new directive will establish controls on the incineration of most wastes that are not covered by the previous 1994 directive. The new directive will set limits for releases of some hazardous substances in stack gases and water. The directive is expected to be adopted by the end of 2000 or early 2001. All new MSW incinerators built after the directive comes into force must satisfy the limits in the directive within 2 years, whilst existing incinerators have a period of 5 years to satisfy the criteria. In addition to the EU regulations, various national guidelines for incinerators are also presently in place and these will have to comply with the directive within two years of it coming into force (EC 1999).

5.1 Releases to Air

This section presents data on substances known to emitted in stack gases from incinerators. Most research on air emissions has focused on dioxins and upon the behaviour of a few toxic heavy metals. Data from research upon other emitted chemicals are sparse. In addition, a very large number of the chemicals emitted from incinerators remain unidentified.

Emissions from incinerator stacks to air are discussed below under the following categories: organic compounds; heavy metals; gases and particulates. The EC have proposed limits for air emissions from incinerators in their new directive for only a few of the compounds falling under these categories. These proposed limits are given in the table below.

Table 5.1 EC Air Emission Limit Values

Substance	**Proposed EC Limit (mg/Nm^3)**
Dioxins	0.1 ng TEQ/Nm^3
Mercury	0.05[b]
Cadmium + Thallium	Total 0.05[b]
Sb, As, Pb, Cr, Co, Cu, Mn, Ni, V	Total 0.5[b]
Carbon Monoxide	50[c]
SO^2	50[c]
Nox	200[c]
HCl	10[c]
HF	10[c]
Particles	10

[a] Average values measured over a sample period of a minimum of 6 hours and a maximum of 8 hours.
[b] All average values over the sample period of a minimum of 30 minutes and a maximum of 8 hours.
[c] Daily average value

5.1.1 Organic Compounds

Dioxins

Polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are a group of chemicals often referred to simply as dioxins. There are more than 200 individual congeners (members) of the PCDD/Fs group. The most widely known and most toxic congener is 2,3,7,8-TCDD. It has been described as the most toxic chemical known to mankind and is a recognised as a human carcinogen. Dioxins are persistent in the environment, toxic and bioaccumulative (build up in the tissues of living organisms). A more detailed description of the toxic impacts of dioxins on health is given in appendix A.

The toxicity of individual dioxins and furans varies by several orders of magnitude. Because analytical data may report 17 different congeners as well as totals for homologue groups (i.e. all congeners containing the same number of chlorine atoms), it is often necessary to summarise data so that individual samples can be directly compared. This is generally done by expressing the amount of dioxins present as toxic equivalents (TEQs) relative to 2,3,7,8-TCDD. The most common TEQ system used is the international toxic equivalents system (I-TEQ). The TEQ system works by assigning TCDD, the most toxic congener, a toxic equivalence factor (TEF) value of 1. The toxicity of all other congeners is expressed relative to this, such that they are assigned a TEF value between 0 and 1. The I-TEQ of a sample containing a mixture of dioxins is obtained by multiplying the concentration of each congener by its TEF and summing the results.

One important consideration in relation to dioxin air emissions from incinerators is that regulations consider only the chlorinated varieties. It has been known for some time that incinerators generate and emit brominated and mixed chloro-bromo substituted dioxins in appreciable quantities (see: Schwind *et al.* 1988). These are regarded as of an equal toxicological significance relative to the chlorinated dioxins, producing a similar array of biological impacts at similar molar concentrations (Weber and Greim 1997). Despite these compounds being highly persistent when associated with fly ash particles, little attention has been directed at evaluation of their significance to human health and there are currently no obligations on the part of incinerator operators to monitor and control these chemicals.

Formation of Dioxins in Incinerators

Dioxins are produced as unintentional by-products of many manufacturing and combustion processes, especially processes that use, produce or dispose of chlorine or chlorine derived chemicals. All types of incinerators produce them. Research has shown that while dioxins can be destroyed in the combustion zone of incinerators, they can be regenerated in the post-combustion zone by processes that are dependent on the temperature profile (Blumenstock *et al.* 2000, Huang and Buekens 1995, Fangmark *et al.* 1994). The predominant formation pathway of dioxins has been reported to be de novo synthesis (Johnke and Stelzner 1992), and they are also formed from precursors that are either constituents of the waste or are also formed by chemical recombination of materials in the waste. The chlorobenzenes and chlorophenols are two such groups (Huang and Buekens 1995). PVC, a common constituent in municipal waste, has also been identified as a dioxin precursor (USEPA 1997).

Prior to incineration, raw waste is itself known to contain dioxins. However, it has been demonstrated that the process of waste incineration creates dioxins. For instance, past and present, calculations (mass balances) both show that the total amount of dioxins coming out of an incinerator in the various waste products is greater than the amount going into the incinerator as raw waste (Williams 1990; Hansen 2000). This appears to still be the case for modern and updated incinerators operating in the late 1990s, although very little in the way of scientific data is available from the scientific literature with the exception of a recent Danish study (Hansen 2000).

In another example from Spain, a mass balance estimate based on measurements from eight operating municipal waste incinerators showed that more dioxins are emitted from the incinerators than were present in the raw waste (Fabrellas *et al.* 1999). Estimates showed the level of dioxins (PCDD/Fs) input in raw waste to the incinerator amounted to 79.8 g I-TEQ/year. This compared to the total estimated output of flue gases (1-1.2 g I-TEQ/year), fly ashes (46.6-111.6 g I-TEQ/year), and bottom ashes (2-19 g I-TEQ/year). A dioxin mass balance conducted on another Spanish municipal waste incinerator was ambiguous. One test showed a greater dioxin output than input whereas another test showed a greater dioxin input than output (Abad *et al.* 2000). This is not particularly surprising because emissions of dioxins and other substances from individual incinerators are highly variable depending on waste input and combustion conditions. In addition, the precision of such estimates is often not high, encompassing a wide range of values.

Dioxin Inventories and Incineration

During the 1980s and early to mid-1990s MSW incineration, in particular, was identified as a major source of dioxins emitted to atmosphere. For example, the Dutch government organisation RIVM estimated that incineration was responsible for about 79% of all dioxins emitted to air in the Netherlands for the year 1991. In the UK, MSW incinerators were estimated as responsible for about 53-82% of all dioxins emitted to air in 1995. In the US such facilities accounted for about 37% of total annual air emissions (see Pastorelli *et al.* 1999). A summary of data from 15 countries, described as a "global" inventory, showed that incineration accounted for about 50% of dioxin emissions to air in 1995 (Fiedler 1999). MSW incineration has been identified as being responsible for the greatest proportion of dioxin air emissions compared to other types of incineration (eg. Alcock *et al.* 1998), although from "global" inventory data for 15 countries, Fiedler (1999) noted that all sectors of incineration in 1995 were major emitters in many countries. This included MSW incinerators, hazardous waste incinerators, sewage sludge incinerators, waste wood incineration and crematoria. Table 1.2 shows the estimated dioxin air emissions for different types of incinerators for 1997 in the UK.

Even recently, incinerators have been estimated to account for a high proportion of atmospheric dioxins. For example, (Hansen 2000), has conducted a flow analysis for dioxins in Denmark for 1998-1999. Notwithstanding improvements in technology, municipal solid waste incineration was identified as the single largest source of dioxin releases to atmosphere, estimated at between 11-42g I-TEQ per year. It is estimated that a further 35-275g I-TEQ of dioxins contained in incinerator residues is disposed of to landfill each year. This report also draws attention to the potential importance of the brominated and mixed halogenated dioxins (Section 5.1.1) and estimates that between 2 and 60g of brominated dioxins are emitted to atmosphere from Danish MSW incinerators per year.

Table 5.2. PCDD/F air emission estimates for the UK, (numbers in bold type represent estimates calculated from measured air emissions, other number are estimates)

PROCESS	1997 Range/Low (g TEQ/annum)	1997 Range/High (g TEQ/annum)
MSW incineration	**122**	**199**
Chemical waste incineration (10 sites)	**0.02**	**8.7**
Medical Waste Incineration (5 sites)	**0.99**	**18.3**
Sewage Sludge Incineration (5 sites)	**0.001**	**0.37**
Cement Manufacture (5 sites)	**0.29**	**10.4**
Crematoria	1	35
Domestic Wood Combustion (clean)	2	18
Domestic Wood Combustion (treated)	1	5

Source: Alcock *et al.* 1998.
Footnote: The estimate for total dioxin air emissions from all sources is Range/Low 219 and Range/High 663 g TEQ/annum.

A 1997 publication cited by the commission of the European Communities (EC 1998) noted that incineration of non-hazardous waste may contribute up to 40% of all dioxin air emissions in Europe. Nevertheless, in some European countries, it has been estimated that the contribution of MSW incineration to national inventories has fallen significantly during the mid- to late 1990s. This is due to closure of old incinerators, which emitted high levels of dioxins to air and the fitting of pollution abatement equipment to both remaining plant and new installations. Estimates suggest that such improvements will have resulted in the significant reduction of dioxin emissions from incinerators to air. For instance, strong downward trends of air emissions have been identified in countries with modern technology or rigid legislation (Fiedler 1999). Considering atmospheric emissions alone, in the UK, Her Majesty's Inspectorate of Pollution (HMIP) and the Department of the Environment (DoE) estimated that the contribution to the total annual emission would fall from 53-82% in 1995, to around 4-14% in the future. Similarly, the German UBA estimated a contribution of 33% for the years 1989-1990 falling to 3% for the years 1999-2000. These estimated data remain to be confirmed with empirically derived data.

The need for confirmation is important. It has been acknowledged, for instance, in the above UK HMIP study, that there are large uncertainties in estimations of incinerator air emissions used in dioxin inventories. In the case of the UK study, this is because air emissions have generally been estimated from only very limited measurements and have also used information derived from non-UK studies. A recent UK study which corrected for these sources of uncertainty to some extent (Alcock *et al.* 1998), used a different, more precise, estimation method that included measured emission data from individual incinerators between 1995 and 1997 (see table 1.2). It currently represents the most comprehensive survey of measured UK dioxin air emission data. Importantly, the study also used data from waste incinerators, which were operating under normal everyday conditions during the periods of testing. This is more realistic than measurements being taken under "optimum" conditions that are specifically set up for testing under a "test burn" regimen which is, more often than not, the case. The study found that relative to emission data published by the HMIP for 1995, the levels of dioxin emissions from MSW incinerators between 1995-7 had fallen somewhat. Even so, they still represented a significant part of the national inventory, representing 30 to 56% of the total national air dioxin emissions. Clearly the optimistic projections of the regulatory authorities need confirmation before they can be accepted as a realistic projection of trends or as a metric of the current situation.

On the same note, Webster and Connett (1998) draw attention to uncertainties and problems in the methodology commonly employed to derive national dioxin air emission inventory data. These include several points listed below and include two points specifically mentioned in the UK study above: firstly that few empirically measured data from individual incinerators are normally used in the estimate (see first bullet point), and that data on air emissions used are most often derived from testing of incinerators under "optimum" conditions rather than normal day to day operations.

- **Methodology:** The method normally applied for estimating dioxin inventories, "the emission factor approach" relies on a limited number of specific measurements from particular types of incinerators and extrapolates these to represent all incinerators of a particular type. This is likely to underestimate emissions to all media. It does not take account of the fact that there can be enormous variability in emissions from individual incinerators of the same type. In their study,

Webster and Connett (1998) showed that the "emission factor approach" did indeed underestimate dioxin air emissions from incinerators reported in many previous US inventories over the past decade. Instead of applying the emission factor approach, Webster and Connett (1998) summed dioxin air emissions for measured facilities only – an approach that would presumably underestimate these emissions since unmonitored incinerators were not included in the calculations. Even so, this method still produced a significantly greater value for MSW incinerator dioxin emissions to air than using the emission factor approach. The authors therefore stressed the need for adopting the use of actual measurements from individual facilities for inventories.

- **Lack of Data:** On a global basis, Fiedler (1999) reported that the present number of national dioxin emission inventories is very small. Within countries that have recorded dioxin inventories, there is a general lack of comprehensive data on dioxin air emissions from incinerators. For instance, Webster and Connett (1998) identified a paucity of data in the US with specific regard to emissions from incinerators. Many US MSW incinerators had either been tested only once or had never been tested at all. Although this situation appears to be improving, operators and regulators in the past seemed quite happy to deem a plant's emissions to atmosphere acceptable based on one set of measurements derived from a pre-commissioning test-burn. Even now, the frequency and intensity of stack sampling and analysis for dioxins carried out at most incinerators is unacceptably low.

- **Monitoring:** Research has shown that taking only a limited number of measurements is not likely to accurately reflect dioxin emissions to atmosphere from incinerators over the full spectrum of operational conditions. That dioxin emissions from combustion sources may change considerably over time is well illustrated by a UK study (Alcock *et al.* 1998). The study showed that air emissions indexed by samples collected from a cement kiln stack on the same day were found to vary considerably. The first sample collected measured 4.2ng I-TEQ m^{-3} and the second sample taken 5 hours later was determined as 0.06ng I-TEQ m^{-3}.

  A more accurate estimate of atmospheric dioxin emissions can only be established by continuous monitoring of emissions for extended periods of time. Start-up and shut down periods in the operation of MSW incinerators are particularly prone to result in high dioxin emissions. A study on a Belgian incinerator, using continuous monitoring, was undertaken in an attempt to demonstrate that retro-fitted modern pollution control equipment would prevent excedence of the 0.1 TEQ/Nm^3 regulatory limit at all times. In fact the results revealed that monitoring over a period of 6 hours gave an average emission concentration of 0.25 ng TEQ/Nm^3. However, the average over 2 weeks in the same period gave a result of 8.2 to 12.9 ng TEQ/Nm^3 which was substantially greater and clearly exceeded the regulatory limit (De Fre and Wevers 1998).

  The above study shows, in a convincing manner, that taking measurements from individual incinerators under the normal regulatory protocols (i.e. point measurements), can significantly under-estimate the dioxin emissions to air from incinerators. In this case, point measurement under-estimated the average dioxin emissions by a factor of 30 to 50. The significance of this finding to other incineration facilities is simply not known.

- **Dioxins in ash are not considered:** Most mass balance inventories consider only dioxin emissions to atmosphere (Fiedler 1999); The output of dioxins in ash from incinerators is not included and Webster and Connett (1998) consider that the fate of dioxin captured in ash receives insufficient attention. A recent study on a Spanish incinerator showed that stack gas emissions were only responsible for a minor contribution to the total dioxin emitted compared to amounts present in fly ash (Abad *et al.* 2000). The fact that dioxins formed in incineration have become more concentrated in ashes as air pollution control technologies have evolved, thereby generating other hazards, is further discussed in section 5.3.1.

Considered together, the generally flawed sampling methodology employed in regulating releases from incinerators coupled with failure to consider the dioxin mass balance in an holistic manner suggests that it is highly probable that most, if not all, dioxin inventories greatly underestimate releases from incinerators.

Performance of Updated and New Incinerators

As indicated above, most monitoring of atmospheric dioxin releases carried out at incinerators in Europe and reported in the scientific literature has been derived on the basis of point measurements rather than continuous monitoring. This can lead to an underestimate of air emissions. This situation seems set to continue under the proposed EC legislation which specifies compliance monitoring based only two point measurements per year taken over a period of six to eight hours (EC 1999). This basis for regulation and control, as opposed to continual monitoring is unlikely to accurately describe dioxin emissions to air from these facilities.

In many cases, studies carried out on the basis of point measurements have reported that dioxin emissions to air from some European incinerators fall within the new proposed EC limit of 0.1 ng I-TEQ/m^3. For instance, a series of monthly to two-monthly point measurements taken between 1994 and 1997 from a newly constructed German MSW incinerator were below the specified limit (Gass *et al.* 1998). Two point measurements, taken within a day of each other, subsequent to initial testing of a newly constructed MSW incinerator in Venice were below the 0.1 ng I-TEQ/m^3 limit (Pietro and Giuliana 1999). A study on a German hazardous waste incinerator was performed which, in fact, used continuous long-term monitoring. Results of 11 long-term monitored samples taken between 1998-9 showed that air emissions were well within the 0.1 ng I-TEQ/m^3 limit (Mayer *et al.* 1999).

Not all studies, however, have returned data indicating compliance with the 0.1 ng I-TEQ/m^3 regulatory limit. For example, point measurements taken at 1 to 4 monthly intervals, January 1997 to April 1999, from 8 Spanish MSW incinerators revealed that 2 incinerators failed to comply (Fabrellas *et al.* 1999). Emission values were 0.7 and 1.08 ng I-TEQ/m^3. In Poland, analysis of stack emissions from 18 new or updated medical waste incinerators in 1994-7 found that almost half had emissions below 0.1 ng TEQ/m^3, but others exceeded the limit (Grochowalski 1998). For 5 of the incinerators, the limit was considerably exceeded with concentrations ranging from 9.7 to 32 ng TEQ/m3. As discussed previously, a Belgian incinerator exceeded the EC regulatory limit when emissions were measured by continuous monitoring (De Fre and Wevers 1998). The emissions were 8.2 to 12.9 ng TEQ/Nm^3.

It is important to note that the scientific literature reporting air emission levels from new and old incinerators presently operating in many countries, including less industrialised countries, is extremely limited. One study of dioxin emissions to atmosphere from the ten incinerators reported to be operating in Korea (Shin *et al.* 1998) noted a wide variation between different incinerators. Emitted levels ranged from 0.07 to 27.9 Ng TEQ/Nm^3 of dioxin in the stack gases.

Even fewer data have been published on incinerators burning wastes other than MSW. Nonetheless, in Japan, one study reported point measurements on nine industrial waste incinerators (Yamamura *et al.* 1999). Dioxin emissions to air were below 0.1 Ng I-TEQ/Nm^3 for two of the incinerators but were above this level (0.13 to 4.2 Ng I-TEQ/Nm^3) for six of them. Cement kilns in the US that were operated using coal as fuel were found to emit 0.00133 to 3.0 ng I-TEQ/dscm (Schrieber and Evers 1994). In the US, a further study reported on dioxin air emissions from mobile incinerators. (Meeter *et al.* 1997). The on-site remediation of soils at hazardous waste sites by such incinerators is carried out where sites contain compounds that are regarded as difficult to destroy. Data collected primarily from trial burns of 16 incinerators showed that 10 of the incinerators failed to meet the proposed US Environmental Protection Agency (EPA) standard of 0.2 ng TEQ /dscm. The authors commented that a significant number of mobile incinerators used in these applications could have problems meeting the proposed future EPA limit.

5.1.2 Other Organic Compounds

With a very few exceptions, very little research has been carried out on the other organic chemicals known to be emitted to air from incinerators. Of the compounds, which have been studied, the focus has largely been directed at higher molecular weight compounds rather than the less persistent volatile organic compounds that are known to be emitted (Leach *et al.* 1999). Compounds for which data have been reported include polyaromatic hydrocarbons (PAHs) and several groups of highly toxic chlorinated compounds including the polychlorinated biphenyls (PCBs), the polychlorinated napthalenes (PCNs), the chlorobenzenes and the chlorophenols.

PCBs: This group consists of 209 different individual congeners. Around half this number has been identified in the environment. PCBs are persistent, toxic and bioaccumulative. Accordingly, like the dioxins they have a tendency to build up in the fatty tissues of animals and humans where they can persist almost indefinitely. The more highly chlorinated PCB congeners are the most persistent and account for the majority of those found as environmental pollutants. PCBs have become globally ubiquitous chemicals, and they are even found at highly elevated concentrations in the tissues of animals living in what have traditionally been regarded as pristine environments. Arctic marine mammals, such as whales, seals and polar bears have been found to be contaminated with PCBs and other organochorines, (see Allsopp *et al.* 1999). PCBs are known to exert a wide range of toxic effects on health including reproductive, neurological and immunological effects. They are suspected of causing many deleterious health effects both in wildlife and in humans (see Allsopp *et al.* 1997, Allsopp *et al.* 1999). Some PCB congeners also cause "dioxin-like" effects on health since they are structurally similar chemicals.

PCBs produced as industrial chemicals were mainly used for insulation in electrical equipment. Production of PCBs has almost totally ceased worldwide, although there are reports of it continuing in Russia. At least one third of the PCBs that have been produced are estimated to have entered the environment (Swedish EPA 1999). The other

two thirds remain in old electrical equipment and in waste dumps from where they continue to leach into the environment. Although this is by far the major source of PCB pollution in the environment today, some PCBs are also produced as by-products of incineration and certain chemical processes involving chlorine.

PCBs are known to be formed in incinerators (Blumenstock *et al.* 2000, Wikstrom *et al.* 1998, Sakai *et al.* 1996, Fangmark *et al.* 1994) and are present in stack gases released to the atmosphere (Miyata *et al.* 1994, Wilken *et al.* 1993, Magagni *et al.* 1991). Data on levels of PCBs in stack gases are, however, somewhat sparse in the scientific literature. A study on MSW incinerators in Japan in 1992 found that emissions of the highly toxicologically significant coplanar PCBs varied considerably between different incinerators (Miyata *et al.* 1994). The mean level (1.46 ng TEQ/m^3) was greater than the guideline (0.5 ngTEQ/Nm^3) for newly constructed incinerators in Japan. The study concluded that waste incinerators were a source of PCB contamination in humans, food and environment.

