North Yorkshire Waste Action Group
Objection to Allerton Waste Recovery Park:

HEALTH RISKS: ADVERSE EFFECTS FROM INCINERATOR EMISSIONS

December 2011

ADVERSE HEALTH EFFECTS FROM INCINERATOR EMISSIONS

CONTENTS

SUMMARY

1. Health impacts arising from waste incineration is a contentious subject for many reasons (complexity, uncertainty, vested interests, the nature of the 'scientific method', difficulties 'proving' causal relationships, 'confounding factors' including both social factors and other sources of pollution, etc.). Thus, while no one disputes that fact that waste incineration carries risks to human health there is inevitably debate about the degree of harm and the 'acceptability' of the risks.

2. This Chapter draws together the health risks from each of the emissions discussed in the Chapter on "Harmful Emissions and their Properties" and includes synergistic effects such as Polycyclic Aromatic Hydrocarbons (PAHs) depositing on particulates and acting synergistically to damage health. Carcinogenicity is a recurring theme among many of the pollutants emitted by incinerators. Even with emission control measures, there remain carcinogenic, mutagenic and/or teratogenic emissions (e.g. Dioxins Furans, PAHs) and there is no safe lower limit for carcinogens. Despite this evidence (see main text), the extent of health risks is controversial and HPA among others minimise the risks. Their views are therefore discussed.

3. While there are some caveats, it is reasonable to ascribe the following health risks to incinerator emissions:
   - **Cancers**: leukemia, non-Hodgkin's lymphoma, brain, breast, colon, lung, bladder, kidney, liver and stomach. This includes some childhood cancers.
   - **Respiratory Disease &Asthma,** Chronic obstructive pulmonary disease (COPD), making one a degree more prone to viral & other respiratory or other infections
   - **Birth defects** - terminations, live defects, miscarriages.
   - **Premature deaths** of babies, infants and adults including stillbirths
   - **Coronary artery disease**, heart attacks, arteriosclerosis, strokes, SADS (Cardiac arrhythmia also known as "Sudden Adult Death Syndrome" and "Sudden Arrhythmia Death Syndrome") This may be in the form of aggravating existing problems
   - **Multiple chemical sensitivity** with allergies and arthritis
   - **Endocrine system problems** such as **Hypothyroidism** (part of obesity problem) - endocrine glands, **Endometriosis** & other hormones disrupted and **Diabetes 2** (and sometimes diabetes 1) through effect on endocrine glands
   - **Lower IQ and educational achievement,** heavy metals produce symptoms such as memory loss, poor concentration and poor sleep as well as behavioral problems that could account for this
   - **Behavioral problems such as Attention Deficit Disorder,** noting the similarities between heavy metal poisoning and conditions such as autism and ADD/ADHD.

4. In addition, particulates have known links to a number of other less serious but debilitating conditions such as an increase in upper respiratory symptoms (runny or stuffy nose; sinusitis; sore throat; wet cough; head colds; hay fever; and burning or red eyes) and increase in lower respiratory symptoms (wheezing; dry cough; phlegm; shortness of breath; and chest discomfort or pain).

5. Other, but more controversial adverse health effects include contributions to ME/ Chronic Fatigue Syndrome symptoms, a role in Sudden Infant Death Syndrome, cot deaths, autism and other conditions such as MS and depressive illnesses.

6. UK Epidemiological Evidence shows excess cancer both among adults and children around incinerators. For children risks were greater than for adults and the excess cancers were similar for

leukemia and solid tumours of all types, as might be expected with agents that have systemic access to the DNA of all types of fetal cells.

7. The French Institute for Public Health Surveillance examined the relation between cancer risk and past exposure to Municipal Solid Waste (MSW) Incinerators for the populations living near to them. They found excess cancer risks which reflect older exposure conditions (1972-1985) and are therefore not directly applicable to today's incinerators. Crucially, however, they indicated the excess risk at different distances so that one can infer that there remains a risk even after considerable plume dispersion (and therefore dilution). Thus, they do show the reality of adverse health effects. They covered Liver cancer (both sexes), Malignant non-Hodgkins lymphoma (both sexes), Soft-tissue sarcoma (both sexes), All cancers in women and Breast cancer in women.

8. There is now robust scientific evidence on the dangers to health of fine and ultrafine particulates and of the substantial health costs involved. Recent studies have shown the risk to be considerably greater than previously thought. As Dearden says, it is now established beyond reasonable doubt that particulate air pollution causes death by various means. Hence it is impossible to justify increasing levels of these particulates still further by building incinerators or any other major source of particulates. The data makes it clear that attempts should be made to the reduce levels of these particulates whenever possible.

9. Babies (including those *in utero*) and children face a higher health risk from incinerator emissions than adults, mainly because

    1. Children have a relatively faster metabolism than adults and, for example, breathe more rapidly. Thus they take in a greater pollution load relative to body weight than do adults
    2. Children's tissues are developing and are therefore more affected by the same pollutant load than the "static" tissues of adults.
    3. Breast fed babies take on dioxins and other toxic chemicals (e.g. PCBs) through the mother's milk while babies are exposed to their mothers' toxins *in utero*. .

10. Chemically sensitive individuals are also markedly more susceptible to adverse health impacts.

11. It has been known for many years that some toxicants could exhibit high toxicity at very low doses; this phenomenon is termed hormesis. The cancer risk from dioxins appears to follow a hormetic pattern, with toxicity increasing at very low doses and endocrine disruptors such as dioxins follow this pattern. Thus "*evidence is accumulating that low, perhaps even very low, levels of dioxins and other toxicants can carry very serious health risks"*

12. The current regulatory regime in the UK (and EU) falls short of best practice, though it has led to improved air quality as pollution generally has been lowered (this includes incinerators). However, the HPA appear not to have taken account of the fact that the smaller particles (PM_1 and ultrafine particles, $PM_{0.1}$) are more dangerous than the regulated PM_{10} and $PM_{2.5}$s; certainly regulatory limits do not take account of this.

13. The counter-argument by those who do not accept that there are health risks appears to be based around something like "the causal links between emissions from combustion (including incineration) are known, at least in many cases, but the risks are yet to be accepted" or take the view that results are inconsistent or choose to dismiss results from older data because they regard it as irrelevant in the light of modern pollution control techniques. The weight of evidence does not favour such views.