PCNs: are a group of chlorinated compounds that are also persistent, bioaccumulative and toxic. When originally produced they were used in similar applications to PCBs which eventually superseded them. PCNs are known to be produced as unintentional by-products of thermal processes involving chlorine including incineration and metal reclamation (see: Falandysz and Rappe 1997). PCNs have similar properties to dioxins and PCBs and many of them have high toxic potential even at small doses (see: Abad *et al.* 1999, Abad *et al.* 1997).

PCNs have been found to be present in the stack gas of MSW incinerators. The concentration of PCN (mono-to octa-chlorinated) varied from 1.08 to 21.36 ng/Nm^3 in five MSW incinerators in Spain, while levels of dioxins varied from 0.01 to 5 ng ITEQ/Nm^3 (Abad *et al.* 1999). In addition, PCN congeners exhibiting dioxin-like toxicity have been identified in the atmospheric emissions from municipal waste incinerators (Falandysz & Rappe 1997, Takasuga *et al.* 1994).

PCNs from incineration and other combustion sources are present at detectable levels in wildlife and these processes may contribute a significant loading of these highly toxic and persistent chemicals to the environment (Falandysz and Rappe 1997, Falandysz *et al.* 1996) in addition to the environmental burden resulting from historical manufacture.

Chlorinated Benzenes: are formed in incinerators (Blumenstock *et al.* 2000, Wikstrom *et al.* 1998, Fangmark *et al.* 1994) as are the chlorinated phenols (Wikstrom *et al.* 1999). It has been shown that these chemicals are released in stack gases (Wilken *et al.* 1993). The production of hexachlorobenzene (HCB), the fully substituted form of benzene is of particular significance. HCB is persistent, toxic and bioaccumulative. It is toxic to aquatic life, land plants, land animals and humans and has been used extensively as a pesticide and seed dressing. Recent research indicates that HCB can contribute significantly to the dioxin-like toxicity caused by organochlorine chemicals in human milk (van Birgelen 1998). It is listed by the IARC as a Group 2B carcinogen, i.e. it is possibly carcinogenic to humans and also appears to be a tumour promoter. HCB may damage the developing foetus, liver, immune system, thyroid, kidneys and CNS. The liver and nervous system are the most sensitive organs to its effects (ATSDR 1997, Newhook & Meek 1994).

Halogenated Phenols: including 14 chlorinated, 3 brominated and 31 mixed bromo-chloro phenols have been identified in in MSW incinerator flue gas (Heeb *et al.* 1995). These chemicals are of considerable importance since dioxins can be formed by condensation reactions of two halogenated phenol molecules. The concentrations of mixed brominated and chlorinated phenols found in the raw combustion off-gas (4nmol/Nm^3; 1.2ug/Nm^3) and stack gas (1 nmol/Nm^3; 0.5ug/Nm^3) exceeded typical raw gas concentrations of the dioxins (0.2nmol/Nm^3; 0.1ugNm^3) in MSW incineration plant.

Brominated and Mixed Halogenated Dioxins: In addition to chlorinated dioxins and furans numerous other halogenated compounds will be formed during incineration including brominated and mixed chlorinated-brominated dioxins and furans.

Polychlorinated dibenzothiophenes (PCDBTs): are sulphur containing compounds that are structurally very similar to dibenzofurans. The sulphur substitutes for the oxygen atom found in the furan moiety of dibenzofuran structure. Little is known about their toxicology, but due to their structure they are suspected to be toxic. PCDBTs have been detected in the stack gas of waste incinerators (Sinkkonen *et al.* 1991).

PAHs: are a group of compounds which are produced as by-products of incomplete combustion of organic substances. Some are persistent, toxic and bioaccumulative. Some are carcinogenic. PAHs are emitted by incinerators in stack gases (Yasuda and Takahashi 1998, Magagni *et al.* 1991). Waste composition, temperature and excess air during the incineration process determine the quantity of PAHs emitted by a given facility. High emissions to air of PAHs have been shown to occur during start-up of incinerators (see Yasuda and Takahashi *et al.* 1998). Measurements of total PAH incinerator emissions to atmosphere reported in one study were 0.02 to 12 mg/Nm^3 (see: Marty 1993).

VOCs: Few studies have been conducted on the vast array of other chemicals emitted from waste incinerators. However, one study has been undertaken specifically to identify and quantify volatile organic compounds (VOCs) in the stack gas of a MSW incinerator (Jay and Stieglitz 1995). This study identified a total of around 250 different VOC compounds for which concentrations ranged from 0.05 to 100 mg/m^3. The compounds are listed in appendix B. The list includes highly toxic and carcinogenic compounds such as benzene and the substituted phenols, together with other known toxic compounds such as phthalates. Data on the environmental and toxicological significance of many of the VOCs emitted are very limited, but VOCs are known to contribute to ozone formation in the lower atmosphere (see below).

Organic compounds emitted by incinerators are generally monitored on the basis of a group parameter which sums the total amount present in a sample of the flue gas: Total organic Carbon (TOC). In the study reported by Jay and Stieglitz (1995), the 250 compounds identified were found to account for about 42% of the TOC. The remaining 58% were shown to consist of aliphatic hydrocarbons of unknown identity.

Leach *et al.* (1999) have noted that processes which generate large quantities of VOCs are of environmental significance since, mixed with nitrogen oxides and exposed to sunlight, they aid in the formation of photochemical oxidants (ozone and peroxyacyl nitrates), with deleterious impacts upon ambient air quality. The proposed new EC limit for total VOC (expressed as carbon) is 20 mg/ Nm^3.

5.1.3 Heavy Metals

Heavy metals are emitted from all types of incinerators. Many heavy metals are known to be toxic at low concentrations and some are persistent and bioaccumulative. Further information on the toxicity of some heavy metals is given in appendix A. Heavy metals enter the incinerator as components of various materials in the raw waste. The process of incineration leads to their being concentrated by a factor of up to 10 in the waste residues (ashes) as the volume of waste is reduced through combustion (Buchholz and Landsberger 1995). A proportion of these toxic trace metals is emitted in the stack gases of incinerators to atmosphere. The major proportion is generally present in fly ash and bottom ash with the exception of mercury where the greater proportion is vented via the flue stack.

Each metal has its own major source in the raw waste. Mercury is present due to the disposal of batteries, fluorescent light bulbs and paints (Carpi 1997). Cadmium is present in paints, PVC plastics and the pigments used to colour plastics. Lead is present in batteries, plastics and pigments (Valerio *et al.* 1995, Korzan and Heck 1990), and antimony is present in flame-retardants (van Velzen and Langenkamp (1996) used in plastic items.

On a global scale, incineration contributes significantly to atmospheric emissions of many heavy metals, as shown in table 5.1 (EEA 2000). Within the EU, figures for 1990 estimated incineration to be responsible for 8% (16t/yr) of all cadmium emissions and 16% (36t/yr) of mercury emissions. Emissions of chromium amounted to 46 tonnes and over 300 tonnes of lead in addition (EC 1998). A variety of flue gas treatment systems have been devised in order to reduce stack emissions of heavy metals (EEA 2000). Stack gas data for hazardous waste incinerators indicate that the fabric filter removal efficiencies (with the metals retained in the ash arisings) are in the order of 95% for most metals except mercury.

The EEA (2000) note that control of mercury releases constitutes a special problem in incineration. Almost 100% of the elemental mercury present in waste is emitted via the stack gases because it does not adsorb to filter dusts or ashes. Elemental mercury comprises about 20-50% of the total mercury emitted. The remainder is in the form of divalent mercury which may be predominantly mercury chloride (HgCl2). After emission to the atmosphere, divalent mercury, which is water soluble, may be deposited close to the incinerator. On the other hand, elemental mercury may be transported for long distances by atmospheric currents before it is eventually converted to the divalent form. This can then become deposited on the ground (Carpi 1997).

Despite the acknowledged significance of the fate of toxic heavy metals present in the waste-streams, published data on the concentrations of heavy metals in stack emissions appears to be very limited. Nonetheless, according to an emissions inventory in the Netherlands, stack emissions of cadmium and mercury were reduced considerably between 1990 and 1995 from MSW incinerators as a result of modernisation (Born 1996). During this period, the contribution to the total Dutch air emissions of cadmium reduced from 44 to 13% and mercury from 53 to 11%. The reduction of atmospheric emissions (assuming the data are reliable) means that metals retained in the facility by pollution control devices will be retained in fly ash residues.

Table 5.3. Worldwide Atmospheric Emissions of Trace Metals from Waste Incineration

Metal	Emissions (1000 tonnes/year)	Emissions (as a % of total emissions)
Antimony	0.67	19.0
Arsenic	0.31	3.0
Cadmium	0.75	9.0
Chromium	0.84	2.0
Copper	1.58	4.0
Lead	2.37	20.7
Manganese	8.26	21.0
Mercury	1.16	32.0
Nickel	0.35	0.6
Selenium	0.11	11.0
Tin	0.81	15.0
Vanadium	1.15	1.0
Zinc	5.90	4.0

5.1.4 Particulate Matter

Minute particles of matter suspended in the air, often called particulates, are present as a result of both natural and human activities. Those of natural origin are derived from wind blown soil particles, sea salt, dusts from volcanic eruptions, spores from fungi and pollen grains from plants. Those from human activities are the result of combustion processes, such as coal-burning, incineration and vehicle exhaust. As a broad generalisation, natural particulates are generally larger in size (>2.5μm) than the finer particulates formed from combustion processes (<2.5 μm), (QUARG 1996, COMEAP 1995, EPAQS 1995). It is these finer particulates, known as "respirable particles" which are of great concern in relation to human health. Particulate pollution is implicated in the worsening of respiratory illnesses such as asthma, and increasing premature mortality from respiratory and heart diseases. This is because the respirable particulates are small enough to be inhaled into the extremities of the lung airways, whereas larger particles are prevented from reaching the deep airways by the respiratory system's protective mechanisms. In particular, those particulates sized <0.1μm, termed ultrafine particles, are of greatest concern in regard to adverse effects on human health. A more detailed description of particulates and their health impacts is given in appendix A.

Incineration gives rise to atmospheric emissions of particulates (EC 1998). Poorly controlled incineration plants can emit high levels of particulate matter and contribute to local environmental problems. Modern incinerators emit lower levels, but data suggests that the particulates emitted are fine in size and therefore would be contributing to adverse health effects (EC 1998). Indeed, the majority of particles formed from combustion processes, including all types of waste incineration, are ultrafine particles that are less than 0.1 mm in size. Even the most modern MSW incinerators do not have technology that prevents the release of ultrafine particles. Collection efficiencies for respirable particles (less than 2.5 μm) are between 5 and 30 % using current bag filter technology. For particles less than 1 μm in size, which includes all ultrafine particles, most will pass through incinerator filtration systems unabated. Furthermore, there are indications that some of the modern pollution abatement equipment installed in incinerators, particularly ammonia injection, which attempt to reduce oxides of nitrogen, may actually increase the air emissions of the finest, most dangerous particles (Howard 2000).

At present, there is only limited information on the chemical composition of particulates. Emissions to the atmosphere from incinerators include, for example, particles formed of mineral oxides and salts from the mineral constituents in the waste (Oppelt 1990). Heavy metals and organic chemicals such as dioxins, PCBs and PAHs can adhere onto the surface of the particles. Metals may absorb in a number of different forms including metal oxides, soluble salts and metal carbonates. The chemical nature of particulates, for instance, the form of metal, or the type of other potentially toxic chemical adhered to the particle surface, may ultimately influence the effects on health resulting from exposure (QUARG 1996, Seaton 1995, Marty 1993).

Ultrafine particles have been found to be highly chemically reactive, even when they originate from material which itself is not reactive. This is solely due to their minute size. Research has shown that a proportionally higher number of

surface atoms are present as the particles size decreases. This leads to their surface becoming highly charged and therefore chemically reactive. In addition, ultrafine metal particles have been shown to be especially chemically reactive (Jefferson and Tilley 1999).

MSW incinerators typically have a mixed waste input containing heavy metals and halogenated organic compounds. They emit ultrafine metal particulates. Since these particles are especially reactive, it can be argued that MSW incinerators will therefore produce a more toxic ultrafine particulate aerosol than for example a coal-fired power station (Howard 2000). In this regard, incinerators are of utmost concern regarding health of the general public.

The new EC directive on incineration of waste does not give any limits for PM10, or perhaps even more appropriately, PM 2.5, that is respirable particles, less than 2.5 μm. In this way the directive ignores the particulate pollution from incinerators which is of most relevance to public health. The directive does specify a limit for total dust emissions to air of 10 mg/m^3 from incinerators. Data published in the 1980s gave air emissions of particulate from UK MSW incinerators ranging from 18-4105 mg/m^3 (Williams 1990), and from US hazardous waste incinerators ranging from 4-902 mg/m^3 (Dempsey and Oppelt 1993). A recent report on MSW incinerators in Sweden reported particulate emissions of 0.003 to 64 mg/m^3. Four out of 21 Swedish incinerators exceeded the EC limit on dust emissions (Greenpeace Nordic 2000).

5.1.5 Inorganic Gases

Inorganic acidic gases, notably hydrogen chloride (HCl), hydrogen fluoride (HF), hydrogen bromide (HBr), sulphur oxides (SO_x), and nitrogen oxides (NO_x) are formed and emitted by incinerators. These gases arise as a consequence of the elements chlorine, fluorine, bromine, sulphur and nitrogen being present in waste (Williams 1990). NO_x are also formed as a result of the direct combination of nitrogen and oxygen, a process that is accelerated at high temperatures.

HCl is emitted in greater quantities from incinerators than from coal-fired power stations. This is due to chlorine in the waste, notably in the form of plastics such as PVC (Williams 1990). The new EC directive sets a limit (daily average value) of 10 mg/m^3 for HCl and 1 mg/m^3 for HF (EC 1998). A recent study of 21 Swedish MSW incinerators reported that HCl emissions to air from 17 of them exceeded the EC limit, often to a substantial degree (Greenpeace Nordic 2000). The average release from the 21 incinerators was 44 mg/Nm^3 with a range of 0.2-238 mg/Nm^3.

Oxides of nitrogen (NO_x), including nitrogen dioxide (NO_2), and oxides of sulphur (SO_x), including sulphur dioxide (SO_2), are emitted from industrial combustion processes including all types of incinerators. These gases can also influence the pH of rain, making it acidic. Over time, acid rain can have deleterious effects on soil and water quality, and adversely affect ecosystems. Like exposure to particulate air pollution, exposure to NO_x and SO_x is also linked to adverse effects on respiratory health of individuals with pre-existing respiratory disorders. For instance, research has shown associations between increased air pollution levels of SO_2 and increased premature deaths in individuals who had pre-existing respiratory or cardiovascular illness. Similarly an association is evident with increased hospital admissions in individuals with pre-existing respiratory illness such as asthma or chronic obstructive pulmonary disease. Studies have also shown associations between exposure to NO_2 and worsened symptoms of respiratory illness although the data is not consistent or conclusive (Ayres 1998).

NO_x and SO_x emissions also result in the formation of particulates, known as secondary particulates. The formation of secondary particulates occurs as a consequence of these gases undergoing chemical reactions in the atmosphere. They originate from the chemical oxidation of sulphur and nitrogen oxides in the atmosphere to acids, which are subsequently neutralised by atmospheric ammonia. The particles formed include ammonium sulphate and ammonium nitrate. These particles, which are generally soluble in nature, persist in the air for long periods of time. A less abundant type of secondary particle is ammonium chloride which originates from HCl gas. Like primary particles, secondary particles can have a wide variety of other potentially toxic organic compounds adsorbed onto their surfaces such as PAHs, and dioxins (QUARG 1996, COMEAP 1995, EPAQS 1995) (see section 5.1.4). Like primary particulates from incinerators, secondary particulates are also thought to have adverse impacts on human health (e.g. see EC 1998)

Presently, NOx emissions from incinerators are not regulated through EC limits although limits are proposed in the new EC directive. A limit (daily average value) for nitrogen monoxide and nitrogen dioxide, expressed as nitrogen dioxide, of 200 mg/m^3 is proposed (for existing incineration plants with a capacity exceeding 3 tonnes per hour, or new incineration plants). A recent study of 12 Swedish MSW incinerators documented emissions ranging from 1.2 – 236 mg/Nm^3. 4 of the 12 exceeded the EC limit.

The EC directive on incineration of wastes proposes a limit (daily average value) for sulphur dioxide of 50 mg/m^3. A

recent report on 10 Swedish incinerators found that emissions ranged from 1.2 to 236 mg/Nm^3. Of the 10 incinerators, 9 of them had emissions that exceeded the EC limit (Greenpeace Nordic 2000).

5.1.6 Other Gases

Carbon dioxide (CO_2) is emitted by incinerators. Municipal waste contains around 25% by weight of carbon and this is released as CO_2 when waste is burned. Approximately one tonne of CO_2 is produced per tonne of waste incinerated. CO_2 is a greenhouse gas that affects climate change and releases have to be kept as small as possible (EEA 2000). There is no EC limit on emissions of CO_2 from incinerators.

Carbon monoxide is also released from incinerators. It is potentially toxic and is also a greenhouse gas. Research suggests that increases in CO levels in the air may be linked to health impacts in certain susceptible individuals with pre-existing heart disease (Ayres 1998). A recent study on Swedish incinerators found that of the 15 incinerators which recorded emissions, 10 exceeded the new EC limit of 50 mg/Nm^3 (Greenpeace Nordic 2000). Emissions ranged from 2.6 to 249 mg/Nm^3.

5.2 Releases to Water

Incinerators emit wastes to water from cleaning equipment. Published scientific data on these emissions is very limited. Wastewater from wet exhaust gas cleaning contains heavy metals, the most significant in terms of quantity emitted and toxicity being lead, cadmium, copper, mercury, zinc and antinomy. Wastewater from wet slag removal equipment contains high levels of neutral salts and also contains unburned organic material from the residue (EEA 2000).

5.3 Releases to Ashes

Ashes from waste incineration generally contain the same pollutants as air emissions, but may differ in concentration and composition (EEA 2000). Fly ashes and bottom ashes contain dioxins and heavy metals although, as for air emissions, little is known about many other compounds present in fly ash.

5.3.1 Organic Compounds

Information about the contents of organic compounds in bottom ashes is scarce, with the exception of dioxins for which there are some data (EEA 2000).

Dioxins

Dioxin emissions from incinerators to air and water have decreased in recent years due to improvements in pollution control equipment. However, it is difficult to tell whether the total releases of dioxins from incinerators have declined at the same time. It is highly probable that while emissions to air via stack gases have decreased, releases with the ashes have increased. Indeed, it has been proposed that the total dioxin releases from incineration probably have not been reduced greatly in recent decades (Wikstrom 1999). A theoretical assessment of the total emissions from a MSW incinerator in Sweden also found that a reduction of dioxins emitted in flue gases would result in an increase in ash (GRAAB 1996). Thus, the total dioxin releases from the plant would remain the same, regardless of improvements in air pollution abatement technology.

There are relatively few data about dioxins in fly ashes and bottom ashes because many installations are not obligated to control them (Fabrellas *et al.* 1999, Greenpeace Nordic 1999). A theoretical assessment of releases from an incinerator in Sweden suggested that 97% of the total dioxin emissions from an incinerator would be present in the ash. This is in close agreement with direct measurements from an incinerator (Spittelau) in Austria, which showed that 99.6% of the total dioxin releases were in ash residues (Greenpeace Austria 1999). A study on a Spanish incinerator also noted that only a minor proportion of dioxin emissions is through stack gases, the majority being in ashes (Abad *et al.* 2000). In addition to chlorinated dioxins, it is also likely that other halogenated dioxins and furans are present in ashes, as in flue gases, such as brominated and mixed chlorinated/brominated compounds. A study on medical and MSW incinerator fly ashes found results suggesting that iodinated dioxins are also likely to be present (Kashima *et al.* 1999).

With regard to levels of dioxins in incinerator residues, the highest levels have been found in fly ash. Levels characteristically range from parts per trillion (ppt) to parts per billion (ppb), (EEA 2000). Research on eight MSW incinerators in Spain found mean levels in fly ash between 0.07 and 3.5 ng I-TEQ/g (ppb) (Fabrellas *et al.* 1999). Another study on a MSW incinerator in Spain reported levels which fell within this range from two measurements, which were 0.37 and 0.65 ng I-TEQ/g (ppb) (Abad *et al.* 2000). Particularly high levels were reported for one Spanish incinerator in 1997 (41 ppb TEQ) although levels in 1999 were lower (Stieglitz *et al.* 1999).