14. Quantitative calculations of the health risks associated with a modern MSW incinerator based on current allowed emission levels show that the risk of dying from incinerator emissions over the 25 year operating life of an incinerator is 6.23×10^{-6}, and the 70-year lifetime risk is 1.74×10^{-5}. Both of these values are well above the *de minimis* acceptable lifetime target level of 10^{-6} (i.e. 1 in a million) used by the US Environmental Protection Agency and recommended by the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment. These figures demonstrate that even new incineration plant such as the proposed EfW (incinerator) plant at AWRP has the potential to cause serious health risks.

Conclusions

15. There is considerable evidence from a wide variety of sources that incineration is linked directly to a wide range of adverse health impacts. Moreover, calculations of the risks of modern incinerators that meet current emission standards show that they exceed the generally accepted risk criterion of 1 in a million. There is a considerable body of opinion that holds that objective scientific evaluation indicates that there are reasonable grounds for concern about potentially dangerous effects of incinerator emissions on human health, with babies and young children being amongst the most vulnerable. However, official UK bodies do not share this scientific consensus

16. Under the law of the European Union, the application of the Precautionary Principle has been made a statutory requirement. It presupposes that potentially dangerous effects deriving from a phenomenon, product or process have been identified, and that scientific evaluation does not allow the risk to be determined with sufficient certainty. This is the situation regarding the health risks associated with incineration in general and therefore the health risks associated with emissions from the AWRP EfW (incinerator) plant.

17. EU Treaty Article 174(2) as amended at Nice 2004 recognized that scientific evaluation can be inconclusive and accorded priority to public health:

    "*a precautionary approach must be paramount, as opposed to acting only where proof or very strong suspicion of harm can be demonstrated".* And

18. We therefore contend that the Precautionary Principle must be applied to your consideration of the proposed EfW (incinerator) plant at AWRP. We very strongly object both to the harm that this plant would do to the health of the local area and beyond and to the health costs it would burden the NHS with.

19. Given that

- *"the protection of public health, including the effects of the environment on public health, must be given priority"* [EU Treaty Article 174(2)]
- There is a large body of scientific evidence and opinion that indicates there are reasonable grounds for concern about potentially dangerous effects of incinerator emissions on human health, with babies and young children being amongst the most vulnerable.
- Incineration is linked directly to a wide range of adverse health impacts including cancers, heart disease, diseases of the respiratory tract, endocrine system disorders and the effects of toxic heavy metals.
- Modern incinerators that meet current emission standards exceed the generally accepted risk criterion for cancer.
- Local planning policies and the UK's onligations under the Stockholm convention would be run counter to granting planning permission for the EfW (incineration) plant.

- Incineration uis not a sustainable development.

and that

- there are cheaper and cleaner alternatives which are free of the health risks associated with incineration

We urge you to refuse planning permission for the AWRP EfW (incinerator) plant

1: INTRODUCTION

1. Health impacts arising from waste incineration is a contentious subject for many reasons (complexity, uncertainty, vested interests, the nature of the 'scientific method', difficulties 'proving' causal relationships, 'confounding factors' including both social factors and other sources of pollution, etc.). Thus, while no one disputes that fact that waste incineration carries risks to human health there is inevitably debate about the degree of harm and the 'acceptability' of the risks.

2. The Chapter on *Harmful Emissions and their Properties* reviews the main emissions from Municipal Solid Waste (MSW) incinerators. They are fed by a variable and uncertain mix of materials so emissions are not constant but include varying quantities of substances harmful to man, wildlife or the environment. Emissions include chemicals derived from substances found in the waste or produced during its decomposition or both together with combustion products (e.g. NO_x). Despite emission control measures, there remain carcinogenic, mutagenic and/or teratogenic emissions (e.g. dioxins (PCDDs), and furans (PCDFs)), endocrine disruptors[i] (e.g. dioxins, PCBs, PBDEs). Other related compounds are polychlorinated biphenyls (PCBs), many of which are endocrine disrupters, polybrominated diphenyl ethers (PBDEs) and polybrominated biphenyls (PBBs). Their effect can be enhanced by their presence on particulates (these can act synergistically with Polycyclic Aromatic |Hydrocarbons (PAHs) which can deposit on particulates, providing a path for longer term deposition in the body. Some particulates are sufficiently small to enter the sensitive lung tissue and damage it, causing premature death in extreme cases. Further, there are acid gas emissions; NO_x reacts with ammonia, moisture, and other compounds to form nitric acid vapour and related particles, inhalation of which may cause or worsen respiratory diseases such as emphysema, bronchitis and/or aggravate existing heart disease.

3. The health risks associated with these pollutants are known but the dose-response relationship at low doses is subject to uncertainty. In the face of such uncertainty it is essential to:

    - Establish evidence through studies that have used proper epidemiological evidence
    - Make allowance for the time at which they were carried out since incinerator emission control systems have improved markedly over the last decade
    - Take account of which groups of people are most at risk
    - Apply the Precautionary Principle when making decisions

4. There is a substantial amount of evidence relating to health risks but interpreting it is not always easy. This Chapter reviews studies which establish the risks of pollutants typically associated with incinerators as well as studies showing excess mortality and morbidity around a number of incinerators and summarises the many health risks associated with incineration.

[i] The **endocrine system** is a system of glands, each of which secretes a type of hormone directly into the bloodstream to regulate the body. It is an information signal system like the nervous system, yet its effects and mechanism are classifiably different. Its' effects are slow to initiate, and prolonged in their response, lasting for hours to weeks. Hormones are substances (chemical mediators) released from endocrine tissue into the bloodstream where they travel to target tissue and generate a response. Hormones regulate various human functions, including metabolism, growth and development, tissue function, and mood. Endocrine glands are ductless nature. In addition to these endocrine glands, many other organs that are part of other body systems, such as the kidney, liver, heart and gonads, have secondary endocrine functions.

5. The extent of health risks is controversial and the Environment Agency and the Health Protection Agency among others minimise the risks. This is discussed below as it will influence planning decisions.

6. In the chapter on Harmful Emissions we noted that DEFRA[1] have stated that "*Public concern is a material planning consideration and has in part led to previous applications* [for waste incinerators] *being refused (e.g. Kidderminster). Public concern founded upon valid planning reasons can be taken into account when considering a planning application*" However, in discussing health effects, it is essential to take account of the Precautionary Principle which is enshrined in European law.

2: PRECAUTIONARY PRINCIPLE

7. In considering what he called "*toxic harm allocation*", Michaelson[2] pointed out that toxics present a classic public choice dilemma: the balancing of desired goods against the threat they pose to human life: "*Though its rules vary with the statutes and substances in question, toxic harm allocation may be understood as a game with three players—industry, producing the harm; (the regulatory authority), allocating it; and individuals, receiving it—who cooperate or compete to set, measure, and regulate the levels of toxins in the environment*".

8. This question of "*toxic harm allocation*" applies to the EfW (incinerator) plant at AWRP. In considering the planning application, it is necessary to balance the claimed "benefits" of incinerating NYCC's and York's MSW in the EfW (incinerator) plant at AWRP with the clear interest of local people to avoid the various categories of environmental and health harm that may befall them (see later). In such situations, it has become customary to invoke the Precautionary Principle.

9. The **Precautionary Principle**[ii] states that if an action or policy has a suspected risk of causing harm to the public or to the environment, in the absence of scientific consensus that the action or policy is harmful, then the burden of proof that it is *not* harmful falls on those taking the action. This principle allows policy makers to make discretionary decisions in situations where there is the possibility of harm from taking a particular course or making a certain decision when extensive scientific knowledge on the matter is lacking. The principle implies that there is a social responsibility to protect the public from exposure to harm, when scientific investigation has found a plausible risk. These protections can be relaxed only if further scientific findings emerge that provide sound evidence that no harm will result.

10. There are several definitions of the **Precautionary Principle**. An early definition arose from the work of the Rio Conference, or "Earth Summit" in 1992[iii]. Principle #15 of the Rio Declaration notes that:

> "*In order to protect the environment, the precautionary approach shall be widely applied by States according to their capabilities. Where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation*."

[ii] The Precautionary Principle, which is essentially used by decision-makers in the management of risk, should not be confused with the element of caution that scientists apply in their assessment of scientific data.

[iii] The Rio Declaration on Environment and Development, often shortened to Rio Declaration, was a short document produced at the 1992 United Nations "Conference on Environment and Development" (UNCED), informally known as the Earth Summit. The Rio Declaration consisted of 27 principles intended to guide future sustainable development around the world

11. Perhaps the most comprehensive definitions of the **Precautionary Principle** is the so-called Wingspread Statement, quoted by Science & Environmental Health Network[3]:
"*When an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically. In this context the proponent of an activity, rather than the public, should bear the burden of proof. The process of applying the precautionary principle must be open, informed and democratic and must include potentially affected parties. It must also involve an examination of the full range of alternatives, including no action". The PP does not seek to establish zero risk, since all human activity involves some risk. It does, however, involve an assessment, either subjective or objective, of both risk and benefit from a proposed activity, leading to a decision as to whether or not the proposed activity should be permitted. Involved in such a decision are a number of factors, including whether or not valid and realistic alternatives are available"*.

    The **Precautionary Principle** does not seek to establish zero risk, since all human activity involves some risk. It does, however, involve an assessment, either subjective or objective, of both risk and benefit from a proposed activity, leading to a decision as to whether or not the proposed activity should be permitted. There are a number of factors involved in such a decision, including whether or not valid and realistic alternatives are available.

12. Under the law of the European Union (binding in this country), the application of the Precautionary Principle has been made a statutory requirement[4,5]. On 2 February 2000, the European Commission issued a Communication on the Precautionary Principle[6] in which it adopted a procedure for the application of this concept, but without giving a detailed definition of it. it (Annex 1 gives more details This pointed out the need to balance the freedom and rights of individuals, industry and organizations with the need to reduce the risk of adverse effects to the environment, human, animal or plant health in a proportionate and non-discriminatory manner. Its scope covers situations where preliminary objective scientific evaluation, indicates that there are reasonable grounds for concern about potentially dangerous effects on the environment, human, animal or plant health being inconsistent with the level of protection chosen for the Community.

13. Paragraph 2 of article 191 of the Lisbon Treaty[7] states that
"Union policy on the environment shall aim at a high level of protection taking into account the diversity of situations in the various regions of the Union. It shall be based on the Precautionary Principle and on the principles that preventive action should be taken, that environmental damage should as a priority be rectified at source and that the polluter should pay."

14. The 2 February 2000 European Commission Communication indicates that the Precautionary Principle should be considered within a structured approach to the analysis of risk which comprises three elements: risk assessment, risk management, risk communication and that it is particularly relevant to the management of risk. It is presupposed that potentially dangerous effects deriving from a phenomenon, product or process have been identified, and that scientific evaluation does not allow the risk to be determined with sufficient certainty. We contend that this applies to risks associated with emissions from the AWRP EfW (incinerator) plant.

15. The 2 February 2000 European Commission Communication advises that implementation of an approach based on the Precautionary Principle should start with a scientific evaluation, as complete as possible, and where possible, identifying at each stage the degree of scientific uncertainty. We have adopted this approach in this Chapter. However, it is not possible to give precise ranges of uncertainty because of different perceptions of the extent of risk and the factors that make up that risk.

16. The EU Treaty Article 174(2) as amended at Nice 2004 recognized that scientific evaluation can be inconclusive and accorded priority to public health:

> "*a precautionary approach must be paramount, as opposed to acting only where proof or very strong suspicion of harm can be demonstrated. The Precautionary Principle should be applied where the possibility of harmful effects on health or the environment has been identified and preliminary scientific evaluation proves inconclusive for assessing the level of risk. Account should be taken of social and environmental costs in examining the level of risk, but the protection of public health, including the effects of the environment on public health, must be given priority*".

17. As revealed in the Chapter on Harmful Emissions and below there is, at the very least, the possibility of harm from deciding to allow the EfW (incinerator) plant at AWRP. While there is extensive scientific research into the health effects of incinerator emissions, there is not a general consensus on its magnitude. Under these circumstances, the Precautionary Principle must apply.