Lower concentrations are apparent in bottom ash samples, typically ppt levels (EEA 2000). For instance, mean values for 3 MSW incinerators in Spain were 0.006, 0.013 and 0.098 ng I-TEQ/g (ppb), (i.e. 6, 13 and 98 ppt TEQ), (Fabrellas *et al.* 1999). Similarly, levels in bottom ashes from five MSW incinerators in Bavaria, Germany ranged from 1.6 to 24 ppt TEQ (Marb *et al.* 1997). Ash from 18 new or updated medical waste incinerators in Poland sampled in 1994-7 had substantially higher levels of dioxins ranging from 8-45 ppb TEQ (Grochowalski 1998).

Based on limited sampling, Abad *et al.* (2000) noted that although the highest concentrations of dioxins are present in fly ash, the high production of bottom ash in incinerators means that the annual output of dioxins in bottom ash is comparable to that of fly ash. However, a study of eight MSW incinerators in Spain calculated that the overall output of dioxins was higher for fly ash (Fabrellas *et al.* 1999). The total yearly output of dioxins from 8 MSW incinerators reported to be operating in Spain, based on point measurements, was flue gases 1-1.2, fly ashes 46.6-111.6 and bottom ashes 2-19 g I-TEQ/y (Fabrellas *et al.* 1999).

As mentioned in the previous section, dioxin inventories most often underestimate releases from incinerators because ashes are not included in calculations. A report on output of dioxins from Swedish incinerators has proposed that the Swedish EPA have grossly underestimated total incinerator emissions by underestimating ash contamination in ashes (Greenpeace Nordic 1999).

Other Organic Compounds

As previously discussed in this report, emissions of organic compounds to stack gases are multitudinous and fly ashes are similarly laden with numerous compounds. The EEA (2000) note that fly ash contains concentrated organic compounds, such as PAHs and soot as well as chlorinated organic compounds. PCBs are known to be present in fly ash (see e.g. Sakai *et al.* 1996). PCBs were reported to be detected in fly ash of hospital and MSW waste incinerators (Magagni *et al.* 1994), and in sewage sludge incinerator fly ash and bottom ash (Kawakami *et al*. 1998). The level of PCBs in fly ash from sewage sludge incinerators was 7.1 ng/g with the proportion of PCBs to dioxins being similar to that found in MSW incinerators. PCNs have also been identified in incinerator fly ash (Schneider *et al*. 1998).

A study on fly ash from MSW incinerators identified 72 different phenolic compounds in the ash including many unknown ones (Nito and Takeshita 1996). Most of the compounds were hydroxy compounds of PAHs, polychlorinated PAHs, PCBs and dioxins. The study noted that some of these halogenated hydroxy compounds may be persistent and toxic and their toxicities should be evaluated because they will be leached from fly ash into the environment after disposal in landfill. Another study identified many new kinds of aza-heterocyclic hydrocarbons (azaarenes and other basic compounds in fly ash (Nito and Ishizaki 1997). These compounds are produced by incomplete combustion and this study confirmed that incinerators are a source of them. The study identified 63 and 18 kinds of azaareness from two different fractions of fly ash respectively. Of these compounds, quinoline, alkylquinoline, benzoquinoline, benzacridine, azapyrene, azabenzopyrene, phenylpyridine, biphenylamine and their isomers comprised the majority. Of concern is that many of them are known to be carcinogenic or mutagenic compounds. Leaching of such compounds from fly ash in landfill would release these toxic chemicals into the environment.

5.3.2 Heavy Metals

Both fly ash and bottom ash residues from incinerators contain many heavy metals. Fly ash generally has higher metal concentrations than bottom ash if the large, unburned metal fragments from the bottom ash are excluded (Bucholz and Landsberger 1995). Table 5.4 shows concentrations of heavy metals detected in fly ash and bottom ash from two Spanish MSW incinerators (Alba *et al.* 1997) and table 5.5 shows concentrations detected in ashes from a US incinerator (Bucholz and Landsberger 1995). The concentrations of heavy metals in incinerator ashes are very high compared to background levels in the environment. For instance, if concentrations in bulk ash (combined fly + bottom ash) are compared with average concentrations of heavy metals found in soil globally, it is clear that bulk ash contains elevated amounts of many metals (Bucholz and Landsberger 1995). In addition, the process of incineration greatly enhances the mobility and bioavailability of toxic metals compared with raw municipal waste (Schumacher *et al.* 1998). Consequently, there is greater potential for leaching of metals into the environment from ashes dumped in landfill than from ordinary waste (see section 5.4.1).

A study on incinerator ashes from veterinary college incinerators in which animal carcasses are burned found that levels of metals varied considerably between incinerators (Thompson *et al.* 1995). Generally levels of metals in the ashes were much lower than levels found in MSW incinerator ashes. One exception was zinc, which was at a similar level. It was noted that burning of plastics in the waste may contribute to lead and zinc content in the ashes.

Given that incinerator companies are not required under national laws in many countries to routinely monitor ashes, published data on heavy metal levels in ashes and exceeding of those regulatory limits which are in place are sparse. One study in the US of hazardous waste incinerators that the metals which most frequently exceeded regulatory limits were arsenic, nickel and lead (Dempsey and Oppelt 1993).

Table 5.4 Ranges of elemental abundance in MSW incinerator ashes and in soil. All concentrations are in mg/kg unless otherwise specified.

Element	Fly Ash	Bottom Ash	Soil
Ag	46-55.3	17.5-28.5	0.1
Al	3.19-7.84%	6.20-6.68%	7.1%
As	269-355	47.2-52.0	6
Br	3830-3920	676-830	5
Cd	246-266	47.6-65.5	0.06
Co	11.3-13.5	65.2-90.3	8
Cr	146-169	623-807	100
Cu	390-530	1560-2110	20
Hg	59.1-65.0	9.1-9.7	0.03
In	1.50-1.67	0.45-0.71	0.07
Mo	14-26	100-181	2
Pb	3200-4320	2090-2860	10
Se	6.7-11.2	<2.52	0.2
Sn	470-630	300-410	10
Th	2.85-3.21	4.31-4.86	5
Ti	3300-6300	7500-18100	5000
V	27-36	46-137	100
Zn	13360-13490	6610-6790	50

Source: Buchholz and Landsberger (1995).

Table 5.5 Minor and trace element concentrations in MSW incinerator residues

Element	Fly Ashes (mg/kg dry residue)	Bottom Ashes (mg/kg dry residue)
Cr	365 18	210 8
Zn	9382 208	2067 ± 9
Pb	5461 236	1693 ± 22
Ni	117 2	53 ± 3
Cu	1322 90	822 ± 4
As	<50	<50
Cd	92 2	<12.5
Hg	0.29 0.03	<0.035

Source: Alba *et al.* (1997).

5.4 Disposal of Ashes

Fly ashes are potentially toxic because of their heavy metal and salt content and consequently they require proper management (Alba *et al.* 1997). They also contain other organic toxic chemicals including dioxins. According to the EEA (2000), the disposal of fly ash from waste incineration plants is a serious problem. Under some regulations fly ashes could be classified as hazardous waste (Alba *et al.* 1997). Indeed, due to the high content of lead and cadmium in fly ash, it is classified as toxic waste under Italian law (Magagni *et al.* 1994). In response to concerns regarding incinerator ash disposal, the International Ash Working Group was established to compile and evaluate available information (Sawell *et al.* 1995) and has subsequently published its findings (Chandler *et al.* 1997).

Unlike fly ash, bottom ash is generally not classified as special waste. Nevertheless, bottom ashes also contain toxic substances and according to information cited by Brereton (1996), the potential leaching rates of metals from bottom ashes are such that there is clearly an environmental concern attached to their disposal.

Presently, fly ash is usually disposed of in landfill whilst bottom ash is disposed of in landfill or is used in

construction materials. In Canada, most European countries and Japan, bottom ash is handled separately from fly ash, whereas the current trend in the US is to combine all the residues and dispose of this waste in dedicated landfills (Chandler *et al.* 1997). The cost of disposal of ashes is a significant impact on the total cost of incineration (Brereton 1996). Utilisation of ash for construction purposes reduces the costs of ash disposal. However, the hazardous nature of incinerator ashes, and the eventual release of hazardous compounds such as persistent chemicals or heavy metals back into the environment calls into question this use. Furthermore, Shane *et al.* (1993) showed that the extent to which ashes were mutagenic varied with time. For example, samples taken at different times from the same incinerator varied in their mutagenic potential. Since it is unlikely that incinerator ashes are regularly checked for mutagenicity this again raises questions about further uses. It has been noted that another possible use for incinerator ash is as a fertiliser. However, the uptake of certain metals such as cadmium from MSW ash amended soil into edible plants, and thus into the human food supply, often precludes the use of fly ash in this manner (see Shane *et al.* 1993). The uses of fly ash and bottom ash for construction and other purposes are further discussed in section 5.4.1 below.

5.4.1 Disposal of Fly Ash

In the UK, it has been reported that fly ashes are disposed of in ordinary landfills, some of which are unlined (Mitchell *et al.* 1992). This is of great concern because toxic components in the ashes, in particular heavy metals, will contaminate subsoil above background levels. Depending on the pH of the soil, rainfall can leach metals from the landfilled ash into groundwater used for drinking. Leaching is greatest under acidic conditions. Since the ashes are frequently co-disposed with ordinary municipal waste, the surrounding soils can be acidified through organic acids which are the breakdown products of landfilled waste. This leads to greater leaching of heavy metals (Marty 1993). Furthermore, dumping of incinerator ash in landfills is of greater significance than normal waste going to landfill because not only is the concentration of metals higher in ash than normal waste, but it is also likely to be in a more soluble form and therefore more likely to leach. In one UK study, it was noted that levels of zinc, lead and cadmium were of particular concern in incinerator fly ash (Mitchell *et al.* 1992). With regard to dioxins, according to the EEA (2000), these chemicals are strongly bound to the surface of ash residues, are highly insoluble in water, and consequently they will not leach to a significant extent from landfills to groundwater.

Tests on leaching of metals from incinerator ashes have shown that the quantity of elements/heavy metals, which leach, is determined in particular by pH. The more acidic the solution used, the greater the amount of leaching (e.g. Fleming *et al.* 1996, Buchholz and Landsberger 1995). Significant releases of cadmium, lead and chromium however have been found to leach under neutral conditions with distilled water (Mangialardi *et al.* 1998). Lead has been deemed the most leachable heavy metal from fly ash (Chandler *et al.* 1997). Studies on the leaching of heavy metals from incinerator ash with water that simulates acid rain, has shown that leaching of metals to a significant degree occurs most readily with the first washing of the ashes (Buchholz and Landsberger 1995). This study noted that from this initial leaching, the metals/elements Ag, Ba, Be, Cr, Cu, Mo, Pb, S, Ti, and Zn appeared to pose the greatest threat to groundwater. Leaching over longer time periods was much less, but As, Cd, Cu, Hg, Pb, S and Zn were identified as potential long-term hazards over the lifetime of ash dumped in a landfill. In terms of very long time periods over hundreds to thousands of years, it has been noted that little is known about the long-term leaching behaviour of incinerator residues (Chandler *et al.* 1997). This is of immense concern given that landfills are unlikely to be managed indefinitely.

Currently, at landfills where leachate from the waste is collected, it is generally disposed of to municipal wastewater treatment plants. Such leachates from fly ash in landfills may be particularly high in lead and cadmium (Chandler *et al.* 1997). These and other trace metals will thus be directly discharged to the environment where leachate is disposed of via the general wastewater treatment system.

In addition to leaching of chemical contaminants from landfills, pollutants may also re-enter the environment via landfill fires. Landfill fires have been reported to be common in Finland and research has shown the release of dioxins, PCBs, PAHs and other contaminants from such fires in Sweden and Finland (see Ruokojärvi *et al.* (1995).

Pre-treatment of fly ash before disposal is being used increasingly in an attempt to reduce leaching. In their document on dangerous substances in waste, the EEA stipulate that fly ash cannot be landfilled without pre-treatment (EEA 2000). The focus on pre-treatment has been towards a minimum cost treatment which brings leachability into conformance with guidelines for disposal. This most commonly involves stabilisation of the ash in cement. According to Brereton (1996), the stabilised waste may then be suitable as some form of fill, or should be suitable for regular landfill. Chandler *et al.* (1997) report that some incinerators in Germany, Sweden, Switzerland and Austria stabilise fly ash using cement. Once stabilised, the use of fly ash in construction materials is not common in many countries. Exceptions are the Netherlands where about 50% of fly ash is used as filler in asphalt and Austria where the ash is used in concrete construction

(Greenpeace Austria 1999). On this note, it is of concern that a study on the use of fly ash for construction material has shown that these materials may subsequently leach metals (Fleming *et al*. 1996). Furthermore, whether fly ash is directly landfilled, is stabilised and then landfilled, or is stabilised in construction materials, it is important to realise that weathering and erosion will eventually result in the re-entry of persistent pollutants from the ash, including heavy metals, back into the environment.

Another treatment of fly ash has involved further thermal treatment in an attempt to reduce dioxin content. This has been successful under experimental conditions (e.g. Buekens and Huang 1998). However, nothing appears to have been reported on the formation of other potentially toxic chemicals as a result of the process. Moreover, heavy metals will remain in the waste.

5.4.2 Disposal of Bottom Ash

Like fly ash, bottom ash from incinerators is either land-filled or is used for construction purposes. Tests on leachate from bottom ash in landfills has revealed leaching of inorganic salts, but negligible leaching of heavy metals in the short term (Chandler *et al.* 1997). In some European countries, including, Denmark, France, Germany, The Netherlands, significant quantities (40 to 60% or more) of bottom ash from incinerators is being used in construction purposes (Chandler *et al.* 1997). It is largely used as base and sub-base for road construction. It is also used under cycle paths. Research on the use of bottom ashes in concrete has determined that such concrete has a lower compressive strength than concrete made with conventional aggregate (Chang *et al*. 1999). It is important to note that there are serious and legitimate concerns regarding the use of bottom ash in construction materials due to the presence of toxic components in the ash which could later enter the environment. The future release of these compounds due to weathering and degradation may have detrimental consequences for man, particularly in cases where the substances may enter the food chain (Korzun and Heck 1990).

Some of the possible dangers of utilising fly ash and bottom ash have recently become apparent in the UK (ENDS 2000a). Many MSW incinerators were compulsory closed in the UK by the end of 1996 to comply with the EC "Air Framework Directive" (84/36/EEC) and the "Incineration of Municipal Wastes Directive" (89/429/EEC), (see Leach *et al.* 1999). One of the remaining and presently operating incinerators that was deemed to comply with the EC directives was the Byker incinerator, sited in Newcastle. From 1994 to 1999, a mixture of fly ash and bottom ash from the Byker incinerator in Newcastle has been used on allotments and on paths. Concern by local residents about possible toxic substances in the ash prompted the local health authority and council to organise an analysis of dioxins and heavy metals in the ash. Initial results showed high levels of dioxins in the ash and residents were advised that children under two years of age should not play on the allotments, eggs and animal produce from the allotments should not be consumed, and all vegetables should be washed or peeled before eating. The final results of the analysis showed that levels of several heavy metals in the ash and dioxins were far higher than usual background levels. The average concentration of dioxin was very high, 1373 ng TEQ/kg, with a maximum concentration of 4224 ng TEQ/kg. These levels exceed the relevant German regulatory guidelines for dioxins. For instance, restrictions on growing of agricultural crops are recommended above 40 ng TEQ/kg, and it is recommended that remediation should be carried out if playgrounds exceed 100 ng TEQ/kg and if residential areas exceed 1000 ng TEQ/kg. With the exception of mercury, all the other heavy metals tested exceeded the Dutch trigger values for soils, as shown in table 5.6. The Dutch guidelines are used by planning authorities in Britain. As a consequence of the high levels of toxic substances in the ash, all of it had to be removed. This was at a cost to the local council of £50-70,000. It is of great concern that the use of this ash for paths and allotments was permitted to happen by the regulatory authorities and begs the question whether similar incidences have occurred but remained unnoticed in the UK or in other countries.

The new EC directive (EC 1999) does not propose any limits for the quantity of heavy metals in fly ash or bottom ash. This is of concern given the fact that most heavy metals from incineration are sequestered in the ashes and pose an environmental contamination problem. The directive does state however that

"*appropriate tests shall be carried out to establish the physical and chemical characteristics and polluting potential of the different incineration residues. The analysis shall concern in particular the total soluble fraction and heavy metals soluble fraction*".

It also states that

"*residues shall be recycled as far as possible directly in the plant or outside in accordance with relevant Community legislation and national provisions*". Thus the EC condones the use of ashes for other purposes, which could lead to future environmental contamination and threats to health as discussed and exemplified above.

Table 5.6 Levels of metals (mg/kg) and dioxins and furans (ng/kg) in 16 Byker ash samples compared with Dutch Trigger Values

Substance	**Mean (mg/kg)**	**Range (mg/kg)**	**Dutch Trigger Value (mg/kg)**
Arsenic	12	7-23	20
Cadmium	5	0.4-11	1
Chromium	88	13-182	100
Copper	1,195	10-3,620	50
Mercury	0.2	0.1-0.6	0.5
Nickel	55	14-187	50
Lead	399	17-620	50
Zinc	659	31-1,420	200
Dioxins	1,373 ng TEQ/kg	11-4224 ng TEQ/kg	

Source: Buchholz and Landsberger (1995).

6. THE SOLUTION: REDUCE, RE-USE AND RECYCLE and PHASE OUT INCINERATION.

A lack of landfill space, tighter regulations to restrict the quantity of waste going to landfill together with environmental problems with old landfills have driven municipalities in many countries to look for new methods of handling waste. Presently, 60% of waste generated throughout countries in the European Union goes to landfill (Hens *et al.* 2000). This situation is made worse by the growing amount of waste being generated. For example:

- Total waste production in the EU rose by nearly 10% between 1990 to 1995 and a further 20% increase has been predicted to occur by 2010 (EEA 1999).
- In Estonia, Slovenia, Lithuania, Slovak Republic, Bulgaria, Hungary, Czech Republic, Romania, and Poland, economic growth may lead to a doubling of municipal waste generation by 2010 (EEA 1999).
- In Asia, municipal waste from urban areas is predicted to double by 2025 (World Bank 1999).

One of the methods being chosen to deal with the current waste crisis is incineration, a method which is promoted as reducing the volume of solid waste thereby lessening the burden on landfill. However, incinerators are not the solution to the waste problem. Indeed, they are symptoms of non-existent and/or ill-conceived policies for the management of material resources. In a world of shrinking resources, it is irrational to let valuable resources "go up in smoke," and doubly so when the smoke is known to carry persistent and other hazardous chemicals. Incineration cannot be regarded as a sustainable technology for waste management and has no place in a world striving to change towards zero discharge technologies.

It is notable that incineration has already been banned by the government of the Philippines, a move primarily instigated by public opposition to incineration. The Philippines is the first country in the world to ban incineration on a national scale. The Philippine Clean Air Act of 1999 specifically bans the incineration of municipal, medical and hazardous wastes and recommends the use of alternative techniques (for municipal waste) and non-burn technologies. Waste reduction, re-use and recycling are being promoted. The Clean Air Act mandates a three-year phase out period for existing medical incinerators, and during this time, limits hospitals to incineration of infectious waste.

6.1 Problems of Incineration

6.1.1 Environment and Health

No matter how modern an incinerator is, these facilities inevitably result in the release of toxic emissions to air and the production of toxic ashes and residues. This leads to contamination of the environment and to potential exposure of animals and humans to hazardous pollutants. Many hazardous compounds are released from incinerators including organic chemicals such as chlorinated and brominated dioxins, PCBs and PCNs, heavy metals, sulphur dioxide and nitrogen dioxide. Furthermore, innumerable substances are emitted which are of unknown toxicity. The entire impact on human health of exposure to the whole mixture of chemicals emitted from incinerators is unknown. However, studies imply that individuals who work at waste incinerators and who live near incinerators have suffered from increases in the rate of mortality as well as many other diseases and effects that diminish the quality of their lives. Moreover, a prestigious scientific body has recently expressed "substantial" concern about the impacts of incinerator-derived dioxin releases on the health and well-being of broader populations, regardless of the implementation of maximum achievable control technology (NRC 2000).

6.1.2 Economics

The economics of waste management in general, and in particular incineration, are extremely complex and are outside the scope of this report. Briefly, it has been noted that incineration is a technology of the previous industrial era and is only economically feasible if much of its cost is externalised ie. borne by the general public. Pollution control constitutes a major proportion of the cost, but using such technology to reduce the toxics entering the air cannot help but redistribute them back to deposits in the ash.

A recent trend has been to generate electricity from burning waste in MSW incinerators. This can only be seen as a by-product of incineration and not a contributor to sustainable energy production. Indeed, incinerators such as MSW incinerators, are inefficient energy producers with only 20% of the energy generated by the waste usually being captured. Murray (1999) has described incineration as inefficient both as a disposal option and as an energy generator. It leads not to material conservation and hazard reduction but to material destruction and hazard creation.

In the UK, a situation has arisen whereby contracts with incinerator operators lock local authorities into long term commitments to provide huge amounts of waste each year. This works against waste prevention, re-use and recycling since local authorities would have to pay financial penalties to incinerator owners if waste was reduced and diverted to re-use/recycling schemes.