3: OVERVIEW OF HEALTH RISKS

18. Emissions will inevitably include particulates and/or aerosols which can damage health. There is the added factor that the risks arising from particulates can act synergistically with those of polycyclic aromatic hydrocarbons as the latter can deposit on particulates and thereby provide a path for longer term deposition in the body. Thus dust and aerosols from the plant and from rubbish awaiting incineration may carry irritant, toxic or carcinogenic chemicals. These emissions will have a cumulative effect on people in the surrounding area; there is no safe lower limit for carcinogens. No-one can be confident that this risk is low because the very nature of rubbish is that it contains unexpected materials (e.g. a little asbestos, however illegally present). The main risk is cancer and the effects are cumulative.

19. Despite emission control measures, there remain carcinogenic, mutagenic and/or teratogenic emissions (e.g. dioxins furans, PAHs) and endocrine disruptors (e.g. dioxins, PCBs, PBDEs) together with the possibility that their effect is enhanced by their presence on particulates, some of which are sufficiently small to enter the sensitive lung tissue and damage it, causing premature death in extreme cases. Further, there are acid gas emissions; NO_x reacts with ammonia, moisture, and other compounds to form nitric acid vapor and related particles, inhalation of which may cause or worsen respiratory diseases such as emphysema, bronchitis and/or aggravate existing heart disease.

20. Ozone arises from incinerators as a result of NO_x reacting with volatile organic compounds in the presence of heat and sunlight. It can damage lung tissue and reduce lung function, mostly in susceptible populations (children, elderly, and asthmatics). Ozone can be transported by wind currents and cause health impacts far from the original sources. NO_x also readily reacts to form a wide variety of toxic products: nitroarenes[iv] (suspected human carcinogens[8]), nitrosamines

[iv] The nitroarenes comprise a large class of structurally related chemicals normally found in particulate emissions from many combustion sources, notably diesel exhausts. Carcinogenicity results with experimental animals typically show tumour formation both at the site of injection and at sites away from it. The chemicals also show genotoxic activity in a variety of *in vitro* and *in vivo* assays, and metabolic pathways for the creation of reaction products with the ability to cause gene mutations or changes in the structure of DNA in tissues from animals as well as humans. Although adequate human studies of the relationship between exposure to these chemicals and human cancer have been reported, they are *reasonably anticipated to be a human carcinogen*.

(carcinogenic in a wide variety of animal species, suggesting carcinogenicity in humans[v].[9,10]) and also the nitrate radical some of which may cause biological mutations.

21. There are many endocrine disrupting compounds including industrial by-products and pollutants. Some are pervasive and widely dispersed in the environment. Some are persistent organic pollutants (POPs)[vi] while others are rapidly degraded in the environment or human body or may be present for only short periods of time[11]. Health effects attributed to endocrine disrupting compounds include a range of reproductive problems (reduced fertility, male and female reproductive tract abnormalities, and skewed male/female sex ratios, loss of fetus, menstrual problems[12], changes in hormone levels; early puberty; brain and behavior problems; impaired immune functions; and various cancers[13].

22. Specifically, there are a number of health effects arising from acute exposure (not relevant to incinerators) to PCBs[vii]. PCB use was therefore banned worldwide in 1977. Recent studies show the endocrine interference of certain PCB congeners is toxic to the liver and thyroid[14], increases childhood obesity in children exposed prenatally[15] and may increase the risk of developing diabetes.[16,17]

23. PBDEs have the potential to disrupt thyroid hormone balance and contribute to a variety of neurological and developmental deficits, including low intelligence and learning disabilities[18,19]. Many of the most common PBDE's were banned in the European Union in 2006[20].Studies with rodents have suggested that even brief exposure to PBDEs can cause developmental and behavior problems in juveniles[21,22]and exposure interferes with proper thyroid hormone regulation. Research has correlated halogenated hydrocarbons, such as PCBs, with neurotoxicity[23]. As PBDEs are similar in chemical structure to PCBs, and it has been suggested that PBDEs act by the same mechanism as PCBs[24].

24. There are heavy metals in the incinerator emissions. The symptoms of toxic heavy metal poisoning and the symptoms of autism, PDD, Aspergers, & ADD/ ADHD are very similar.

25. There are some caveats to the effects described above since doses will be low and some of the data is inferred from animal experiments, However, there is no safe dose for cancers and some of the above materials are cumulative. Bearing in mind such caveats, it is reasonable to ascribe the following health risks to incinerator emissions:

- **Cancers:** leukemia, non-Hodgkin's lymphoma, brain, breast, colon, lung, bladder, kidney, liver and stomach. This includes some childhood cancers.
- **Birth defects** - terminations, live defects, miscarriages.
- **Premature deaths** of babies, infants and adults including stillbirths
- **Respiratory Disease &Asthma, COPD**[viii], making one a degree more prone to viral and other respiratory or other infections

---

[v] About 300 Nitrosamines and N-nitroso compounds have been tested, with 90% found to be carcinogenic in a wide variety of experimental animals. Most nitrosamines are mutagens and a number are transplacental carcinogens. Most are organ specific. For instance, dimethylnitrosamine causes liver cancer in experimental animals, whereas some of the tobacco specific nitrosamines cause lung cancer. Since nitrosamines are metabolized the same in human and animal tissues, it seems highly likely that humans are susceptible to the carcinogenic properties of nitrosamines.

[vi] POPs are organic compounds that are resistant to environmental degradation through chemical, biological, and photolytic processes and thus can persist in the environment, be capable of long-range transport, accumulate in human and animal tissue and hence in food chains.