6.1.3 Sustainability

The Convention for the Protection of the Marine Environment of the North East Atlantic (the OSPAR Convention, formed from the amalgamation of the former Oslo and Paris Conventions) entered into force in March 1998 and covers the 15 States of the North East Atlantic

Region and the European Union. At the OSPAR meeting held in Sintra in June 1998, Ministers at OSPAR agreed on a clear commitment for the cessation of release of hazardous substances within one generation (by 2020). In essence, the commitment means that a target has been set for the cessation of discharges, emissions and losses of hazardous substances (or the processes that generate them) and their substitution with non-hazardous alternatives. In practice this means a shift away from dirty technologies towards clean production and zero emission strategies. Incinerators can never comply with the zero emissions strategy or be classed as a clean production technology. This old, dirty technology is not in agreement with sustainable development or political commitments already made within Europe. In effect, under the provisions of the OSPAR agreement, incineration is finally and irrevocably made obsolete.

6.2 Current EU Policy and Waste Management

Waste policy in the EU widely accepts the hierarchy of waste management to be (in order of priority): waste prevention – re-use – recycling – thermal decomposition with energy recovery (i.e. incineration with energy recovery). In spite of this general consensus, and a growing coherence of this hierarchy in policy lines of individual EU member states as a consequence of EU-Directives, the majority of waste in Europe is either landfilled or incinerated. Importantly, these are the methods which also entail the highest and most serious environmental and health risks (Hens *et al.* 2000).

A move towards a waste policy aimed at reducing health effects should put more emphasis on prevention and re-use. Presently, EU waste policy is not founded upon health data. Fortunately the available data on health effects from waste management do not conflict, and in important aspects even coincide with the hierarchy proposed by the EU (Hens 2000). For example, waste prevention is deemed to be the most important (no waste equals no health effects), followed by re-use and recycling. Despite this, the lack of consideration of the environment and human health is clearly visible in EU policy. For instance, regulations put in place for incineration by the EU together, with national limits on this issue, are based on what is technically achievable rather than on health and environmental data.

Although emission limits set in the new EU directive have resulted in the closure and upgrading of some older incinerators in European countries, the policy itself is already outdated with regard to the OPSPAR agreement to phase out the releases of all hazardous substances within one generation. The EU directive is based on the conception that small releases of hazardous substances are acceptable. This is the conventional (though misguided) approach which proposes that chemicals can be managed at "safe" levels in the environment. However, it is already known, or is a scientific opinion, that there are no "safe" levels of many environmental chemical pollutants such as dioxins, other persistent, bioaccumulative and toxic chemicals and endocrine disruptors. In addition, the abandonment of the principle is increasing in political circles. For instance, with regard to incineration, the UK environment minister, Mr. Michael Meacher, recently recognised the futility of the conventional approach to chemicals regulation when he said:

Q440…"I repeat that emissions from incinerator processes are extremely toxic. Some of the emissions are carcinogenic. We know scientifically that there is no safe threshold below which one can allow such emissions" (cited in Howard 2000).

Despite the commitment by the OSPAR Convention for the cessation of all hazardous substances by 2020, a recent trend for plans to build new incinerators by the government in the UK and other European countries continues.

6.3 The Way Forward: Adoption of the Precautionary Principle and Zero relaese Strategy

6.3.1 Adoption of the Precautionary Principle

The precautionary principle acknowledges that, if further environmental degradation is to be minimised and reversed, precaution and prevention must be the overriding principles of policy. It requires that the burden of proof should not be laid upon the protectors of the environment to demonstrate conclusive harm, but rather on the prospective polluter to demonstrate no likelihood of harm. The precautionary principle is now gaining acceptance internally as a foundation for strategies to protect the environment and human health (Stairs and Johnston 1991).

Current regulation for incinerators is not based on the precautionary principle. Instead it attempts to set limits for the discharge of chemicals into the environment which are designated as "safe". In the current regulatory system the burden of proof lies with those who need to 'prove' that health impacts exist before being able to attempt to remove the cause of the problem and not with the polluters themselves (Nicolopoulou-Stamati *et al.* 2000). Based on knowledge regarding the toxic effects of many environmental chemical pollutants, which has accumulated over recent decades, a more legitimate viewpoint is that "chemicals should be considered as dangerous until proven otherwise".

We have now reached a situation, and indeed did some time ago, where health studies on incineration have

reported associations between adverse health effects and residing near to incinerators or being employed at an incinerator. These studies are warning signs which should not result in government inactivity, but rather to decisions being taken which implement the precautionary principle. There is already sufficient human health and environmental contamination evidence to justify a phase out of the incineration process based on the precautionary principle. To wait for further proof from a new generation of incinerators from an already harmful and dirty technology would be a blatant disregard for the environment and human health.

6.3.2 Adoption of Zero Discharge

The aim of "zero discharge" is to halt environmental releases of all hazardous substances. Although it is sometimes discussed as being simplistic or even impossible, it is a goal whereby regulation can be seen as resting places on the way to achieving it (Sprague 1991).

Zero discharge necessitates the adoption of clean production techniques both in industry and agriculture. It is essential that the change to clean production and material use should be fully supported by fiscal incentives and enforceable legislation.

The principle of clean production has already been endorsed by the Governing Council of the UNEP and has received growing recognition at a wide range of international fora. For instance, the adoption of the one generation goal for the phase out of all hazardous substances by the OSPAR Convention in 1998 necessitates instigating clean production technology under a zero discharge strategy.

In terms of waste management strategies, incineration is a dirty technology that can never fulfil the criteria of zero discharge. The way forward for waste management in line with a zero emissions strategy and hence towards sustainability, lies in waste prevention, re-use and recycling. In other words the adoption of the already well known principle of "REDUCE, RE-USE AND RECYCLE".

6.3.3 Implementation of REDUCE, RE-USE AND RECYCLE

We live in a world in which our resources are generally not given the precious status by industry and agriculture which they deserve. In part, this has led to the creation, particularly in industrialised countries, of a "disposable society" in which enormous quantities of waste, including "avoidable waste" are generated. This situation needs to be urgently changed so that the amount of waste produced both domestically and by industry is drastically reduced.

Ways to help waste reduction include the use of economic instruments and environmental taxes. The use of these measures is supported by the EC and a number of environmental taxes are already in place in several European countries (Steenwegen 2000). However, far more action is presently required to stimulate the change needed for much more waste reduction to become a reality.

Current levels of recycling in European countries vary considerably. For instance, The Netherlands recycles 46% of municipal waste whereas the UK only manages 8%. Intensive re-use and recycling schemes could deal with 80% of municipal waste. It is recognised that fiscal measures can play a considerable role in encouraging re-use and recycling schemes whilst discouraging least desirable practices such as incineration and landfill (Steenwegen 2000).

Measures to be taken in the drive towards increased waste reduction, re-use and recycling, and therefore towards lessening the adverse health effects from waste management should include:

- The phase out of all forms of industrial incineration by 2020, including MSW incineration. This is in line with the OSPAR Convention for the phase out of emissions, losses and discharges of all hazardous substances by 2020.

- Financial and legal mechanisms to increase re-use of packaging (e.g. bottles, containers) and products (e.g. computer housings, electronic components).

- Financial mechanisms (such as the landfill tax) used directly to set up the necessary infrastructure for effective recycling.

- Stimulating markets for recycled materials by legal requirements for packaging and products, where appropriate, to contain minimum amounts of recycled materials.

- Materials that cannot be safely recycled or composted at the end of their useful life (for example PVC plastic) must be phased out and replaced with more sustainable materials.

- In the short term, materials and products that add to the generation of hazardous substances in incinerators must be prevented from entering the waste stream at the cost of the producer. Such products would include electronic equipment, metals and products containing metals, such as batteries and florescent lighting, and PVC plastics

(vinyl flooring, PVC electrical cabling, PVC packaging, PVC-*u* window frames etc) and other products containing hazardous substances.

and more generally:

- Further the development of clean production technologies which are more efficient in terms of material and energy usage, produce cleaner products with less wastes and which ultimately can operate in a "closed loop" configurations to serve the needs of society in a more equitable and sustainable manner;

- Implement fully the Precautionary Principle, such that, in the future, we may be better able to avoid problems before they occur. The continuation and further development of scientific research has a fundamental role to play in identification of potential problems and solutions, but we must be ready to take effective precautionary action to prevent environmental contamination and degradation even in the face of considerable and often irreducible uncertainties.

7. REFERENCES

Aelvoet W., Nelen V., Schoeters G., Vanoverloop J., Wallijn E., Vlietinck R. (1998). Risico op gezondheidsschade bij kinderen van de Neerlandwijk to Wilrijk, Studie uitgevoerd in opdracht van de Neerlandwijk to Wilrijk, Gezondheidsbeleid, Document 1998/TOX/R/030. (in Dutch). (Cited in van Larebeke 2000).

Alba N., Gasso S., Lacorte T. and Baldasano J.M. (1997). Characterization of municipal solid waste incineration residues from facilities with different air pollution control systems. Journal of the Air and Waste Management Association 47: 1170-1179.

Abad E., Caixach J. and Rivera J. (1997). Dioxin like compounds from MWI emissions: assessment of polychlorinated napthalenes presence. Organohalogen Compounds 32: 403-406.

Abad E. Caixach J. and Rivera J. (1999). Dioxin like compounds from MWI emissions: assesment of polychlorinated napthalenes presence. Chemosphere 38 (1): 109-120.

Abad E., Adrados M.A., Caixach J., Fabrellas B. and Rivera J. (2000). Dioxin mass balance in a municipal waste incinerator. Chemosphere 40: 1143-1147.

Akagi, H., Malm, O., Kinjo, Y., Harada, M., Branches, F.J.P, Pfeiffer, W.C. and Kato, H. (1995). Methylmercury pollution in the Amazon, Brazil. The Science of the Total environment 175: 85-95.

Alcock R., Gemmill R. and Jones K. (1998). An updated UK PCDD/F atmospheric emission inventory based on a recent emissions measurement programme. Organohalogen Compounds 36: 105-108.

Allsopp M., Santillo D. and Johnston P. (1997). Poisoning the Future: Impact of Endocrine-Disrupting Chemicals on Wildlife and Human Health. Greenpeace International. ISBN 90-73361-40-0.

Allsopp M.,. Santillo D., Johnston P. and Stringer R. (1999). The Tip of the Iceberg: State of Knowledge of Persistent Organic Pollutants in Europe and the Arctic. Greenpeace International. ISBN 90-73361-53-2.

Allsopp M., Erry B., Stringer R., Johnston P. and Santillo D. (2000). Recipe for Disaster: a review of persistent organic pollutants in food. Greenpeace Research Laboratories. ISBN 90-73361-63-X.

An H., Englehardt J., Fleming L. and Bean J. (1999). Occupational health and safety amongst municipal solid waste workers in Florida. Waste Management Research 17: 369-377.

Angerer J., Heinzow D.O. Reimann W., Knorz W. and Lehnert G. (1992). Internal exposure to organic substances in a municipal waste incinerator. Int. Arch. Occup. Environ. Impact Assess. Rev. 8: 249-265. (Cited in NRC 2000).

Ardevol E., Minguillon C., Garcia G., Serra M.E., Gonzalez C.A., Alvarez L., Eritja R. and Lafuente A. (1999). Environmental tobacco smoke interference in the assessment of the health impact of a municipal waste incinerator on children through urinary thioether assay. Public Health 113: 295-298.

ATSDR (1993). Agency for Toxic Substances and Disease Registry. Study of Symptom and Disease Prevalence, Caldwell Systems, Inc. Hazardous Wast Incinerator, Caldwell County, North Carolina. Final Report. ATSDR/HS-93/29. U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia, USA. (Cited in NRC 2000).

ATSDR (1997). ATSDR's toxicological profiles on CD-ROM. U.S. Department of Health and Human Services, Public Health Service, CRC Press Inc, Boca Raton.

Ayres J.G. (1997). Trends in air quality in the UK. Allergy 52 (suppl 38): 7-13.

Ayres J.G. (1998). Health effects of gaseous air pollutants. In: Air Pollution and Health. Issues in Environmental Science and Technology 10 (eds.) R.E. Hester and R.M. Harrison. The Royal Society of Chemistry. ISBN 0-85404-245-8.

Babone F., Bovenzi M., Cavallieri F. and Stanta G. (1994). Air pollution and lung cancer in Trieste, Italy. Am. J. Epidemiol 141: 1161-1169 (Cited in Biggeri *et al.* 1996).

Bache C.A., Elfving D.C., Lisk D.J. (1992). Cadmium and lead concentration in foliage near a municipal refuse incinerator. Chemosphere 24 (4): 475-481.

Bache C.A., Gutenmann W.H., Rutzke M., Chu G., Elfving D.C. and Lisk D.J. (1991). Concentration of metals in grasses in the vicinity of a municipal waste incinerator. Arch. Environ. Contam. Toxicol. 20: 538-542.

Baldassarri, L., Bocca, A., di Domenico, A., Fulgenzi, A. and Lacovella, N. (1994 GC-MS isomer-specific determination of PCBs and some chlorinated pesticides in milk and cheese samples. Organohalogen Compounds 20: 221-224.

Bernard, A.M., Vyskocil, A., Kriz, J., Kodl, M. and Lauwerys, R. (1995). Renal effects of children living in the vicinity of a lead smelter. Environmental Research 68: 91-95.

Blumenstock M., Zimmermann R., Schramm K.W. and Kettrup A. (2000). Influence of combustion conditions on the PCDD/F-, PCB-, PCBz and PAH-concentrations in thechamber of a waste incineration pilot plant. Chemosphere 40: 987-993.

Biggeri A., Barbone F., Lagazio C., Bovenzi M. and Stanta G. (1996). Air pollution and lung cancer in Trieste, Italy: Spatial analysis of risk as a function of distance from sources. Environmental Health Perspectives 104 (7): 750-754.

Born J.G.P (1996). Reduction of (dioxin) emissions by municipal solid waste incineration in the Netherlands. Organohalogen Compounds 27: 46-49.

Brereton C. (1996). Municipal solid waste – incineration, air pollution control and ash management. Resources, Conservation and Recycling 16: 227-264.

Bresnitz E.A., Roseman J., Becker D. and Gracely E. (1992). Morbidity among municipal waste incinerator workers. American Journal of Industrial Medicine 22: 363-378.

Buchholz B.A. and Landsberger S. (1995). Leaching dynamics studies of municipal solid waste incinerator ash. Journal of Air and Waste Management Association 45: 579-590.

Buekens A. and Huang H. (1998). Comparative evaluation of techniques for controlling the formation and emission of chlorinated dioxins/furans in municipal waste incineration. Journal of Hazardous Materials 62: 1-33.

Carpi A., Weinstein L.H. and Ditz D.W (1994). Bioaccumulation of mercury by sphagnum moss near a municipal solid waste incinerator. Air and Waste 44 (May): 669-672.

Carpi A. (1997). Mercury from combustion sources: a review of the chemical species emitted and their transport in the atmosphere. Water, Air, and Soil Pollution 98: 241-254.

Chandler A.J., Eighmy T.T., Hartlen J., Hjelmar O., Kosson D.S., Sawell S.E., van der Sloot H.A. and Vehlow J. (1997). Studies in Environmental Science 67: Municipal solid waste incinerator residues. The International Ash Working Group (IAWG). Published by Elsevier 1997.

Chang N-B., Wang H.P., Huang W.L. and Lin K.S. (1999). The assessment of reuse potential for municipal solid waste and refuse-derived fuel incineration ashes. Resources, Conservation and Recycling 25: 255-270.

Collett R.S., Oduyemi K. and Lill D.E. (1998). An investigation of environmental levels of cadmium and lead in airborne matter and surface soils within the locality of a municipal waste incinerator. The Science of the Total Environment 209: 157-167.

COMEAP, Committee on the Medical Effects of Air Pollutants (1995). Non-biological particles and health. Department of Health, UK. London: HMSO.

De Fre R. and Wevers M. (1998). Underestimation in dioxin inventories. Organohalogen Compounds 36: 17-20.

DeMarini D.M., Shelton M.L. and Bell D.A. (1996). Mutation spectra of chemical fractions of a complex mixture: role of nitroarenes in the mutagenic specificity of municipal waste incinerator emissions. Mutation Research 349: 1-20.

Deml E., Mangelsdorf I. And Greim H. (1996). Chlorinated dibenzodioxins and dibenzofurans (PCDD/F) in blood and human milk of non occupationally exposed persons living in the vicinity of a municipal waste incinerator. Chemosphere 33 (10): 1941-1950.

Dempsey C.R. and Oppelt E.T. (1993). Incineration of hazardous waste: a critical review update. Air and Waste 43: 25-73.

DETR (2000). Waste Strategy 2000, England and Wales, Part1, Part2. Published by Stationary Office Ltd. ISBN 010 146 932 2.

Diggle P.J. (1990). A point process modelling approach to raised incidence of a rare phenomenon in the vicinity of a prespecified point. J.R.Stat.Soc A 153: 349-362. (Cited in Elliot *et al.* 1992).

DoE/WO (1995) Making waste work: A strategy for sustainable waste management in England and Wales. UK Department of the Environment White Paper, CM3040, The Stationery Office, London

Domingo J.L., Granero S., Schuhmacher M., Llobet J.M., Sunderhauf W. and Muller L. (1998). Vegetation as a biomonitor of PCDD/PCDFs in the vicinity of a municipal solid waste incinerator. Organohalogen Compounds 36: 157-160.

EA (1997). Report on the operation of incinveration plant at the Coalite Chemical Works, Bolsover, Derbyshire, from commissioning to closure and the subsequent prosecution of the last operator Coalite Products Ltd by HM Inspectorate of Pollution under section 5 of the Health and Safety at Work Act, 1974. UK Environment Agency. HO-0/97-500-C-AZMI, 71pp

EC (1998). Proposal for a Council Directive on the incineration of waste. Brussels, 07.10.1998. COM(1998)558final. 98/0289 (SYN).

EC (1999). European Parliament and Council Directive on the incineration of waste. Brussels 12.07.1999. COM (1999) 330final. 98/0289 (COD).

EEA (1999). Environment in the European Union at the turn of the century.

EEA (2000). Dangerous Substances in Waste. Prepared by: J. Schmid, A.Elser, R. Strobel, ABAG-itm, M.Crowe, EPA, Ireland. European Environment Agency, Copenhagen, 2000.

Eikman T. (1994). Environmental toxicological assessment of emissions from waste incinerators. Fresenius Envir Bull 3: 244-249.

Elliot P., Eaton N., Shaddick G. and Carter R. (2000). Cancer incidence near municipal solid waste incinerators in Great Britain. Part 2: histopathological and case-note review of primary liver cancer cases. British Journal of Cancer 82 (5): 1103-1106.

Elliot P., Hills M., Beresford J., Kleinschmidt I., Jolley D., Pattenden S., Rodrigues L., Westlake A. and Rose G. (1992). Incidence of cancers of the larynx and lung near incinerators of waste solvents and oils in Great Britain. The Lancet 339 (April 4): 854-858.

Elliot P., Shaddick G., Kleinschmidt I., Jolley D., Walls P., Beresford J. and Grundy C. (1996). Cancer incidence near municipal solid waste incinerators in Great Britain. British Journal of Cancer 73: 702-710.

ENDS (1999). EC proposals on incineration may scupper several UK plants. ENDS Report 291, April: 38-39.

ENDS (2000a). Regulatory foul-ups contributed to Byker ash affair. Environmental Data Services Report 304 (May): 17-18.

ENDS (2000b). Agency reports decline in pollution around Welsh incinerator. ENDS Report 304, May: 19-20.

EPAQS, Expert Panel on Air Quality Standards, (1995). Particles. Published by HMSO. ISBN 0 11 753199 5.

Fabrellas B., Sanz P., Abad E. and Rivera J. (1999). The Spanish dioxin inventory: Proposal and preliminary results from municipal waste incinerator emissions. Organohalogen Compounds 41: 491-494.

Falandysz J. and Rappe C. (1997). Specific pattern of tetrachloronapthalenes in black cormorant. Chemosphere 35 (8): 1737-1746.

Falandysz, J., Strandberg, L., Bergqvist, P.-A., Strandberg, B. & Rappe, C. (1996). Chloronaphthalenes in stickleback Gasterosteus aculeatus from the southwestern part of the Gulf of Gdansk, Baltic Sea. Organohalogen Compounds 28: 446-451

Fangmark I., Stromberg B., Berge N. and Rappe C. (1994). Influence of postcombustion temperature profiles on the formation of PCDDs, PCDFs, PCBzs, and PCBs in a pilot incinerator. Environmental Science and Technology 28 (4): 624-629.

Feng Y. and Barratt R. (1999). Distributions of lead and cadmium in dust in the vicinity of sewage sludge incinerator. J. Environ. Monit. 1: 169-176.

Fiedler H. (1999). National and regional dioxin and furan inventories. Organohalogen Compounds 41: 473-476.

Fleming L.N., Abinteh H.N. and Inyang H.I. (1996). Leachant pH effects on the leachability of metals from fly ash. Journal of Soil Contamination 5 (1): 53-59.