[vii] These include chloracne (a severe acne-like condition resulting from skin contact) and increased risk of skin cancer, liver cancer, and brain cancer

[viii] Chronic obstructive pulmonary disease (**COPD**) refers to chronic bronchitis and emphysema, a pair of two commonly co-existing diseases of the lungs in which the airways become narrowed

- **Coronary artery disease**, heart attacks, arteriosclerosis, strokes, SADS (Cardiac arrhythmia[ix], also known as "Sudden Adult Death Syndrome" and "Sudden Arrhythmia Death Syndrome") This may be in the form of aggravating existing problems
- **Multiple chemical sensitivity** with allergies and arthritis
- **Endocrine system problems such as**
    - **Hypothyroidism** (part of obesity problem) - endocrine glands
    - **Endometriosis** & other hormones disrupted.
    - **Diabetes 2** (and sometimes diabetes 1) through effect on endocrine glands
- **Lower IQ and educational achievement,** heavy metals produce symptoms such as memory loss, poor concentration and poor sleep as well as behavioral problems that could account for this
- **Behavioral problems such as Attention Deficit Disorder,** noting the similarities between heavy metal poisoning and conditions such as autism and ADD/ADHD. (see above)

26. This list suggests a range of problems, depending on the dose/response relationship of individuals and the actual doses. At least the routes and potential causes are identifiable from the above discussion. In addition, particulates have known links to a number of other less serious but debilitating conditions such as an increase in upper respiratory symptoms (runny or stuffy nose; sinusitis; sore throat; wet cough; head colds; hay fever; and burning or red eyes) and increase in lower respiratory symptoms (wheezing; dry cough; phlegm; shortness of breath; and chest discomfort or pain).

27. A somewhat provocatively titled Country Doctor article[25] opposed to incineration lists a number of other adverse health effects which are less easy to evaluate. These comprise:

- **ME** (gasses affect P450[x] & T-lymphocyte[xi] diversion) and Chronic Fatigue Syndrome. *Surely not the whole picture though heavy metals, particularly cadmium contribute to the causation of chronic fatigue.*
- **T-lymphocyte diversion**/depletion causes Sudden Infant Death Syndrome[xii], cot deaths, autism and other conditions such as MS. *Again, this would appear to be a case of either not being the whole picture (e.g. SIDS/cot deaths may have a number of causes, for example undetected Supra-Ventricular Tachycardia) while possible causes for MS and autism are unknown, though there are a number of suspicions.*
- **Clinical depression** suicides, apathy, part of obesity problem – *again a multi-faceted problem, though heavy metals could perhaps be among the many causes of depression*.

4: EVIDENCE OF HEALTH RISKS

28. The Parliamentary Office of Science and Technology recognise that air pollution causes health problems[26]. They stated that air pollution legislation has mainly been and still remains focused on

[ix] A large and heterogeneous group of conditions in which there is abnormal electrical activity in the heart The heart beat may be too fast or too slow, and may be regular or irregular.

[x] The **cytochrome P450** superfamily (officially abbreviated as **CYP**) is a large and diverse group of enzymes. The function of most CYP enzymes is to catalyze the oxidation of organic substances. The substrates of CYP enzymes include metabolic intermediates such as lipids and steroidal hormones, as well as xenobiotic substances such as drugs and other toxic chemicals. CYPs are the major enzymes involved in drug metabolism and bioactivation, accounting for about 75% of the total number of different metabolic reactions.

[xi] T lymphocytes belong to a group of white blood cells known as lymphocytes, and play a central role in cell-mediated immunity.

[xii] Typically the infant is found dead after having been put to bed, and exhibits no signs of having suffered. SIDS is a diagnosis of exclusion. It can only be applied to an infant whose death is sudden and unexpected, and remains unexplained after the performance of an adequate postmortem investigation including an autopsy; investigation of the scene and circumstances of the death; exploration of the medical history of the infant and family.

reducing the adverse human health effects of air pollutants, although during the 1980s acid rain and ecosystem damage were a principal concern (and climate change is today – see Climate Change Chapter). They reported that the levels of air pollutants measured today (2002) can still give rise to significant health impacts. In 1992, the Department of Health (DH) set up a Committee on the Medical Effects of Air Pollutants (COMEAP) to examine the potential toxicity and effects on health of air pollutants. In their 1998 report COMEAP[27] concluded that up to 24,000 deaths were 'brought forward' in the UK in 1995/1996 due to the short term effects of air pollution[xiii]. They stated that research indicates that long-term exposure could have an even greater impact, although this has been difficult to quantify and gave an overview of health problems for which there is moderately consistent evidence that a causal link with air pollution exists (Table 1).

Table 1 Reasonably Established Health effects of air pollutants

Mostly elderly and young people and those with respiratory diseases such as asthma or bronchitis are affected. • SO_2 - coughing, tightening of chest, irritation of lungs • NO_2 - irritation and inflammation of lungs • PM_{10} - inflammation of lungs, worsening of symptoms of people with heart and lung conditions, linkage of long term exposure to coronary heart disease and lung cancer • *CO* - prevention of normal transport of oxygen by blood, resulting in the reduction of oxygen supply to the heart • *ozone* - pain on deep breathing, coughing, irritation and inflammation of lungs • *benzene* - cause of cancer • *1,3-butadiene* - cause of cancer • *polycyclic aromatic hydrocarbons (PAHs)* - toxicity and cause of cancer • *lead* - linkage of exposure to impaired mental function and neurological damage in children

Source: Parliamentary Office of Science and Technology

29. Many if these are substances are emitted from incinerators. The table was prepared before there was much focus on $PM_{2.5}$s and the omission of dioxins and furans, covered by the Stockholm Convention, is curious. Nevertheless it offers a first indication that health effects are real and that the list of effects given in paragraph 20 is reasonable. This is therefore a first indication of the possibility of harm from the proposed EfW (incinerator) plant at AWRP but it does not indicate the scale of the effect.

4.1 Interpreting the Evidence - The Need for Care

30. In principle the health impacts of incinerators could be seen from considering the death rate upwind and downwind of several incinerators since the combination of a high stack and prevailing wind means that pollutants are dispersed predominantly downwind. This might be seen most clearly by considering "at risk" groups (e.g. children and asthmatics). However, care is needed in interpreting any such data because apparent variations in mortality can be due to various complicating factors which have nothing to do with incineration that make interpretation more difficult than it seems at first sight. Such factors should be carefully assessed to avoid misleading results; indeed any approach that does not use proven epidemiological methodology should be treated with caution.

31. A simple example makes the point. Maps of, say, infant mortality rates in locations upwind and downwind of a mass-burn incinerator may look superficially striking. However, being based on raw data, they may be misleading due to a number of additional factors that serve to confuse the picture. For example infant mortality is strongly correlated with weight at birth and depends on socio-economic group, itself strongly linked to social well-being and at least in England and

[xiii] In 1995 around 12,500 deaths were 'brought forward', which means vulnerable people might have lived longer if air pollution was not a factor.

Wales, on ethnicity and socio-economic conditions. Thus infant mortality reflects a lot of complicated process, notably poverty (broadly defined) and ethnicity and there is a strong ethnic contrast and strong socio-economic differences across the incinerator site. This illustrates why epidemiological studies are essential; simply using the raw data and failing to take into account lots of other factors risks confusion between correlation and causation.

32. Another confounding factor is the age of the incinerator. This is because air pollution control is now significantly better in modern well-designed incinerators so results from earlier incinerators need correct interpretation. This is illustrated by examining data from a relatively early French study before going on to examine a wide range of studies, mainly from other countries.

4.2: French Health Risk Assessment

33. Some of these difficulties were overcome in a major study reported by the French Institute for Public Health Surveillance[28] whose objective was to analyze the relation between cancer risk and past exposure to MSW Incinerators (MSWI) for the populations living near them. This ecological retrospective incidence study examined cancer cases diagnosed during past (1990-1999); population exposure to MSWI was estimated only as a function of the geographic zone of residence. It followed extensive coverage in the French media that alerted the authorities and the population to the possibility of an increased cancer risk among people living in the vicinity of incinerators (egg Gilly-sur-Isère, Cluny, Maincy, and Nivillac) which exhibited significant pollution, though measurements were spasmodic. Dioxin emissions from a MSWI and risk of non-Hodgkins lymphoma showed an excess risk of non-Hodgkins lymphoma in the cantons (rural administrative subdivisions) exposed to emissions from the local incinerator. This was the rationale for further study of exposure through incinerator dust and gases to dioxins and of the long-term effect of low doses on local residents, especially as previous (French) studies had been inconclusive. The authors recognised that other pollutants emitted by incinerators might also be involved, including heavy metals, PAHs and dust.

34. The geographic zones used as statistical units were census blocks, called IRIS (*Ilots Regroupés pour l'Information Statistique*). A rich set of social and demographic information was available for every block, each of which has a relatively homogenous population of approximately 2000 inhabitants. Five possible confounding factors mentioned in the literature could therefore be taken into account: urban density, the urban or rural character of the place of residence, socioeconomic status, airborne traffic pollution and industrial pollution. Health data were collected for 1 January 1990 to 31 December 1999. The study area included four districts: Isère, Haut-Rhin, Bas-Rhin, and Tarn which had general cancer registries old enough to cover the study period. Some 16 incinerators had emitted pollutants even before this period and this earlier emission period corresponded to the period of local population exposure. It was defined to make the subsequent development of cancer plausible. This exposure period ranges from 1972 at the earliest to 1985, as a function of emission dates for these different incinerators. With few emission measurements available, MSWI emissions were estimated as precisely as possible to characterize exposure levels retrospectively, based on the judgment of experts, taking the technical characteristics of each incinerator into account. The study team simulated the dispersion of each incinerator's plume by computer, taking account of meteorological and topographic indicators (roughness, relief) using second-generation Gaussian models.

35. The cancer incidence rates observed in the census blocks were related to the expected reference incidence rate from cancer registry. The authors compared the standardized incidence rates obtained in the census blocks with the highest, intermediate, and lowest exposure levels. Excess risks could thus be calculated according to exposure. Overall, the study analyzed 135 567 cases of cancer in 2272 census blocks. There was an indication of a statistically significant linear

exposure/risk relation for some cancer sites. The excess risk for persons living in highly exposed census blocks compared with those living in slightly exposed blocks was 6.8% for liver cancer, 1.9% for non-Hodgkins lymphoma, 9.1% for soft-tissue sarcoma, 2.8% for all cancers in women, and 4.9% for breast cancer. On the other hand, we found no statistical relation for lung cancer or bladder cancer (see Table 2).

36. The study used precise exposure measurements with many data points collected and analyzed and took account of the potential confounding factors that could be measured at a collective scale. Also noteworthy is the precision and reliability of the health data collected, due to the cooperation of the cancer registries and the geo-referencing of cases. Naturally, a study of this type cannot establish a causal relation between exposure to incinerator fumes and the various cancers. While the excess risks measured are relatively low, the study also establishes a linear exposure/risk relation, which is compatible with causality. The authors claim that this was the first study to show such a result for breast cancer.

Table 2: Excess risk of cancer, by site, for inhabitants of census blocks with intermediate and high exposure, compared with residents of slightly exposed census blocks

	Excess risk for residents of census blocks with intermediate exposure (50_{th} percentile) compared with the 2.5_{th} percentile.	**Excess risk for residents of census blocks with high exposure (90_{th} percentile) compared with the 2.5_{th} percentile**	**P values**
Liver cancer (both sexes)	6.8% (0.1–14.1)	9.7% (0.1–20.3)	p<0.05
Malignant non-Hodgkins lymphoma (both sexes)	1.9% (0.0–3.8)	8.4% (0.2–17.2)	p<0.05
Soft-tissue sarcoma (both sexes)	9.1% (-1.7–20.9)	12.9% (-2.3–30.6)	p=0.1
All cancers in women	2.8% (0.7–5.1)	4.0% (0.9–7.2)	p<0.05
Breast cancer in women	4.8% (2.0–7.7)	6.9% (2.9–11.0)	p<0.05

37. The authors properly stressed that the risk detected reflects old exposure situations—from 1972 through 1985—not currently transposable because of the major reduction in incinerator emissions since the 1990s. While the authors indicate that further analysis would be needed before the results can provide guidelines for risk management, they do indicate the reality of health effects.

38. The crucial point from a modern perspective is that the intermediate blocks show raised incidence of the various cancers examined. This means that such affects were seen at a distance from the incinerator when the plume had undergone significant dispersion. Such dispersion reduces concentrations of pollutants in the atmosphere and is therefore an analogue for the effect of modern air pollution control equipment in the sense that conditions at ground level some way downwind are similar to those much closer to a modern incinerator with state-of–the-art sir pollution control equipment. In other words, past results properly interpreted are relevant to today's situation and cannot be simply dismissed.