Fomin A. and Hafner C. (1998). Evaluation of genotoxicity of emissions from municipal waste incinerators with Tradescantia-micronucleus bioassay (Trad-MCN). Mutation Research 414: 139-148.

Foxall C.D. and Lovett A.A. (1994). The relationship between soil PCB and PCDD/DF concentrations in the vicinity of a chemical waste incinerator in south Wales, UK. Organohalogen Compounds 20: 35-40.

Gass H.C., Jager E., Menke D. and Luder K. (1998). Long term study for minimization of the PCDD/PCDF – emissions of a municipal solid waste incinerator in Germany. Organohalogen Compounds 36: 175-178. GC-MS isomer-specific determination of PCBs and some chlorinated pesticides in milk and cheese samples. Organohalogen Compounds 20: 221-224.

Gatrell A.C. and Lovett A.A. (1989). Burning Questions: Incineration of wastes and implications for humans health. Paper presented at the Institute of British Geographers Annual Conference, Coventry Polytechnic, Jan 5th 1989.

Gonzalez, C., Kogevinas, M., Gadea, E., Huici, A., Bosch, A., Bleda, M., Papke, O., 2000. Biomonitoring study of people living near or working at a municipal solid-waste incinerator before and after two years of operation. Arch. Environ. Health 55:259-267.

Goyer, R.A. (1993). Lead toxicity: current concerns. Environmental Health Perspectives 100: 177-187.

GRAAB (1996). Tekniskt underlag dioxinier, MU 96:10.

Granero S., Domingo J.L., Schuhmacher M., Llobet J.M. and de Kok H.A.M. (1999). Monitoring PCDD/Fs in the vicinity of an old municipal waste incinerator, 1996-1998. Part 1: Soil monitoring. Organohalogen Compounds 43: 143-146.

Gray E., Peat J., Mellis C., Harrington J., and Woolcock, A. (1994). Asthma severity and morbidity in a population sample of Sydney school children: Part I – Prevalence and effect of air pollutants in coastal regions. Aust. N.Z. J. Med. 24:168-175. (Cited in NRC 2000).

Greenpeace Austria (1999). Waste incinerating plants in Austria. Vienna, August 1999.

Greenpeace Nordic (1999). Piles of Dioxin: Dioxin in ashes from waste incinerators in Sweden. Greenpeace, Nordic, November 1999.

Greenpeace Nordic (2000). Hot Air: Will Swedish Incinerators Satisfy the EU?

Grochowalski A. (1998). PCDDs and PCDFs concentration in combustion gases and bottom ash from incineration of hospital wastes in Poland. Chemosphere 37 (9-12): 2279-2291.

Gustavsson P. (1989). Mortality among workers at a municipal waste incinerator. American Journal of Industrial Medicine 15: 245-253.

Gustavsson P., Evanoff B. and Hogstedt C. (1993). Increased risk of esophageal cancer among workers exposed to combustion products. Archives of Environmental Health 48 (4): 243-245.

Gutenman W.H., Rutzke M., Elfving D.C. and Lisk D.J. (1992). Analysis of heavy metals in foliage near a modern refuse incinerator. Chemosphere 24 (12): 1905-1910.

Hansen E. (2000). Substance flow analysis for dioxins in Denmark. Environmental Project No. 570 2000. MiljØprojeckt. (Danish Environmental Protection Agency).
Harada, M. (1997). Neurotoxicity of methylmercury; Minamata and the Amazon. In Mineral and Metal Neurotoxicology. Yasui, M., Strong, M.J., Ota, K. and Verity, M.A.[Eds]. CRC Press Inc., ISBN 0849376645.
Heeb N.V., Dolezal I.S., Buhrer T., Mattrel P. and Wolfensberger M. (1995). Distribution of halogenated phenols including mixed brominated and chlorinated phenols in municipal waste incineration flue gas. Chemosphere 31 (4): 3033-3041.

Hens L., Nicolopoulou-stamati P., Howard C.V., Lafere J. and Staats de Yanes (2000). Towards a precautionary approach for waste management supported by education and information technology. In: Health Impacts of Waste Management Policies. Proceedings of the seminar "Health Impacts of Waste Management Policies", Hippocrates Foundation, Kos, Greece, 12-14 November 1998. Eds. P. Nicolopoulou-Stamati, L.Hens and C.V. Howard. Kluwer Academic Publishers.

Holdke B., Karmus W. and Kruse H. (1998). Body burden of PCB in whole human blood of 7-10 year old children living in the vicinity of a hazardous waste incinerator. Das Gesundheitswesen 60 (8-9): 505-512. (Abstract only).

Howard C.V. (2000). Particulate aerosols, incinerators and health. In: Health Impacts of Waste Management Policies. Proceedings of the seminar "Health Impacts of Waste Management Policies", Hippocrates Foundation, Kos, Greece, 12-14 November 1998. Eds. P. Nicolopoulou-Stamati, L.Hens and C.V. Howard. Kluwer Academic Publishers.

Howard C.V. (2000b). Foreward. In: R.L. Maynard and C.V. Howard (eds). Particulate Matter: Properties and Effects Upon Health, BIOS Scientific Publishers Ltd., Oxford, UK. pp 63-84, ISBN 1-85996-172X.

Huang H. and Buekens A. (1995). On the mechanisms of dioxin formation in combustion processes. Chemosphere 31 (9): 4099-4117.

Hulster A. and Marschner H. (1992). Transfer of PCDD/PCDF from contaminated soils to food and fodder crop plants. Organohalogen Compounds

Jay K. and Stieglitz L. (1995). Identification and quantification of volatile organic components in emissions of waste incineration plants. Chemosphere 30 (7): 1249-1260.

Jefferson D.A. and Tilley E.E.M. (1999). The structural and physical chemistry of nanoparticles. In: R.L. Maynard and C.V. Howard (eds). Particulate Matter: Properties and Effects Upon Health, BIOS Scientific Publishers Ltd., Oxford, UK. pp 63-84, ISBN 1-85996-172X. (Cited in Howard 2000).

Jimenz B., Eljarrat E., Hernandez L.M., Rivera J. and Gonzalez M.J. (1996). Polychlorinated dibenzo-p-dioxins and dibenzofurans in soils near a clinical waste incinerator in Madrid, Spain. Chemometric comparison with other pollution sources and soils. Chemosphere 32 (7): 1327-1348.

Johnke B. and Stelzner E. (1992). Results of the German dioxin measurement programme at MSW incinerators. Waste Management and Research 10: 345-355.

Johnston P.A., Santillo D. and Stringer R. (1996). Risk assessment and reality: recognsing the limitations. In: Environmental Impact of Chemicals: Assessment and Control. Quint M.D., Taylor D. and Purchase R. (eds.). Published by The Royal Society of Chemistry, special publication no. 176, ISBN 0-85404-795-6 (Chapter 16: 223-239).

Johnston P., Stringer R., Santillo D. and Howard V. (1998). Hazard, exposure and ecological risk assessment. In: Environmental Management in Practice, Volume 1: Instruments for Environmental Management. B. Nath, L. Hens, P. Compton and D. Devuyst (eds.).

Publ. Routledge, London. ISBN 0-415-14906-1: pp. 169-187.

Kashima Y., Mitsuaki M., Kawano M., Ueda M., Tojo T., Takahashi G., Matsuda M., Anbe K., Doi R. and Wakimoto T. (1999). Characteristics of extractable organic halogens in ash samples from medical solid waste incinerator. Organohalogen Compounds 41: 191-194.

Kawakami I., Sase E., Tanaka M. and Sato T. (1998). Dioxin emissions from incinerators for sludge from night soil treatment plants. Organohalogen Compounds 36: 213-216.

Kitamura K., Kikuchi Y., Watanbe S., Waechter G., Sakurai H. and Takada T. (2000). Health effects of chronic exposure to polychorinated dibenzo-p-dioxins (PCDD), dibenzofurans (PCDF) and coplanar PCBs (Co-PCB) of municipal waste incinerator workers. Journal of Epidemiology 10 (4): 262-270.

Knox E.G. (2000). Childhood cancers, birthplaces, incinerators and landfill sites. International Journal of Epidemiology 29: 391-397.

Knox E.G. and Gilman E.A. (1998). Migration patterns of children with cancer in Britain. J. Epidemiol Community Health 52: 716-726.

Korzun E.A. and Heck H.H. (1990). Sources and fates of lead and cadmium in municipal solid waste. Journal of Air and Waste Management Association 40 (9): 1220-1226.

Kurttio P., Pekkanen J., Alfthan G., Paunio M., Jaakkola J.J.K. and Heinonen O.P. (1998). Increased mercury exposure in inhabitants living in the vicinity of a hazardous waste incinerator: A 10-year follow-up. Archives of Environmental Health 53 (2): 129-137.

Leach J., Blanch A. and Bianchi A.C. (1999). Volatile organic compounds in an urban airborne environment adjacent to a municipal incinerator, waste collection centre and sewage treatment plant. Atmospheric Environment 33: 4309-4325.

Lee H., Kim M., Choi S., Park J., Moon K. and Ghim Y-S. (1997). Quantification of chronic inhaled exposure induced by dioxin emissions from some municipal waste incinerator using Monte-Carlo simulation. Organohalogen Compounds 34: 74-78.

Lee J-T. and Shy C.M. (1999). Respiratory function as measured by peak expiratory flow rate and PM10 six communities study. Journal of Exposure Analaysis and Environmental Epidemiology 9: 293-299.

Legator M.S., Singleton C.R., Morris D.L. and Philips D.L. (1998). The health effects of living near cement kilns; a symptom survey in Midlothian, Texas. Toxicology and Industrial Health 14 (6): 829-842.

Liem, A., Hoogerbrugge, R., Kootstra, P., de Jong, A., Marsman, J., den Boer, A., den Hartog, R., Groenemeijer, G. and van't Klooster, H. (1990). Levels and patterns of dioxins in cow's milk in the vicinity of municipal waste incinerators and metal reclamation plants in the Netherlands. Organohalogen Compounds 1: 567-570.

Lloyd O.L., Lloyd M.M., Williams F.L.R. and Lawson A. (1988). Twinning in human populations and in cattle exposed to air pollution from incinerators. British Medical Journal 45: 556-560.

Lorber M., Pinsky P., Gehring P., Braverman C., Winters D. and Sovocool W. (1998). Relationship between dioxins in soil, air, ash and emissions from a municipal solid waste incinerator emitting large amounts of dioxins. Chemosphere 37 (9-12): 2173-2196.

Ma X.F., Babish J.G., Scarlett J.M., Gutenmann W.H. and Lisk D.J. (1992). Mutagens in urine sampled repetitively from municipal refuse incinerator workers and water treatment workers. Journal of Toxicology and Environmental Health 37: 483-494.

MAFF (1992). Third report of studies on dioxins in Derbyshire carried out by the Ministry of Agriculture, Fisheries and Food. Publ: Food Safety Directorate, 31pp.

MAFF (1997a). Dioxins and PCBs in cows' milk from farms close to industrial sites: Rotherham 1997. Food Surveillance Information Sheet Number 133, Publ: Ministry of Agricuture Fisheries and Food, 4pp

MAFF (1997b). Ministry of Agriculture, Fisheries and Food, Food Safety Directorate. Dioxins in cow's milk from farms cl;ose to industrial sites. Food surveillance inforamtion sheet number 100, January 1997, 11pp.

Magagni A., Boschi G. and Schiavon I. (1991). Hospital waste incineration in a MSW combustor: chlorine, metals and dioxin mass balance. Chemosphere 23 (8-10): 1501-1506.

Magagni A., Boschi G., Cocheo V. and Schiavon I. (1994). Fly ash produced by hospital and municipal solid waste incinerators: presence of PAH, PCB and toxic heavy metals. Organohalogen Compounds 20: 397-400.

Malkin R., Brandt-Rauf P., Graziano J. and Parides M. (1992). Blood lead levels in incinerator workers. Environmental Research 59: 265-270.

Mangialardi T., Piga L., Schena G. and Sirini P. (1998). Characteristics of MSW incinerator ash for use in concrete. Environmental Engineering Science 15 (4): 291-297.

Marb C., Hentschel B., Vierle O., Thoma H., Dumler-Gradl R., Swerev M., Schädel S. and Fiedler H. (1997). PCDD/PCDF in bottom ashes from municipal solid waste incierators in Barvaria, Germany. Organohalogen Compounds 32 : 161-166.

Marty M.A. (1993). Hazardous combustion products from municipal waste incineration. Occupational Medicine 8 (3): 603-619.

Mayer J., Rentschler W. and Sczech J. (1999). Long-term monitoring of dioxin emissions of a hazardous waste incinerator during lowered incineration temperature. Organohalogen Compounds 41: 239-242.

McGregor D.B., Partensky C., Wilbourn J. and Rice J.M. (1998). An IARC evaluation of polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans as risk factors in human carcinogenesis. Environmental Health Perspectives 106 (suppl.2): 755-760.

Meeter P., Siebert P.C., Warwick R.O., Canter D.A. and Weston R.F. (1997). Dioxin emissions from soil burning incinerators. Organohalogen Compounds 32: 441-443.

Meneses M., Schuhmacher M., Granero S., Llobet J.M. and Domingo J.L. (1999). The use of Monte Carlo simulation techniques for risk assessment: study of a municipal waste incinerator. Organohalogen Compounds 44: 453-456.

Michelozzi P., Fusco D., Forastiere F., Ancona C., Dellorco V. and Perucci C.A. (1998). Small area study of mortality among people living near multiple sources of air pollution. Occupational and Environmental Medicine 55 (9): 611-615.

Mitchell D.J., Wild S.R. and Jones K.C. (1992). Arrested municipal solid waste incinerator fly ash as a source of heavy metals to the UK environment. Environmental Pollution 76: 79-84.

Miyata H., Aozasa O., Mase Y., Ohta S., Khono S. and Asada S. (1994). Estimated annual emission of PCDDs, PCDFs and non-ortho chlorine substituted coplanar PCBs from flue gas from urban waste incinerators in Japan. Chemosphere 29 (9-11): 2097-2105.

Miyata H., Kuriyama S., Nakao T., Aozasa O. and Ohta S. (1998). Contamination levels of PCDDs, PCDFs and non-ortho coplanar PCBs in blood samples collected from residents in high cancer-causing area close to batch-type municipal soild waste incinerator in Japan. Organohalogen Compounds 38: 143-146.

Mocarelli P., Gerthoux P.M., Ferrai E., Patterson D.G., Keszak S.M., Brambillia P., Vincoli N., Signerini S., Tramacere P., Carreri V., Sampson E.J., Turner W.E. and Needham L.L. (2000). Paternal concentrations of dioxin and sex ratio of offspring. The Lancet 355: 1858-1863.

Moller H. (1996). Change in male:female ratio among newborn infants in Denmark. The Lancet 348: 828-829.

Murray R. (1999). Creating Wealth from Waste, 171pp. ISBN 1 898309 07 8.

National Research Council (2000). Waste Incineration & Public Health. ISBN 0-309-06371-X, Washington, D.C.: National Academy Press.

Newhook, R. & Meek, M.E. (1994). Hexachlorobenzene: evaluation of risks to health from environmental exposure in Canada. Environmental Carcinogenesis and Ecotoxicology Reviews- Journal of Environmental Science and Health, Part C 12(2): 345-360.

Nicolopoulou-Stamati P., Howard C.V. Parkes M. and Hens L. (2000). Introductory Chapter: Awareness of the health impacts of waste management. Proceedings of the seminar "Health Impacts of Waste Management Policies", Hippocrates Foundation, Kos, Greece, 12-14 November 1998. Eds. P. Nicolopoulou-Stamati, L. Hens and C.V. Howard. Kluwer Academic Publishers: pp2-25.

NIOSH (1995). (National Institute for Occupational Safety and Health). 1995. NIOSH Health Hazard Evaluation Report. HETA 90-0329-2482. New York City Department of Sanitation, New York. U.S. Department of Health and Human Services, Public Health Service, Centres for Disease Control and Prevention, National Institute for Occupational Safety and Health. (Cited in NRC 2000).

Nito S. and Takeshita R. (1996). Identification of phenolic compounds in fly ash from municipal waste incineration by gas chromatography and mass spectrometry. Chemosphere 33 (11): 2239-2253.

Nito S. and Ishizaki S. (1997). Identification of azaarenes and other basic compounds in fly ash from municipal waste incinerator by gas chromatography and mass spectrometry. Chemosphere 35 (8): 1755-1772.

Nouwen J., Cornelis C., De Fre R. and Geuzens P. (1999). Health risk assessment of dioxin exposure: The Neerland-Wijk (Wilrijk, Belgium). Organohalogen Compounds 44: 485-487.

Ohta S., Kuriyama S., Nakao T., Aozasa, O. and Miyata H. and Tanahashi M. (1997). 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 32: 155-160

Oppelt E.T. (1990). Air emissions from the incineration of hazardous waste. Toxicology and Industrial Health 6 (5): 23-51.

Osius, N. and Karmaus, W., (1998). Thyroid hormone level in children in the area of a toxic waste incinerator in South Essen. Gesundheitswesen 60:107-112. (Abstract only).

Osius N., Karmaus W., Kruse H., Witten, J. (1999). Exposure to polychlorinated biphenyls and levels of thyroid hormones in children. Environ. Health Persp. 107: 843-849.

Papke O., Ball M. and Lis A. (1993). Potential occupational exposure of municipal waste incinerator workers with PCDD/PCDF. Organohalogen Compounds 9:169-172.

Papke O., Ball M., Menzel H.M., Murzen R., Turcer E. and Bolm-Audorff U. (1994). Occupational exposure of chemical waste incinerators workers to PCDD/PCDF. Organohalogen Compounds 21: 105-110.

Pastorelli G., De Lauretis R., De Stefanis P., Morselli L. and Viviano G. (1999). PCDD/PCDF from municipal solid waste incinerators in Italy: an inventory of air emissions. Organohalogen Compounds 41: 495-498.

Petts J. (1992). Incineration risk perceptions and public concern: experience in the U.K. improving risk communication. Waste Management and Research 10: 169-182.

Pietro P. and Giuliana D.V. (1999). Atmospheric emissions of PCDD/PCDFs from the municipal solid waste incinerator of Fusina (Venice). Orgaqnohalogen Compounds 40: 469-472.

Pilspanen W.H., Czuczwa J.M. and Sobeih I.M. (1992). Work area air monitoring for chlorinated dioxins and furans at a municipal waste power boiler facility. Environmental Science and Technology 26: 1841-1843.

Pluss A. and Ferrell R.E.Jr. (1991). Characterization of lead and other heavy metals in fly ash from municipal waste incinerators. Hazardous Waste and Hazardous Waste Materials 8 (4): 275-292.

QUARG (1996). Airborne Particulate Matter in the United Kingdom. Third Report of the Quality of Urban Air Review Group (QUARG), May. ISBN 0 9520771 3 2.

Ramos L., Eljarrat E., Hernandez L.M., Alonso L., Rivera J., and Gonzalez M.J. (1997). Levels of PCDDs and PCDFs in farm cow's milk located near potential contaminant sources in Asturias (Spain). Comparison with levels found in control, rural farms and commercial pasteurized cow's milks. Chemosphere 35 (10): 2167-2179.

Rapiti E., Sperati A., Fano V., Dell'Orco V. and Forastiere F. (1997). Mortality among workers at municipal waste incinerators in Rome: a retrospective cohort study. American Journal of Industrial Medicine 31: 659-661.

Roffman A. and Roffman H.K. (1991). Air emissions from municipal waste combustion and their environmental effects. The Science of the Total Environment 104: 87-96.

Rowat S.C. (1999). Incinerator toxic emissions: a brief summary of human health effects with a note on regulatory control. Medical Hypotheses 52 (5): 389-396.

Ruokojärvi P., Ruuskanen J., Ettala M., Rahkonen P and Tarhanen J. (1995). Formation of polyaromatic hydrocarbons and polychlorinated organic compounds in municipal waste landfill fires. Chemosphere 31 (8): 3899-3908.

Rydhstroem H. (1998). No obvious spatial clustering of twin births in Sweden between 1973 and 1990. Environmental Research, 76: 27-31.

Sakai S., Hiraoka M., Takeda N. and Shiozaki K. (1996). Behaviour of coplanar PCBs and PCNs in oxidative conditions of municipal waste incineration. Chemosphere 32 (1): 79-88.

Sandalls, F.J., Berryman, R.J., Bennett, S.L. & Ambidge, P.F. (1997). Investigations into the emissions of dioxins and furans from the Coalite works, near Bolsover, Derbyshire. Publ: UK Environment Agency, report no.HO-9/97-500-C-AZMK, 21pp.

Sawell S.E., Chandler A.J., Eighmy T.T., Hartlen J., Hjelmar O, Kosson D., Van der Sloot H.A. and Vehlow J. (1995). An international perspective on the characteristisation and management of residues from MSW incinerators. Biomass and Bioenergy 9 (1-5): 377-386.