4.3: Cardiopulmonary Mortality and Fine Particulate Pollution

39. Health effects are determined by the number and size of particles and not the weight. Measurements of the particle size distribution by weight will give a false impression of safety due to the higher weight of the larger particulates. The British Society for Ecological Medicine[29] (BSEM) cites numerous studies into the health effect of particulates which come from a range of sources from around the world. Episodes of increased particulate pollution have been associated with increased cardiovascular mortality[30,31,32,33,34,35,36] and increased respiratory mortality[33,37].

BSEM note that about 150 time-series studies around the world have shown transient increases in mortality with increases in particulates and that cohort studies have shown a long-term effect on mortality[28,29,31].

40. It is possible to quantify this excess mortality? For PM10s, it has been estimated that the increased mortality works out as about a 0.5-1% increase in mortality for each 10µg per cubic metre rise in PM10s[38] for acute exposures and a 3.5% rise for chronic exposures[39]. For PM2.5s the increase in mortality is much greater, especially for cardiopulmonary mortality (see Table 3) which derives from four major studies in the USA. Together these show that fine particulates have been associated with both respiratory and cardiovascular disease and with lung cancer. This is consistent with the discussion in the Harmful Emissions chapter..

Table 3: increasing mortality with increasing levels of $PM_{2.5}$ pollution

Study	Ref & Year	No of Participants	Follow up	Adjusted excess c/p mortality	Difference in $PM_{2.5}$s in µg/m^3	Adjusted excess c/p mortality for rise of 10µg/m^3
Six Cities	28 1993	8,111	1974-1991	37%	18.6	19.8%
ACS Cancer Prevention II	29 1995	552,138	1982-1989	31%	24.5	12.7%
Cancer Prevention II	31 2002	500,000	1982-1998	9%	10	9%
Women' Health Initiative (WHI)	40 2007	65,893	1994-2002	76%	10	76%

41. Two large cohort studies in the USA (the Six City Study, and the American Cancer Society (ACS) study, drawn from the Cancer Prevention II study) showed increasing mortality with increasing levels of $PM_{2.5}$ pollution. In both studies the strongest correlation was between lung cancer and smoking. However, after adjusting for smoking and other variables they showed that higher fine particulate pollution was associated with increased all-cause mortality and with increased cardiopulmonary mortality. In the six cities study, after allowing for smoking and individual factors, mortality rates showed highly significant associations ($p<0.005$) with the levels of fine particles and sulphate particles in the cities, with the most polluted city giving an adjusted all-cause mortality rate of 1.26 compared to the least. This related to a PM2.5 difference of 18.6µg per cubic metre: cardiopulmonary mortality was increased by 37% and lung cancer mortality was also 37% higher.

42. In the ACS study, 552,138 adults (drawn from the Cancer Prevention II study) were followed from 1982 to 1989 and deaths analysed against mean concentrations of sulphate air pollution in 1980 and the median fine particulate concentration from 1979-1983, both obtained for each participant's area of residence from EPA data. This study is particularly important because it didn't simply match death certificates with pollution levels; it actually examined the characteristics (race, gender, weight and height) and lifestyle habits of all 552,138 people. Thus the study was able to rule out confounding factors of tobacco smoking (cigarettes, pipe and cigar); exposure to passive smoke; occupational exposure to fine particles; body mass index (relating to a person's weight and height); and alcohol use. While the strongest correlation was between lung cancer and smoking, both pollution measures showed highly significant association with all-cause mortality and with cardiopulmonary mortality: sulphates were also associated with lung cancer. After adjusting for smoking and other variables, higher fine particulate pollution was associated with a 17% increase in all-cause mortality and a 31% increase in cardiopulmonary mortality for a 24.5 µg per cubic metre difference in PM2.5s.

43. This study also controlled for changes in outdoor temperature. It found that fine-particle pollution was related to a 15% to 17% difference in death rates between the least polluted cities and the most-polluted cities.

44. The ACS results are highly significant and led the EPA to place regulatory limits on $PM_{2.5}$s, establishing the National Ambient Air Quality Standards in 1997 (BSEM op cit). These regulations were challenged by industry but ultimately upheld by the US Supreme Court after the data from all the studies had been subjected to intense scrutiny including an extensive independent audit and a re-analysis of the original data. These regulations have resulted in significant economic benefits; for example, a White House report from the Office of Management and Budget in September 2003 calculated the benefits in terms of reductions in hospitalizations, premature deaths and lost working days as between $120 and $193 billion over the last 10 years. This data implies that incinerators and all other major sources of $PM_{2.5}$ particulates will generate substantial health costs as well as increasing mortality[5].

45. An analysis published in 2002 of the Cancer Prevention II study participants linked the individual factors, pollution exposures and mortality data for approximately 500,000 adults as reported in the ACS study above, bringing the follow-up to 1998[41]. The report doubled the follow-up period and reported triple the number of deaths, a wider range of individual factors and more pollution data, concentrating on fine particles. Although smoking remained the strongest factor associated with mortality, fine particulate pollution remained significantly associated with all-cause and cardiopulmonary mortality and, after the longer follow-up period, fine particulates were significantly associated with lung cancer mortality

46. BSEM (*op cit*) also review studies that reanalysed both the Six City and ACS studies, finding that the increased cardiopulmonary mortality associated with particulate pollution was primarily due to cardiovascular disease. Similarly, more detailed examination of the causes of death in the Cancer Prevention II Study to look for clues to possible patho-physiological mechanisms found the link was strongest with ischaemic heart disease[xiv]: a 10µg per m^3 increase in $PM_{2.5}$ was associated with an 18% increase in deaths from ischaemic heart disease (22% in never smokers).

47. A more recent prospective study, the Women's Health Initiative (WHI), followed 65,893 postmenopausal women (initially free of cardiovascular disease) over 6 years, to examine the effects of the fine particulate pollution in the neighbourhood of each participant on the first cardiovascular or cerebrovascular incident and on mortality. It found consistent results for mortality and morbidity: each increase of 10µg per m^3 in fine particulate pollution was associated with a 76% increase in deaths from cardiovascular disease and an 83% increase in deaths from cerebrovascular disease[42]. The effect was independent of other variables but obese women and those who spent more time outdoors were more vulnerable to the effect. WHI involved a more homogeneous study population and had a number of other methodological advantages over the earlier studies, resulting in greater sensitivity, and more reliable estimates[5]. However, part of the greater effect in this study may be due to gender: there has been some evidence in other studies that women are more susceptible to the cardiovascular effects of fine particulates than men[5].