Scarlett J.M., Babish J.G., Blue J.T., Voekler W.E. and Lisk D.J. (1990). Urinary mutagens in municipal refuse incinerator workers and water treatment workers. J. Toxicol. Environ. Health 31: 11-27. (Cited in Ma *et al.* 1992).

Schecter A. (1994). Exposure Assessment: Measurement of dioxins and related chemicals in human tissues. In: Dioxins and Health. (pp449-477). Plenum Press, New York, ISBN 0-306-44785-1.

Schecter A. Miyata H., Ohta S., Aozasa O., Nakao T. and Masuda Y. (1999). Chloracne and elevated dioxin and dibenzofuran levels in the blood of two Japanese MSW incinerator workers and of the wife of one worker. Organohalogen Compounds 44: 247-250.

Schecter A.J., Malkin R., Papke O., Ball M. and Brandt-Rauf P.W. (1991). Dioxin levels in blood of municipal incinerator workers. Med Sci Res. 19: 331-332.

Schmid P. and Schlatter Ch. (1992). Polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) in cow's milk from Switzerland. Chemosphere 24 (8): 1013-1030.

Schneider M., Stieglitz L., Will R. and Zwick G. (1998). Formation of polychlorinated napthalenes on fly ash. Chemosphere 37 (9-12): 2055-2070.

Schreiber R.J. and Evans J.J. (1994). Dioxin emission results from recent testing at cement kilns. Organohalogen Compounds 20: 373-376.

Schuhmacher M., Domingo J.L., Granero S., Llobet J.M., Eljarrat E. and Rivera J. (1999a). Soil monitoring in the vicinity of a municipal solid waste incinerator: Temporal variation of PCDD/Fs. Chemosphere 39 (3): 419-429.

Schuhmacher M., Domingo J.L., Llobet J.M., Muller L. and Jager J. (1997a). Levels of PCDDs and PCDFs in grasses and weeds collected near a municipal solid waste incinerator. The Science of the Total Environment 201: 53-62.

Schuhmacher M., Granero S., Domingo J.L., Llobet J.M. and de Kok H.A.M. (1999b). Monitoring PCDD/Fs in the vicinity of an old municipal waste incinerator, 1996-1998. Part II: Vegetation monitoring. Organohalogen Compounds 43: 123-126.

Schuhmacher M., Granero S., Xifro A., Domingo J.L., Rivera J. and Eljarrat E. (1998). Levels of PCDD/Fs in soil samples in the vicinity of a municipal solid waste incinerator. Chemosphere 37 (9-12): 2127-2137.

Schuhmacher M., Meneses M., Granero S., Llobet J.M. and Domingo J.L. (1997b). Trace element pollution of soils collected near a municipal solid waste incinerator: human health risk. Bull. Environ. Contam. Toxicol. 59: 861-867.

Paul. Minnesota. Archives of Environmental Health 49 (5): 366-374.Schwartz J. and Marcus A. (1990). Mortality and air pollution in London: a time series analysis. American Journal of Epidemiology 131 (1): 185-194.

Schwartz J., Slater D., Larson T.V., Pierson W.E. and Koenig J. (1993). Particulate air pollution and hospital emergency room visits for asthma in Seattle. American Review of Respiratory Diseases 147: 826-831.

Schwind, K._H., Hosseinpour, J., (1988) Brominated/chlorinated dibenzo-p-dioxins and dibenzofurans. Part 1: Brominated/chlorinated and brominated dibenzo-p-dioxins and dibenzofurans in fly ash from a municipal waste incinerator. Chemosphere 17 (9): 1875-1884.

Seaton A. (1995). Particulate air pollution and acute health effects. The Lancet 345: 176-178.

Shane B.S., Gutenmann W.H. and Lisk D.J. (1993). Variability over time in the mutagenicity of ashes from municipal solid-waste incinerators. Mutation Research 301: 39-43.

Shin D., Yang W., Choi J., Choi S. and Jang Y.S. (1998). The effects of operation conditions on PCDD/Fs emission in municipal solid waste incinerators: stack gas measurement and evaluation of operating conditions. Organohalogen Compounds 36: 143-146.

Shy C.M., Degnan D., Fox D.L., Mukerjee S., Hazucha M.J., Boehlecke B.A., Rothenbacher D., Briggs P.M., Devlin R.B., Wallace D.D., Stevens R.K. and Bromberg P.A. (1995). Do waste incinerators induce adverse respiratory effects? An air quality and epidemiology study of six communities. Environmental Health Perspectives 103: 714-724.

Sinkkonen S., Paasivirta J., Koistinen J. and Tarhanen J. (1991). Tetra- and pentachlorodibenzothiophenes are formed in waste combustion. Chemosphere 23 (5): 583-587.

Sprague J.B. (1991). Environmentally desirable approaches for regulating effluents from pulp mills. Wat. Sci. Techno. 24: 361-371.

Stairs K.C. and Johnston P. (1991). The precautionary action approach to environmental protection. Environ. Poll 1 - ICEP.1: 473-479.

Startin J.R., Wright C., Kelly M. and Charlesworth E.A. (1994). Dioxin concentrations in the blood of individuals resident on farms near Bolsover, UK. Organohalogen Compounds 21: 117-120.

Steenwegen C. (2000). Can Ecological taxes play a role in diminishing the health impacts of waste management? In: Health Impacts of Waste Management Policies. Proceedings of the seminar "Health Impacts of Waste Management Policies", Hippocrates Foundation, Kos, Greece, 12-14 November 1998. Eds. P. Nicolopoulou-Stamati, L.Hens and C.V. Howard. Kluwer Academic Publishers.

Stieglitz L., Hell K., Matthys K., Rivet F. and Buekens A. (1999). Dioxin studies on a MSW-incinerator. Organohalogen Compounds 41: 117-120.

Sunyer J., Saez M., Murillo C., Castellsague J., Martinez F., Anto J.M. (1993). Air pollution and emergency room admissions for chronic obstructive pulmonary disease: A 5-year study. American Journal of Epidemiology 137 (7): 701-705.

Swedish EPA (1998). Persistent Organic Pollutants: A Swedish Way of an International Problem. ISBN 91-620-1189-8.

Takasuga, T., Inoue, T., Ohi, E. & Ireland, P. (1994) Development of an all congener specific, HRGC/HRMS analytical method for polychlorinated naphthalenes in environmental samples. Organohalogen Compounds 19: 177-182

ten Tusscher G.W., Stam G.A. and Koppe J.G. (2000). Open chemical combustions resulting in a local increased incidence of orofacial clefts. Chemosphere 40: 1263-1270.

Thompson L.J., Ebel J.G.Jr., Manzell K.L., Rutzke M., Gutenmann W.H. and Lisk D.J. (1995). Analytical survey of elements in veterinary college incinerator ashes. Chemosphere 30 (4): 807-811.

UK Environment Agency (1997). Report on the operation of incineration plant at the Coalite Chemical Works, Bolsover, Derbyshire, from commissioning to closure and the subsequent prosecution of the last operator Coalite Products Ltd by H.M. Inspectorate of Pollution under section 5 of the Health and Safety at work act 1974. Publ: UK Environment Agency, report no. HO-9/97-500-C-AZMI, 71pp.

USEPA (2000). Exposure and Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and related compounds. Part 1, Volume 3, Chapter 3, p27. EPA/600/P-00/001 Ab-Ae, March/May 2000.

USEPA (1997) Estimating Air Emissions from Sources of Dioxins and Furans. Publ. United States Environmental Protection Agency Office of Air Quality Planning and Standards, Office of Air And Radiation, 1997 Report Number EPA-454/R-97-003, Research Triangle Park, North Carolina.

Valerio F., Pala M., Piccardo M.T., Lazzarotto A., Balducci D and Brescianini C. (1995). Exposure to airborne cadmium in some Italian urban areas. The Science of the Total Environment 172: 57-63.

Van Birgelen, A.P.J.M. (1998). Hexachlorobenzene as a possible major contributor to the dioxin activity of human milk. Environmental Health Perspectives 106(11): 683-688.

Van den Hazel P. and Frankort P. (1996). Dioxin concentrations in the blood of residents and workers at a municipal waste incinerator. Organohalogen Compounds 30: 119-121.

Van Doorn R., Leijdekkers CH-M., Bos R.P., Brouns R.M.E. and Henderson P. TH. (1981). Enhanced excretion of thioethers in urine of operators of chemical waste incinerators. British Journal of Industrial Medicine 38: 187-190.

Van Larebeke N. (2000). Health effects of a household waste incinerator near Wilrijk, Belgium. In: Health Impacts of Waste Management Policies. Proceedings of the seminar "Health Impacts of Waste Management Policies", Hippocrates Foundation, Kos, Greece, 12-14 November 1998. Eds. P. Nicolopoulou-Stamati, L.Hens and C.V. Howard. Kluwer Academic Publishers.

Van Velzen D. and Langenkamp H. (1996). Antimony (Sb) in urban and industrial waste and in waste incineration. European Commission EUR 16435 EN.

Verschaeve L. and Schoeters G. (1998). Cytogenetisch populatieoderzoek: commentaren bij het cytogenetisch onderzoek van kinderen in de Neerlandwijk, VITO report 1998/R/TOX/045, Mol, Belgium. (in Dutch). (Cited in van Larebeke 2000).

Viel J.-F., Arveux P., Baverel J. and Cahn J.-Y., 2000. Soft-tissue sarcoma and non-Hodgkin's lymphoma clusters around a municipal solid waste incinerator with high dioxin emission levels. Am. J. Epidem. 152:13-19.

Villalobos S.A., Anderson M.J., Denison M.S., Hinton D.E., Tullis K., Kennedy I. M., Jones A.D., Chang D.P.Y., Yang G. and Kelly P. (1996). Dioxinlike properties of a trichloroethylene combustion-generated aerosol.

Wang J., Hsiue T., and Chen H. (1992). Bronchial responsiveness in an area of air pollution resulting from wire reclamation. Arch. Dis. Child. 67:488-490. (Cited in National Research Council 2000).

Weber, L.W.D. and Greim, H. (1997) The toxicity of brominated and mixed-halogenated dibenzo-p-dioxins and dibenzofurans: An overview. Journal of Toxicology and Environmental Health. 50: 195-215

Webster T. and Connet P. (1990). Risk Assessment: A public health hazard? Journal of Pesticide Reform 10 (1): 26-31.

Webster T. and Connett P. (1998). Dioxin emission inventories and trends: the importance of large point sources. Chemosphere 37 (9-12): 2105-2118.

Wikstrom E. (1999). The role of chlorine during waste combustion. Department of chemistry, Environmental Chemistry, Umea University.).

Wikstrom E. Persson A. and Marklund S. (1998). Secondary formation of PCDDs, PCDFs, PCBs, PCBzs, PCPhs and PAHs during MSW combustion. Organohalogen Compounds 36: 65-68.

Wilken M., Boske J., Jager J. and Zeschmar-Lahl B. (1993) PCDD/F, PCB, chlorbenzene and chlorophenol emissions of a municipal solid waste incinerator plant (MSWI) – variation within a five day routine performance and influence of Mg(OH)2-addition. Organochlogen Compounds :241

Williams F.L.R., Lawson A.B. and Lloyd O.L. (1992). Low sex ratios of births in areas at risk from air pollution from incinerators, as shown by geographical analysis and 3-dimensional mapping. International Journal of Epidemiology 21 (2): 311-319.

Williams P.T. (1990). A review of pollution from waste incineration. Journal of the Institute of Water and Environmental Management 4 (1):2634.

World Bank (1999). The International Bank for Reconstruction and Development/THE WORLD BANK. "What a Waste: Solid Waste Management in Asia," Urban Development Sector Unit, East Asia and Pacific Region, Washington, D.C., June 1999.

Wrbitzky R., Goen T., Letzel S. and Frank F. (1995). Internal exposure of waste incineration workers to organic and inorganic substances. Int Arch Occup Environ Health 68: 13-21.

Yamamura K., Ikeguchi T. and Uehara H. (1999). Study on the emissions of dioxins from various industrial waste incinerators. Organohalogen Compounds 41: 287-292.

Yasuda K. and Takahashi M. (1998). The emission of polycyclic aromatic hydrocarbons from municipal solid waste incinerators during the combustion cycle. Journal of Air and Waste Management. 48: 441-447.

Zanini E., and Bonifacio E. (1991). Lead pollution of soils from a continuous point source: A case study in Italy. J.Environ. Sci. Health A26 (5): 777-796.

Zmirou D., Parent B., Potelon J-L. (1984). Etude epidemiologique des effets sur la sante des rejets atmospheriques d''ne usine d''ncineration de dechets industriels et menagers. Rev. Epidem. et Sante Publ. 32: 391-397. (in French). (Cited in Hens *et al.* 2000, Rowat 1999, Marty 1993).

8. APPENDICES

APPENDIX A

HEALTH EFFECTS OF SPECIFIC POLLUTANTS RELEASED FROM INCINERATORS

1 Particulate Matter

1.1 Introduction

Animal and plant life as we know it evolved in the presence of particulate matter (Howard 2000). This matter consists of minute particles, sometimes called particulates. Natural sources of particulates include soil particles which are blown into the air by the wind, dusts from volcanic eruptions, particles of sea salt ejected into the air by breaking waves on the sea, spores from fungi and pollen grains from plants. These particles vary in size from those coarse-sized particles that are visible to the naked eye to tiny microscopic particles, measured in micrometers (μm). The finer particles tend to remain airborne for long periods of time whereas coarse particles, such as wind blown soil, and plant pollens, tend to fall rapidly and remain airborne for only short periods of time. Most naturally produced particles are generally greater than 20 μm in size (QUARG 1996, COMEAP 1995, EPAQS 1995).

The human respiratory system has evolved to cope with an environmental loading of such naturally produced aerial particles. The muco-cilary lining (mucus and fine hairs) of the airways operates to protect the deeper regions of the lung. Fine particles of less than 10 μm do occur naturally, mainly in the form of re-suspended sea salts. In this form, the fine particulates pose no threat to health and, if inhaled, are simply absorbed into the body (Howard 2000).

Particulates are also formed as a consequence of human activities. When humans started to use fire domestically, exposure to fine insoluble particles of material, less than 10 μm in size, must have occurred on a regular basis (Howard 2000). However, the advent of industrialized society has seen a vast increase in the level of industrial combustion processes around the world.

The major source of man-made particles arises from combustion processes such as incineration of wastes, coal-burning and vehicle exhausts and to a lesser extent from metallurgical and other industrial processes. All of these processes inevitably result in the direct emission of particles into the atmosphere. Such particles which are directly emitted into the atmosphere are called primary particles. Furthermore, as well as this direct emission of particles from combustion processes, some pollutant gases which are released from combustion processes, such as sulphur dioxide and nitrogen dioxide, undergo chemical reactions in the atmosphere in which particles (known as secondary particles) are also formed. These particles are mainly comprised of ammonium sulphate and ammonium nitrate. Secondary particles can also have a wide variety of other toxic organic compounds adsorbed onto their surfaces such as PAHs (QUARG 1996, COMEAP 1995, EPAQS 1995).

Particulates formed from human activities are generally fine particles (less than 10 μm) and even smaller ultrafine particles (less than 0.05 μm i.e. 50 nanometers (nm) in size). There is much scientific evidence which indicates that that particles less than 10 μm, have adverse effects upon the human health. Most notably, the most recent views and data suggest that it is the number of ultrafine particles, and possibly their chemical composition, which causes health impacts (QUARG 1996, Seaton *et al.* 1995). Ultrafine particles may be acidic in nature and an irritant to the lungs or carry toxic substances on their surface such as halogenated organic materials, including dioxins, metals and PAHs (COMEAP 1995).

The respiratory system has mechanisms to expel particulates to protect the deep lung regions. Naturally produced particles are generally of sizes 2.5 to 10 μm or larger. All particles below 10 μm may reach the furthest parts of the lungs but, in general, those particles sized between 2.5 and 10 μm are most likely to be deposited in the upper airways of the lungs. From here they are efficiently removed. Only a small fraction of naturally produced particles will be deposited in the deepest regions of the lungs. The body does have mechanisms which clear particles from the deep parts of the lungs, although they are less efficient than in the upper airways. However, unlike natural particles, a high proportion of particulates from industrial combustion processes are less than 2.5 μm in size. These "respirable" particles can reach and be deposited in the deep regions of the lungs and are thought to have the most significant impacts on health.

While it is recognized that there would have been naturally and man-made combustion processes in the pre-industrial age, the level of particulate production would have been very small by comparison with today. Recent studies in the UK, estimated that primary and secondary particles derived from industry each constitute about a third of the total PM10 burden in air, with the remaining third coming from marine aerosol (see Howard 2000). Differences in the levels of particulates in air are clearly influenced by local sources. For instance, a study in the city of Birmingham, UK, showed that the number of particles present in urban air ranged from 1000 to 100,000 particles per cm^3. Rural air contained lower numbers of particles, averaging 5000 to 10,000 per cm^3 but rose when influenced by nearby traffic. Less polluted air such as that over the North Atlantic Ocean typically contains around 200 particles per cm^3 (QUARG 1996, Seaton 1995).

Since the size of particles has a direct bearing on health impacts, measurements made in recent years have quantified particles in the atmosphere according to size. The most commonly used measurement is known as PM10, which estimates the mass of particles in air which are less

than 10 um in diameter. PM10 reflects the size of particles which are considered to be most likely deposited in the lung. It has been used to quantify particulate concentrations in many studies on health impacts of particulate air pollution.

1.2 Health Effects of Particulates

From the 1930s to the 1950s there were a series of severe air pollution disasters in industrialized cities including London, Meuse Valley Belgium and Donora, Pennsylvania. Dense winter smogs, caused by particulate pollution and sulphur dioxide from coal combustion, resulted in substantial increases in the death rate from respiratory and cardiovascular diseases. These incidents proved that high levels of particulate air pollution in smog caused an increase in daily death rate (see Schwartz 1994a). Such incidents became a thing of the past by the 1960s in western industrialized countries, due to changes in the types of fuel and better pollution control. The nature of air pollution also changed as a result of increases in vehicle emissions. Since the mid-1980s, much research has been carried out to determine whether the lower levels of particulates and other air pollutants to which populations are exposed nowadays also cause an increase in death rate and disease. It has been shown by many human epidemiology studies on this subject that particulate pollution is indeed linked to worsening respiratory diseases and increasing premature mortality from respiratory and heart diseases (see Pope *et al.* 1995a).

Studies on short-term fluctuations in the level of air pollution in a region and the daily death rate of the region have consistently found that small elevations in the daily death rates are associated with short-term increases in particulate air pollution (e.g. see Pope *et al.* 1995a, Schwartz 1994a). Importantly, these studies have not revealed a "safe level" of particulates, that is a threshold, below which the death rate did not increase. Thus, the association between increasing mortality with increasing particulate pollution was apparent in studies even in areas where air pollution was relatively low and was well within US air quality standards. It was estimated from the study data that for every 10 mg/m^3 increase in PM10 there was an associated 0.5-1.5% increase in daily mortality. The premature deaths were due mainly to an increase in deaths from respiratory diseases and secondarily from cardiovascular diseases. Researchers have reviewed the studies and concluded that it is unlikely the observed increased death rates were due to confounding factors that could also affect the death rate such as weather conditions or other air pollutants. It has therefore been suggested that it is reasonable to interpret results from the studies as showing a causal relationship between PM10 and daily mortality (e.g. Pope *et al.* 1995a, Schwartz 1994a).

In addition to research on short-term changes in particulate pollution and mortality rates, studies have also been conducted on the effect of exposure to long-term air pollution on mortality. These studies compared the annual average mortality rates of populations living in different areas, with the annual average concentrations of air pollution in those areas. They again revealed an association between particulate pollution and increased mortality. Death rates were found to be higher in cities with higher levels of fine or sulphate particulate pollution than those with lower levels. In fact, results showed that between 3 and 9% of all urban deaths appeared to be due to particulate air pollution (Pope *et al.* 1995b). Since this figure is so high, these studies have come under criticism for inadequate control of confounding factors. However, two further studies which overcame the problem of confounding (Pope *et al.* 1995c, Dockery *et al.* 1993), including one study on a 8-year follow up of over half a million US adults (Pope *et al.* 1995c), also found that average long-term levels of fine particulate and sulphate air particulate pollution were linked with mortality. The risk of mortality was about 15-25% higher in cities with the highest particulate pollution compared to cities with the lowest levels. The causes of death associated with particulate pollution were respiratory diseases, including lung cancer, and cardiovascular diseases, particularly amongst the chronically ill or elderly. Therefore, studies on both short-term changes in air pollution, and long-term levels of air pollution have indicated an association between mortality and particulate air pollution.

If the relationship between particulate pollution and mortality is causal, then it would be expected that morbidity (ill-health) would also be associated with particulate pollution levels (Pope *et al.* 1995b). This is indeed the case. Many studies have been conducted using a variety of endpoints to monitor health effects and have found an association between short-term changes in particulate air pollution and illness. The results are illustrated in the list of bullet points given below. Overall, these studies suggest that particulate pollution is able to cause temporary worsening of already existing respiratory illnesses.

Particulate air pollution has been associated with:

- Increase in hospital admissions for respiratory illness in winter (e.g. Schwartz 1994a, Pope 1991, Pope 1989) and in summer (e.g. Schwartz 1994a b, c and d, Thurston *et al.* 1993).
- Increase in hospital emergency department visits for respiratory diseases (see Pope 1995a, e.g. Schwartz *et al.* 1993, Sunyer *et al.* 1993).
- Exacerbation of mild and severe asthma attacks in adults and children (e.g. Pope *et al.* 1995b, Walters *et al.* 1994, Ostro 1993, Roemer *et al.* 1993,

Thurston *et al.* 1993, Pope 1989, Bates and Sizto 1987, Whittemore and Korn 1980).

- Increase in respiratory symptoms (particularly lower respiratory symptoms, such as wheezing, dry cough, phlegm, shortness of breath, chest discomfort/pain), (e.g. Sram *et al.* 1996, Schwartz *et al.* 1994b, Braun-Fahrlander *et al.* 1992, Pope and Dockery 1992).
- Small decreases in lung function (e.g. Hoek and Brunekreef 1994 and 1993, Koenig *et al.* 1993, Neas et al. 1992, Pope and Dockery 1992, Pope *et al.* 1991, Dassen *et al.* 1986, Dockery *et al.* 1982).
- Work and school absenteeism through ill health (Sram *et al.* 1996, Ransom and Pope 1991, Ostro 1990, Ostro and Rothschild 1989).

Results of epidemiology studies discussed in this section provide evidence that day-to-day variations in current air particulate concentrations are associated with transient effects on health and increases in the daily death rate. This has provoked a great deal of controversy. Critics have argued that results were artefactual and contrary to common sense and established doctrine. However, a number of independent experts have also reviewed the evidence and have suggested that the associations are causal. This view has been accepted by the UK Department of Health (Maynard 2000).

In addition to transient health effects due to short-term changes in particulate air pollution, research has also been conducted to investigate whether long-term exposure to particulate pollution over one year or more has any long-lasting or cumulative effects on health. Several studies have showed an association. For example, some studies found an association with reduced lung function (e.g. Sram *et al.* 1996, Chestnut *et al.* 1991, Schwartz 1989) and increased respiratory symptoms especially bronchitis (Abbey et al. 1995, see Pope *et al.* 1995b).

In sum, epidemiology data indicates that the link between fine particulate pollution and adverse effects on health is most likely causal (see e.g. Pope 1995b, COMEAP 1995). Less is known about the mechanisms by which particulates impact on health. Presently, it is thought that ultrafine, possibly acidic particles could cause inflammation of tissue in the lung extremities which in turn may provoke respiratory and heart related illnesses and death (e.g. Seaton 1995, see Howard 2000 and 2000b). Research suggests that it may be the smallness of ultrafine particles themselves, their high surface area for the release of transition metals, their insolubility and their possible generation of free radicals which may be the most important factors contributing to make the particle cause inflammation (Donaldson *et al.* 2000).

2. Dioxins

Dioxins are toxic, persistent in the environment and they are bioaccumulative, i.e., they build up in the tissues of animals and humans alike. Dioxins are the unintented by-products of human activities that involve the manufacture and use of elemental chlorine and the combustion of materials that contain any form of chlorine. Dioxin levels in the populations and environments of industrialised regions began increasing markedly following World War II, with the manufacture and dispersal of chlorinated pesticides and other chemicals and their associated wastes.

By the late 1970s and early 1980s, some scientists were beginning to recognize that dioxins were ubiquitous in the populations and environments of industrialised regions. Moreover, they discovered that waste incinerators were releasing dioxins in their stack gases and ashes. By this time, however, the technology was well entrenched. Once policymakers were finally convinced of the widespread nature of the dioxin problem and its link to incineration, some regions had come to depend on incineration, both as a waste disposal option as well as an economic enterprise for the industrial sector. As a consequence, these regions have become even more heavily invested in incinerator control technologies. Unfortunately, these methods primarily change the nature of the dioxin problem but do not solve it. i.e., a larger share of the dioxins that were once released into the air are captured in stack filters or in bottom ashes and placed in landfills. This will retard the dispersal of dioxins into the environment but will not, in the long-term, prevent it.

Dioxin contamination is not restricted to the local areas surrounding incinerators and other sources. Like other persistent organic pollutants, they are transported for thousands of kilometres on air currents and have consequently become globally ubiquitous pollutants. It is thought that every man, woman and child on the planet now carries dioxins in their body tissues.

The toxicology of dioxins, in particular TCDD, has been extensively researched. A considerable amount of work has also been put into conducting research on wildlife and human health effects of dioxin exposure. TCDD was recently classified as a human carcinogen by the International Agency for Research on Cancer (McGregor *et al.* 1998).

Dioxins exert a plethora of toxic effects because they act on a fundamental biochemical regulation system in the body, a system that is common to animals and humans. Dioxins exert their effects through binding to the "Ah receptor", the outcome of which affects several genes (Webster and Commoner 1994). Their toxicological effects are summarised in table 1.

Table 1 Toxicological effects of dioxin

Carcinogenesis	IARC class 1 carcinogen (carcinogenic to humans).
Immune system effects	Suppression of cell-mediated and humoral immunity; increased susceptibility to infectious challenge; auto-immune response.
Male reproductive toxicity	Reduced sperm count; testicular atrophy; abnormal testis structure; reduced size of genital organs; feminized hormonal responses; feminized behavioural responses.
Female reproductive toxicity	Decreased fertility; inability to maintain pregnancy; ovarian dysfunction; endometriosis.
Developmental impacts	Birth defects; foetal death; impaired neurological development and subsequent cognitive deficits; altered sexual development.
Modulation of hormones, receptors, and growth factors	Steroid hormones and receptors (androgens, estrogens and glucocorticoids); thyroid hormones; insulin; melatonin; vitamin A; EGF and receptor; TGF-a and TGF-b; TNF-a, IL-1b, c-Ras, c-ErbA.
Other effects	Organ toxicity (liver, spleen, thymus, skin); diabetes; weight loss; wasting syndrome; altered fat and glucose metabolism.

Source: Aapted from USEPA 1994 and Birnbaum 1994

Occupational studies have reported that exposure to dioxin has been associated with a wide range of effects including chloracne (skin lesions), changes in the levels of liver enzymes, changes in the levels of thyroid hormones, sex hormones, and cells of the immune system (reviewed by Sweeney and Mocarelli 2000). Exposure to dioxins in the workplace has also been associated with an increased risk of cancer when all cancers together are considered (eg. Fingerhut *et al.* 1991, Manz *et al.* 1991, Zorber *et al.* 1990).

For the general population of industrialised countries, research indicates that dioxins are exerting effects on people at current background levels found in the environment. For instance, dioxins can affect the levels of certain hormones, enzymes and immune system cells, at body concentrations at, or near to, the levels currently found in the human populations of industrialised countries. According to DeVito *et al.* 1995:

"Subtle changes in enzyme activity indicating liver changes in levels of circulating reproductive hormones in males, in reduced glucose tolerance potentially indicative of risk of diabetes, and in cellular changes related to immune function suggest the potential for adverse impacts on human metabolism, reproductive biology, and immune competence at or within one order of magnitude of average background body burden levels… Individuals at the high end of the general population range may be experiencing some of these effects. Some more highly exposed members of the population may be at risk for frankly adverse effects including developmental toxicity, reduced reproductive capacity based on decreased sperm counts and potential for increased fetal death, higher probability of experiencing endometriosis, reduced ability to withstand an immunological challenge and others."

Intake of dioxins in the diet of populations in Europe is frequently in excess of the tolerable daily intake (TDI) set by WHO (1-4 pg ITEQ/kg/day), particularly when dioxin-like PCBs are included (WHO 1998, see Allsopp *et al.* 2000). WHO experts acknowledged that subtle effects on health may already be occurring in the general population and that efforts should be made to ensure that intakes are at the lower end of the TDI range. Intake by breast-fed infants is very high compared with the TDI. This is of particular concern because the developing stages of life are most vulnerable to toxic insult from such chemicals.

Studies carried out during the past decade in the Netherlands have investigated the impacts of current background levels of dioxins and PCBs on foetal development, during infancy and childhood. Healthy women from the general population were selected for these studies. Results have revealed undesirable impacts on health of the immune system and nervous system during development that are associated with exposure to dioxins and PCBs during development.

For instance, some of the women had higher levels of PCBs and dioxins in their bodies and breast milk than others. It

was found that infants who were exposed to higher levels of these chemicals in the womb, and via breastfeeding, had changes in the number of certain immune system cells. It is not known what effect such changes could have on their health (Weisglas-Kuperus *et al.* 1995). Regarding effects on the nervous system, there was a slight adverse effect on psychomotor development in infants who were exposed to higher levels of PCBs and dioxins in the womb and during breastfeeding (Koopman-Esseboom *et al.* 1995). There was also a slight adverse effect on neurological development in the children detected at 18 months of age. Tests on neurological development looked at movement co-ordination, (eg. sitting, crawling, standing and walking), and are a way of measuring the quality and integrity of brain function (Huisman *et al.* 1995). At the age of 2 years and 7 months, tests on a subgroup of infants found slight changes in some measures of neurological development in the more highly exposed individuals. These changes were regarded as "unwanted" by researchers, and it was proposed that they could be due to the action of dioxins on thyroid hormones during development (Ilsen *et al.* 1996). A summary of the effects of perinatal exposure to dioxins and PCBs is presented in Table 2.

Table 2: Effects of Perinatal Exposure to Dioxins and PCBs on Infants and Children

Central nervous system	Delayed cognitive development, mildly disordered behavior, and increased activity in children of mothers who were accidentally exposed to extraordinary levels of dioxins/PCBs.
	Deficits in autonomic maturity and reflexes, less preference for a novel stimulus, and defects in short term memory in children whose mothers were exposed to background levels of PCBs and dioxins.
	Delayed motor development, hypotonia and hyporeflexia in children exposed to background levels.
	Increased hypotonia, lower psychomotor developmental indices, less optimal neurological condition, and lower cognitive scores in children whose mothers were exposed to background levels.
Immune system	More frequent occurrence of bronchitis, upper respiratory infections and ear infections among children whose mothers had extraordinary prenatal exposure.
	More frequent ear infections and altered levels of certain cells that have roles in warding off diseases in Inuit infants whose mothers had elevated exposure through their diet of traditional foods.
	Altered levels of certain cells involved in resisting diseases among children whose mothers had background exposures.
Growth, sexual development and reproductive health	Fewer boy children were born to couples in which both parents had high dioxin exposures during the seven year period following a large dioxin release at a chemical manufacturing facility.
	Lower weight at birth and continued diminished height and weight at school age among children whose mothers had extraordinaryl exposure.
	Reduced penis length among boys who were conceived in the earliest years after their mothers had extraordinary exposure.
	Altered birthweight and gestational age among infants of mothers who had occupational exposure to PCBs.
	Lower birthweight and smaller head circumference among infants of mothers whose diets included fish from the Great Lakes.

Growth, sexual development and reproductive health	Lower birthweight and slower postnatal growth until 3 months of age among infants whose mothers had background exposure.
Thyroid function	Subtle alterations in levels of thyroid hormones in pregnant mothers and their infants exposed to background levels of PCBs and dioxins.

3. Heavy Metals

3.1 Lead

Lead has no known nutritional biochemical or physiological function (Goyer 1996). The toxic effects of lead are the same, irrespective of whether it is ingested or inhaled, and blood levels less than10-100 mg/dl in children, and 10-100 mg/dl in adults have been associated with a wide range of adverse effects. These include nervous system disorders, anaemia and decreased haemoglobin synthesis, cardiovascular disease, and disorders in bone metabolism, renal function and reproduction. Of particular concern, is the effect of relatively low exposure on cognitive and behavioural development in children (Pirkle *et al.* 1998, USPHS 1997, Bernard *et al*. 1995, Goyer 1993, Nriagu 1988). It is clear that increased body burden of lead results in decreased scores on measures of intelligence from early infancy through school age. It also results in effects on behaviour of school children, including increased distractibility, short attention span and impulsivity (Rice 1996).

In 1975 the Centre for Disease control (CDC) in Atlanta recommended that the maximum permissible level of blood-lead be 30 ug/dl (for both adults and children). This levels was revised downward in 1985 to 25 ug/dl, and again in 1991, defining a blood-lead of 10 ug/l as an action or intervention level (USPHS 1997). Perhaps even more importantly is the now suggested recommendation that there may be no level of blood-lead that does not produce a toxic effect, particularly in the developing central nervous system (USPHS 1997, Goyer 1993).

3.2 Cadmium

Cadmium has no biochemical or nutritional function, and it is highly toxic to both plants and animals (USPHS 1997, WHO 1992, Alloway 1990). In humans and animals, there is strong evidence that the kidney is the main target organ of cadmium toxicity, following extended exposure (USPHS 1997, Elinder and Jarup 1996, Goyer 1996, Roels *et al.* 1993, Iwata *et al.* 1993, WHO 1992, Mueller *et al.* 1992). Renal damage includes tubular proteinuria (the excretion of low molecular weight proteins) and a decrease in the glomerular filtration rate. The latter results in a depressed re-sorption of enzymes, amino acids, glucose, calcium, copper, and inorganic phosphate. Furthermore, studies have shown that even when cadmium exposure ceases, proteinuria does not decrease, and renal tubular dysfunction and reduced glomerular filtration increase in severity (USPHS 1997, Elinder and Jarup 1996, Goyer 1996, Iwata *et al.* 1993, WHO 1992, Nriagu 1988).

Other toxic effects of cadmium, based on findings from occupation, animal, and epidemiological studies, can be summarised as follows:

Case studies indicate that calcium deficiency, osteoporosis, or osteomalacia (softening of the bones) can develop in some workers after long-term occupational exposure to high levels of cadmium. A progressive disturbance in the renal metabolism of vitamin D and an increased urinary excretion of calcium is often seen, suggesting that bone changes may be secondary to disruption in kidney vitamin D and calcium metabolism (USPHS 1997, Goyer *et al.* 1994, WHO 1992). In the Jinzu River Basin, a cadmium-contaminated area in Japan, a cadmium induced skeletal disorder known as Itai-Itai disease disabled many children born to women of middle age and poor nutrition (Alloway 1996).

The inhalation of high levels of cadmium oxide fumes or dust is intensely irritating to respiratory tissue, and acute high-level exposures can be fatal. Typical non-fatal symptoms can include severe tracheobronchitis, pneumonitis, and pulmonary oedema, which can develop within hours of exposure (USPHS 1997b, Goyer 1996, WHO 1992). At lower levels, lung inflammation have been known to cause emphysema (swelling of the lung air sacs resulting in breathlessness) and dyspnoea (difficult and laboured breathing) (USPHS 1997, Goyer 1996, WHO 1992). Animal studies have confirmed that inhalation exposure to cadmium leads to respiratory injury (USPHS 1997b, WHO 1992).

There have been a number of epidemiological studies intended to determine a relationship between occupational (respiratory) exposure to cadmium and lung and prostatic cancer, and these along with animal studies have provided considerable support for the carcinogenic potential of cadmium (Goyer 1996). Cadmium and certain cadmium compounds are therefore listed by the International Agency for Research on Cancer (IARC) as carcinogenic. The US Department of Health and Human Services in its 8th Report on Carcinogens, lists cadmium and certain cadmium

compounds as Reasonably Anticipated to be Human Carcinogens (USPHS 1998).

In addition to these toxic effects, it has also been suggested that cadmium may play a role in the development of hypertension (high blood pressure) and heart disease (USPHS 1997, Goyer 1996, Elinder and Jarup 1996). It is also known that severe oral exposure can result in severe irritation to the gastrointestinal epithelium, nausea, vomiting, salivation, abdominal pain, cramps and diarrhoea (USPHS 1997b).

3.3 Mercury

Mercury is an extremely toxic, non-essential trace metal, having no biochemical or nutritional function. Biological mechanisms for its removal are poor, and, mercury is the only metal known to biomagnify, that is, progressively accumulate as it passes though the food chain (WHO 1989).

Acute inhalation of high levels of mercury vapour may cause nausea, vomiting, diarrhoea, increases in blood pressure or heart rate, skin rashes, eye irritation, corrosive bronchitis and pneumonitis. And, if not fatal, may be associated with central nervous system (CNS) effects such as tremor or increased excitability (USPHS 1997, Goyer 1996). With chronic exposure, the major effects are on the CNS (tremor, spasms, loss of memory, increased excitability, severe depression, personality changes, even delirium and hallucination), although renal damage, associated with chronically exposed workers, has also been shown (Ratcliffe et al. 1996, Goyer 1996). These effects have also been reported in animal studies (USPHS 1997)

Acute exposure to high levels of mercury salts, or chronic low-dose exposure, is directly toxic to the kidney (Zalups and Lash 1994). In addition, nausea and diarrhoea may result after swallowing large amounts of inorganic mercury salts, and some nervous system effects have also been recorded (USPHS 1997, WHO 1989).

Once metallic mercury has entered the environment it can be methylated by micro-organisms, found for instance in aquatic sediments, to organic forms of mercury, most commonly methylmercury. In this form, it is able to cross cell membranes easily and quickly enters the aquatic food chain. From here it may enter the human food chain. Exposure to methylmercury has resulted in permanent damage to the CNS, kidneys, and the developing foetus. The levels of methylmercury that result in these effects are not usually encountered by the general population, however they were encountered by the population of Minamata, in Japan, who were exposed to high levels of methylmercury from eating contaminated fish and seafood collected from the Bay (USPHS 1997). Symptoms such as brain damage, numbness of extremities, and paralysis, along with the loss of hearing, speech and sight were reported (D'Itri 1991). However even today, the full range of neurological symptoms caused by the ingestion of methylmercury in fish and shellfish has not been fully characterised, and the total number of Minamata Disease sufferers has not been determined (D'Itri 1991). Furthermore, whilst only the Japanese cases have been confirmed as Minamata Disease, other populations in Canada (from chlor-alkali discharges) and Brazil (from gold mining) are potentially at risk. The problem of methylation of past and present inorganic mercury discharges continues, and the long retention time of mercury by sediments delays the elimination of contamination for many years (Harada 1997, Akagi *et al.* 1995, Bryan and Langston 1992, D'Itri 1991).

References for Appendix A

Abbey D.E., Ostro B.E., Petersen F. and Burchette R.J. (1995). Chronic respiratory symptoms associated with estimated long-term ambient concentrations of fine particulates less than 2.5 microns in aerodynamic diameter (PM2.5) and other air pollutants. Journal of Exposure Analysis and Environmental Epidemiology 5 (2): 137-159.

Akagi, H., Malm, O., Kinjo, Y., Harada, M., Branches, F.J.P, Pfeiffer, W.C. and Kato, H. (1995). Methylmercury pollution in the Amazon, Brazil. The Science of the Total environment 175: 85-95.

Alloway, B.J. (1990). Heavy metals in soils. John Wiley and Sons, Inc. New York, ISBN 0470215984.

Alloway, B.J. (1996). Soil pollution and land contamination. In Pollution, causes, effects and control, 3rd Edition. Harrison, R.M. [Ed]. The Royal Society of Chemistry, Cambridge, UK. ISBN 0854045341.

Allsopp M., Erry B., Stringer R., Johnston P. and Santillo D. (2000). Recipe for Disaster: a review of persistent organic pollutants in food. Greenpeace Research Laboratories. ISBN 90-73361-63-X.

ATSDR (1997) ATSDR's toxicological profiles on CD-ROM. U.S. Department of Health and Human Services, Public Health Service, CRC Press Inc, Boca Raton.

Ayres J.G. (1997). Trends in air quality in the UK. Allergy 52 (suppl 38): 7-13.

Ayres J.G. (1998). Health effects of gaseous air pollutants. In: Air Pollution and Health. Issues in Environmental Science and Technology 10 (eds.) R.E. Hester and R.M. Harrison. The Royal Society of Chemistry. ISBN 0-85404-245-8.

Bates D.V. and Sizto R. (1987). Air pollution and hospital admissions in Southern Ontario: The acid summer haze effect. Environmental Research 43: 317-331.

Bernard, A.M., Vyskocil, A., Kriz, J., Kodl, M. and Lauwerys, R. (1995). Renal effects of children living in the vicinity of a lead smelter. Environmental Research 68: 91-95.

Birnbaum, L.S. (1994) The mechanism of dioxin toxicity: relationship to risk assessment., Environmental Health Perspectives, 102(suppl. 9): 157-167

Braun-Fahrlander C., Ackermann-Liebrich U., Schwartz J., Gnehm H.P., Rutishauser M. and Wanner H.U. (1992). Air pollution and respiratory symptoms in preschool children. American Reviews in Respiratory Disease 145: 42-47.

Bryan, G.W. and Langston, W.J. (1992). Bioavailability, accumulation and effects of heavy metals in sediments with special reference to United Kingdom estuaries: a review. Environmental Pollution 76: 89-131.

Chestnut L.G., Schwartz J., Savitz D.A. and Burchfiel C.M. (1991). Pulmonary function and ambient particulate matter: epidemiological evidence from NHANES I. Archives of Environmental Health 46 (3): 135-144.

COMEAP, Committee on the Medical Effects of Air Pollutants (1995). Non-biological particles and health. Department of Health, UK. London: HMSO.

Dassen W., Brunekreef B., Hoek G., Hofschreuder P., Staatsen B., de Groot H., Schouten E. and Biersteker K. (1986). Decline in children's pulmonary function during an air pollution episode. Journal of Air Pollution Control Association 36 (11): 1223-1227.

DeVito, M.J., Birnbaum, L.S., Farland, W,H. & Gasiewicz T.A. (1995) Comparisons of Estimated Human Body Burdens of Dioxinlike Chemicals and TCDD Body Burdens in Experimentally Exposed Animals. Environmental Health Perspectives 103 (9): 820-831.

D'Itri, F.M. (1991). Mercury contamination: what we have learned since Minamata. Environmental Monitoring and Assessment 19: 165-182.

Dockery D.W., Ware J.H., Ferris B.G. Jr., Speizer F.E. and Cook N.R. (1982). Change in pulmonary function in children associated with air pollution episodes. Journal of Air Pollution Control Association 32: 937-942. (Cited in Dockery and Pope 1994).

Dockery D.W., Pope III C.A., Xu X., Spengler J.D., Ware H., Fay M.E., Ferris B.G. and Speizer F.E. (1993). The New England Journal of Medicine 329 (24): 1753-1759.

Donaldson K., Stone V., MacNee W. (2000). The toxicology of ultrafine particles. In: R.L. Maynard and C.V. Howard (eds). Particulate Matter: Properties and Effects Upon Health, BIOS Scientific Publishers Ltd., Oxford, UK. pp 63-84, ISBN 1-85996-172X.

Elinder, C.G. and Jarup, L. (1996). Cadmium exposure and health risks: recent findings. Ambio 25, 5: 370-373.

EPAQS, Expert Panel on Air Quality Standards, (1995). Particles. Published by HMSO. ISBN 0 11 753199 5.

Fingerhut M.A., Halperin W.E., Marlow D.A., Piacitelli L.A., Honchar P.A., Sweeny M.H., Griefe A.L., Dill P.A., Steenland K. and Surunda A.J. (1991). Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin. New England Journal of Medicine 324 (4): 212-218.

Gasiewicz, T., 1997. Exposure to dioxin and dioxin-like compounds as a potential factor in developmental disabilities. Mental Retardation and Developmental Disabilities Research Reviews 3: 230–238.

Goyer, R.A. (1993). Lead toxicity: current concerns. Environmental Health Perspectives 100: 177-187.

Goyer, R.A., Epstein, S., Bhattacharyya, M., Korach, K.S. and Pounds, J. (1994). Environmental risk factors for osteoporosis. Environmental Health Perspectives 102, 4: 390-394.

Goyer, R.A. (1996). Toxic effects of metals. In Casarett & Doull's Toxicology. The Basic Science of Poisons, Fifth Edition, Klaassen, C.D. (Ed). McGraw-Hill Health Professions Division, ISBN 0071054766.

Harada, M. (1997). Neurotoxicity of methylmercury; Minamata and the Amazon. In Mineral and Metal Neurotoxicology. Yasui, M., Strong, M.J., Ota, K. and Verity, M.A.[Eds]. CRC Press Inc., ISBN 0849376645.

Hoek G. and Brunekreef B. (1993). Acute effects of a winter air pollution episode on pulmonary function and respiratory symptoms of children 48 (5): 328-335.

Hoek G. and Brunekreef B. (1994). Effects of low-level winter air pollution concentration on respiratory health of Dutch children. Environmental Research 64: 136-150.

Howard C.V. (2000). Particulate aerosols, incinerators and health. In: Health Impacts of Waste Management Policies. Proceedings of the seminar "Health Impacts of Waste Management Policies", Hippocrates Foundation, Kos, Greece, 12-14 November 1998. Eds. P. Nicolopoulou-Stamati, L.Hens and C.V. Howard. Kluwer Academic Publishers.

Howard C.V. (2000b). Foreward. In: R.L. Maynard and C.V. Howard (eds). Particulate Matter: Properties and Effects Upon Health, BIOS Scientific Publishers Ltd., Oxford, UK. pp 63-84, ISBN 1-85996-172X.

Huisman M., Koopman-Esseboom C., Lanting C.I., Van der Paauw C.G., Tuinstra L.G.M., Fidler V., Weiglas-Kuperus N., Sauer P.J.J., Boersma E.R. and Touwen B.C.L.(1995). Neurological condition in 18-month old children perinatally exposed to PCBs and dioxins. Early human development 43: 165-176.

Ilsen A., Briet J.M., Koppe J.G., Pluim H.J. and Oosting J. (1996). Signs of enhanced neuromotor maturation in children due to perinatal load with background levels of dioxins. Chemosphere 33 (7): 1317-1326.

Iwata, K., Saito, H., Moriyama, M. and Nakano, A. (1993). Renal tubular function after reduction of environmental cadmium exposure: a ten year follow-up. Archives of Environmental Health 48, 3: 157-163.
Koenig J.Q., Larson T.V., Hanley Q.S., Rebolledo V., Dumler K. (1993). Pulmonary function changes in children associated with fine particulate matter. Environmental Research 63: 26-38.

Koopman-Esseboom C., Weisglas-Kuperus N., de Riddler M.A.J., Van der Paauw C.G., Tuinstra L.G.M.T. and Sauer P.J.J. (1995). Effects of PCBs/dioxin exposure and feeding type on the infants mental and psychomotor development. In: Effects of perinatal exposure to PCBs and dioxins on early human development by C. Koopman-Esseboom, ISBN 90-75340-03-6, (Chapter 8).

Manz A., Berger J., Dwyer J.H., Flesch-Janys D., Nagel S. and Waltsgott H. (1991). Cancer mortality among workers in a chemical plant contaminated with dioxin. The Lancet 338 (8773): 959-964.

Maynard R.L. (2000). Introduction. In: R.L. Maynard and C.V. Howard (eds). Particulate Matter: Properties and Effects Upon Health, BIOS Scientific Publishers Ltd., Oxford, UK. pp 63-84, ISBN 1-85996-172X.

McGregor D.B., Partensky C., Wilbourn J. and Rice J.M. (1998). An IARC evaluation of polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans as risk factors in human carcinogenesis. Environmental Health Perspectives 106 (suppl.2): 755-760.

Mueller, P.W., Paschal, D.C., Hammel, R.R., Klincewicz, S.L. and MacNeil, M.L. (1992). Chronic renal effects in three studies of men and women occupationally exposed to cadmium. Arch. Environ. Contam. Toxicol. 23: 125-136.

Neas L.M., Dockery D.W., Spengler J.D., Speizer F.E. and Tollerud D.J. (1992). The association of ambient air pollution with twice daily peak expiratory flow measurements in children American Reviews in Respiratory Diseases 145 (4): A429.

Nriagu, J.O. (1988). A silent epidemic of environmental metal poisoning. Environmental Pollution 50: 139-161.

Ostro B.D. (1990). Associations between morbidity and alternative measures of particulate matter. Risk Analysis 10 (3): 421-427.

Ostro B. (1993). The association of air pollution and mortality:n examining the case for inference. Archives of Environmental Health 48 (5):336-341.

Ostro B. and Rothschild S. (1989). Air pollution and acute respiratory morbidity: an observational study of multiple pollutants. Environmental Research 50: 238-247.

Pirkle, J.L., Kaufman, R.B., Brody, D.J., Hickman, T., Gunter, E.W. and Paschal, D.C. (1998). Exposure of the U.S. population to lead, 1991-1994. Environmental Health Perspectives 106, 11: 745-750.

Pope III C.A. (1989). Respiratory disease associated with community air pollution and a steel mill, Utah Valley. American Journal of Public Health 79: 623-628).

Pope III C.A., Dockery D.W., Spengler J.D. and Raizenne M.E. (1991). Respiratory health and PM-10 pollution: A daily time-series analysis. American reviews in Respiratory Diseases 144: 668-674. (Cited in Pope *et al.* 1995b).

Pope III C.A. and Dockery D.W. (1992). Acute health effects of PM-10 pollution on symptomatic and asymptomic children. American Reviews in Respiratory Diseases 145: 1123-1128. (Cited in Pope *et al.* 1995b).

Pope III C.A., Bates D. and Raizenne M. (1995a). Health effects of particulate air pollution: Time for reassessment? Environmental Health Perspectives 103 (5): 472-480.

Pope III C.A., Dockery D.W. and Schwartz J. (1995b). Review of epidemiological evidence of health effects of particulate air pollution. Inhalation toxicology 7: 1-18.

Pope II C.A., Thun M.J., Namboodiri M.M., Dockery D.W., Evans J.S., Speizer F.E. and Heath C.W. (1995c). Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am. J. Respir. Dis. Critical Care Med. 151: 669-674. (Cited in Pope *et al.* 1995a and 1995b).

QUARG (1996). Airborne Particulate Matter in the United Kingdom. Third Report of the Quality of Urban Air Review Group (QUARG), May. ISBN 0 9520771 3 2.

Ransom M.R. and Pope III C.A. (1991). Elementary school absences and PM10 pollution in Utah Valley. Environmental Research 58: 204-219.

Ratcliffe, H.E., Swanson, G.M. and Fischer, L.J. (1996). Human exposure to mercury: a critical assessment of the evidence of adverse health effects. Journal of Toxicology and Environmental Health 49: 221-270.

Rice D.C. (1996). Behavioural effects of lead: commonalities between experimental and epidemiologic data. Environmental Health Perspectives 104 (Suppl. 2): 337-350.

Roels, H., Bernard, A.M., Cardenas, A., Buchet, J.P., Lauwerys, R.R., Hotter, G., Ramis, I., Mutti, A., Franchini, I., Bundshuh, I., Stolte, H., De Broe, M.E., Nuyts, G.D., Taylor, S.A. and Price, R.G. (1993). Markers of early renal changes induced by industrial pollutants. III. Application to workers exposed to cadmium. British Journal of Industrial Medicine 50: 37-48.

Roemer W., Hoek G. and Brunekreef B. (1993). Effect of ambient winter air pollution on respiratory health of children with chronic respiratory symptoms. American Review of Respiratory Disease 147: 118-124.

Schwartz J. (1989). Lung function and chronic exposure to air pollution: a cross-sectional analysis of NHANES II. Environmental Research 50: 309-321.

Schwartz J. (1994a). Air pollution and daily mortality: a review and meta analysis. Environmental Research 64: 36-52.

Schwartz J. (1994b). Air pollution and hospital admissions for the elderly in Detroit, Michigan. American Journal of Respiratory and Critical Care Medicine. 150: 648-655.

Schwartz J. (1994c). Air pollution and hospital admissions for the elderly in Birmingham, Alabama. American Journal of Epidemiology 139 (6): 589-598.

Schwartz J. (1994d). PM10, ozone, and hospital admissions for the elderly in Minneapolis-St. Paul. Minnesota. Archives of Environmental Health 49 (5): 366-374.

Schwartz J. and Marcus A. (1990). Mortality and air pollution in London: a time series analysis. American Journal of Epidemiology 131 (1): 185-194.

Schwartz J., Slater D., Larson T.V., Pierson W.E. and Koenig J. (1993). Particulate air pollution and hospital emergency room visits for asthma in Seattle. Am Rev Respir Dis 147: 826-831.

Seaton A. (1995). Particulate air pollution and acute health effects. The Lancet 345: 176-178.

Sram R.J., Benes I., Binkova B., Dejmek J., Horstman D., Kotesovec F., Otto D., Perreault S.D., Rubes J., Selevan S.G., Skalik I., Stevens R.K. and Lewtas J. (1996). Teplice Program - The impact of air pollution on human health. Environmental Health Perspectives 104 (Suppl. 4): 699-714.

Sunyer J., Saez M., Murillo C., Castellsague J., Martinez F., Anto J.M. (1993). Air pollution and emergency room admissions for chronic obstructive pulmonary disease: A 5-year study. American Journal of Epidemiology 137 (7): 701-705.

Sweeney M.H. and Mocarelli P. (2000). Human health effects after exposure to 2,3,7,8-TCDD. Food Additives and Contaminants 17 (4): 303-316.

Thurston G.D., Ito K. amd Lippmann M. (1993). The role of particle mass vs. acidity in the sulfate-respiratory hospital admissions association. Preprint 93-MP-11.03. Presented at the 86th annual meeting and exhibition Denver, Colorado, June 13-18.

USEPA (1994) Health Assessment Document for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and related compounds. Volume III of III. EPA/600/BP-92/001c

USEPA (2000). Exposure and Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and related compounds. Part 1, Volume 3, Chapter 3, p27. EPA/600/P-00/001 Ab-Ae, March/May 2000.

USPHS (1997a). Toxicological profile for lead on CD-ROM. Agency for Toxic Substances and Disease Registry. U.S. Public Health Service.

USPHS (1997b). Toxicological profile for cadmium on CD-ROM. Agency for Toxic Substances and Disease Registry.

USPHS (1997c). Toxicological profile for Mercury on CD-ROM. Agency for Toxic Substances and Disease Registry. U.S. Public Health Service

USPHS (1998). 8th Report on Carcinogens 1998 Summary.

Van Birgelen, A.P.J.M. (1998). Hexachlorobenzene as a possible major contributor to the dioxin activity of human milk. Environmental Health Perspectives 106(11): 683-688.

Walters S., Griffiths R.K. and Ayres J.G. (1994). Temporal association between hospital admissions for asthma in Birmingham and ambient levels of sulphur dioxide and smoke. Thorax 49: 133-140.

Webster T. and Commoner B. (1994). Overview: The dioxin debate. In: Schecter A. (Ed.) Dioxins and Health. Publ: Plenum Press. pp1-32.

Weisglas-Kuperus N., Sas T.C., Koopman-Esseboom C., van der Zwan C., Riddler M.A.J., Boishuizen A., Hooijkaas H. and Sauer P.J.J. (1995). Immunological effects of background prenatal and postnatal exposure to dioxins and polychlorinated biphenyls in infants. Pediatric Research 30 (3): 404-410.

Weisglas-Kuperus, N., 1998. Neurodevelopmental, immunological and endocrinological indices of perinatal human exposure to PCBs and dioxins. Chemosphere 37: 1845-1853.

Whittemore A.S and Korn E.L. (1980). Asthma and air pollution in the Los Angeles area. American Journal of Public Health 70: 687-696. (Cited in Dockery and Pope 1994).

World Health Organisation (1989). Mercury. Environmental Health Criteria 86. ISBN9241542861.

WHO (1992). World Health Organisation.. Cadmium. Environmental Health Criteria 135. ISBN 9241571357.

WHO (1998). WHO experts re-evaluate health risks from dioxins. World Health Organisation. WHO/45. 3 June 1998

Zalups, R.K., Lash, L.H. (1994). Advances in understanding the renal transport and toxicity of mercury. Journal of Toxicology and Environmental Health 42: 1-44.

Zorber A., Messerer P. and Huber P. (1990). Thirty-four year mortality follow-up of BASF employees exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) after the 1953 accident. Int. Arch. Occ. Environ. Health 62: 139-157.

Zorber A., Ott M.G. and Messerer P. (1994). Morbidity follow-up study of BASF employees exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) after a 1953 chemical reactor incident. Occup Environ Med 51: 479-486.

APPENDIX B

INDIVIDUAL COMPOUNDS IDENTIFIED IN THE AIR EMISSIONS OF A MUNICIPAL WASTE INCINERATION PLANT

pentane
trichlorofluoromethane
acetonitrile
acetone
iodomethane
dichloromethane
2-methyl-2-propanol
2-methylpentane
chloroform
ethyl acetate
2,2-dimethyl-3-pentanol
cyclohexane
benzene
2-methylhexane
3-methylhexane
1,3-dimethylcyclopentane
1,2-dimethylcyclopentane
trichloroethene
heptane
methylcyclohexane
ethylcyclopentane
2-hexanone
toluene
1,2-dimethylcyclohexane
2-methylpropyl acetate
3-methyleneheptane
paraldehyde
octane
tetrachloroethylene
butanoic acid ethyl ester
butyl acetate
ethylcyclohexane
2-methyloctane
dimethyldioxane
2-furanecarboxaldehyde
chlorobenzene
methyl hexanol
trimethylcyclohexane
ethyl
benzene
formic acid
xylene
acetic acid
aliphatic carbonyl
ethylmethylcyclohexane
2-heptanone
2-butoxyethanol
nonane
isopropyl benzene
propylcyclohexane
dimethyloctane
pentanecarboxylic acid
propyl benzene
benzaldehyde
5-methyl-2-furane carboxaldehyde
1-ethyl-2-methylbenzene
1,3,5-trimethylbenzene
trimethylbenzene
benzonitrile
methylpropylcyclohexane
2-chlorophenol
1,2,4-trimethylbenzene
phenol
1,3-dichlorobenzene
1,4-dichlorobenzene
decane
hexanecarboxylic acid
1-ethyl-4-methylbenzene
2-methylisopropylbenzene
benzyl alcohol
trimethylbenzene
1-methyl-3-propylbenzene
2-ethyl-1,4-dimethylbenzene
2-methylbenzaldehyde
1-methyl-2-propylbenzene
methyl decane
4-methylbenzaldehyde
1-ethyl-3,5-dimethylbenzene
1-methyl-(1-pro-penyl)benzene
bromochlorobenzene
4-methylphenol
benzoic acid methyl ester
2-chloro-6-methylphenol
ethyldimethylbenzene
undecane
heptanecarboxylic acid
1-(chloromethyl)-4-methylbenzene
1,3-diethylbenzene
1,2,3-trichlorobenzene
4-methylbenzyl
alcohol
ethylhex anoic acid
ethyl benzaldehyde
2,4-dichlorophenol
1,2,4-trichlorobenzene
naphthalene
cyclopentasiloxanedecamethyl
methyl acetophenone
ethanol-1-(2-butoxyethoxy)
4-chlorophenol
benzothiazole
benzoic acid
octanoic acid
2-bromo-4-chlorophenol
1,2,5-trichlorobenzene
dodecane
bromochlorophenol
2,4-dichloro-6-methylphenol
dichloromethylphenol
hydroxybenzonitrile

tetrachlorobenzene
methylbenzoic acid
trichlorophenol
2-(hydroxymethyl) benzoic acid
2-ethylnaphthalene-1,2,3,4-tetrahydro 2,4,6-trichlorophenol
4-ethylacetophenone
2,3,5-trichlorophenol
4-chlorobenzoic acid
2,3,4-trichlorophenol
1,2,3,5-tetrachlorobenzene
1,1'biphenyl (2-ethenyl-naphthalene)
3,4,5-trichlorophenol
chlorobenzoic acid
2-hydroxy-3,5-dichlorobenzaldehyde
2-methylbiphenyl
2-nitrostyrene(2-nitroethenylbenzene)
decanecarboxylic acid
hydroxymethoxybenzaldehyde
hydroxychloroacetophenone
ethylbenzoic acid
2,6-dichloro-4-nitrophenol
sulphonic acid
m.w. 192
4-bromo-2,5-dichlorophenol
2-ethylbiphenyl
bromodichlorophenol
1(3H)-isobenzofuranone-5-methyl
dimethylphthalate
2,6-di-tertiary-butyl-p-benzoquinone
3,4,6-trichloro-1-methyl-phenol
2-tertiary-butyl-4-methoxyphenol
2,2'-dimethylbiphenyl
2,3'-dimethylbiphenyl
pentachlorobenzene
bibenzyl
2,4'-dimethylbiphenyl
1-methyl-2-phenylmethylbenzene
benzoic acid phenyl ester
2,3,4,6-tetrachlorophenol
tetrachlorobenzofurane
fluorene
phthalic ester
dodecanecarboxylic acid
3,3'-dimethylbiphenyl
3,4'-dimethylbiphenyl
hexadecane
benzophenone
tridecanoic acid
hexachlorobenzene
heptadecane
fluorenone
dibenzothiophene
pentachlorophenol
sulphonic acid m.w. 224
phenanthrene
tetradecanecarboxylic acid
octadecane
phthelic ester
tetradecanoic acid isopropyl ester
caffeine
12-methyltetradecacarboxylic acid
pentadecacarboxylic acid
methylphenanthrene
nonedecane
9-hexadecene carboxylic acid
anthraquinone
dibutylphthalate
hexadecanoic acid
eicosane
methylhexadecanoic acid
fluoroanthene
pentachlorobiphenyl
heptadecanecarboxylic acid
octadecadienal
pentachlorobiphenyl
aliphatic amide
octadecanecarboxylic acid
hexadecane amide
docosane
hexachlorobiphenyl
benzylbutylphthalate
aliphatic amide
diisooctylphthalate
hexadecanoic acid hexadecyl ester
cholesterol.

Source:
Jay K. and Stieglitz L. (1995). Identification and quantification of volatile organic components in emissions of waste incineration plants. Chemosphere 30 (7): 1249-1260.