48. BSEM (*op cit*) extrapolate these results by assuming that the risk to men would be half as great as for women. With this assumption, if an incinerator increased $PM_{2.5}$ particulates by as little as 1µg per m^3 then cardiovascular mortality would be increased by 5-10% with a similar increase for cerebrovascular mortality.

[xiv] **Ischaemic** or **ischemic heart disease** (IHD), or **myocardial ischaemia**, is a disease characterized by reduced blood supply to the heart muscle, usually due to coronary artery disease (atherosclerosis of the coronary arteries).

49. Acute myocardial infarctions have been found to rise during episodes of high particulate pollution, doubling when levels of $PM_{2.5}$ were 20-25µg per m^3 higher and particulates also increased mortality from stroke (BSEM, *op cit*). A recent study found that each 10µg per m^3 rise in PM_{10} particulates was associated with a 70% increase in Deep Vein Thrombosis (DVT) risk[43] Other studies found that mortality from diabetes and admissions for diabetic heart disease are also increased and these were double the non-diabetic CHD admissions, suggesting that diabetics were particularly vulnerable to the effect of particulate pollution. Higher levels of particulates have been associated with life-threatening arrhythmias, exercise-induced ischaemia, excess mortality from heart failure and thrombotic disease. They have also been associated with increased hospital admissions with asthma and with COPD, increases in respiratory symptoms, higher incidence of asthma, reduced immunity, higher rates of ear, nose and throat infection, loss of time from school in children through respiratory disease and declines of respiratory function[5].

50. BSEM (*op cit*) report many studies showing an association between episodes of increased particulate pollution and increased cardiovascular mortality and increased respiratory mortality. They report that about 150 time-series studies around the world have shown transient increases in mortality with increases in particulates and that cohort studies have shown a long-term effect on mortality. They report estimates approximately a 0.5-1% increase in mortality for each 10µg per m^3 rise in PM_{10} for acute exposures and a 3.5% rise for chronic exposures. For $PM_{2.5}$ the increase in mortality is much greater, especially for cardiopulmonary mortality (see Table 3 above).

51. In the Six Cities and ACS studies most of the cardiopulmonary deaths due to particulates were cardiovascular rather than pulmonary with increases in deaths being 5-7 times greater for cardiovascular causes. The ACS data showed that the excess cardiovascular deaths were primarily due to an 18% increase in deaths from ischaemic heart disease for each 10µg per m^3 rise in $PM_{2.5}$s. The WHI study found an even stronger statistical relationship between raised levels of fine particulates and cardiovascular deaths with a 76% increase in cardiovascular mortality for each 10µg per m^3 increase in $PM_{2.5}$ particulates, and this depended not just on which city a woman lived in but in which part of that city. This study, more than any other, demonstrates the great dangers posed by fine particulates and the highlights the urgent need to remove major sources of these pollutants.

52. According to Professor Howard[44] (op cit), successive studies have concluded there is no threshold, i.e. no level of fine-particle pollution below which no deaths occur. The ACS researchers have found that even air pollution levels that are well within legal limits are killing people, especially older people and those with chronic heart and lung ailments.

53. Professor Howard (op cit), reports that a large number of studies confirm that fine-particle pollution is responsible for, or exacerbating, a wide range of human health problems, including:
    - initiating and worsening asthma, especially in children;
    - increasing hospital admissions for bronchitis, asthma and other respiratory diseases;
    - increasing emergency hospital visits for respiratory diseases;
    - reducing lung function (though modestly) in healthy people as well as (more seriously) in those with chronic diseases;
    - increasing upper respiratory symptoms (runny or stuffy nose; sinusitis; sore throat;
    - wet cough; head colds; hay fever; and burning or red eyes);
    - increasing lower respiratory symptoms (wheezing; dry cough; phlegm; shortness of breath; and chest discomfort or pain); and
    - increasing heart disease.

54. The general thesis that airborne particulates and harm to human health are inextricably linked is supported by a wide range of papers (c.f. Pope et al[45]). To take a sample:

- L M Brown[46] and his colleagues have pointed out that "*long-term exposure to even low concentrations of fine particles may be associated with reduced life expectancy*" Airborne particles are classified according to their size.
- The Environmental Protection Agency[47] cites health studies indicating that particles smaller than 2.5 micrometers ($PM_{2.5}$) are "the major contributor to serious health problems like respiratory illness and premature mortality.
- The recently released Paris Appeal Memorandum, supported by the European Standing Committee of Doctors (representing 2 million doctors), urged a moratorium on building any new incinerators[48].

55. According to Dearden[49]) it is now established beyond reasonable doubt that particulate air pollution causes death by various means. Research shows these include:

1. Cardiovascular morbidity and mortality [Miller et al[50]]
2. Cardiopulmonary mortality [Pope et al[51]]
3. Respiratory, immunological, haematological, neurological and reproductive / developmental problems, sometimes with long time-lags between exposure and health effects [Curtis et al[52]]
4. Every 10 $\mu g/m^3$ increase in fine particulate levels was associated with a 4% increase in deaths from all causes, a 6% increase in deaths from cardiopulmonary illness and an 8% increase in lung cancer mortality [Pope et al[53]]
5. There is particular concern about the effects of particulate pollution on infants. Increases in infant deaths from respiratory causes with a 10 $\mu g/m^3$ increase in PM2.5s have been identified [Woodruff et al[54]]
6. A 10 $\mu g/m^3$ increase in PM2.5s was related to a 5% increase in the risk for wheezing bronchitis [Pino et al[55]]

56. Some of the studies reviewed above consider particulates from a range of sources but even their findings clearly have implications for the health effects of emissions from incinerators. Indeed, as incinerators selectively emit smaller particulates and cause a greater effect on levels of $PM_{2.5}$s than PM_{10}s, they would be expected to have a significant impact on cardiopulmonary mortality, especially cardiovascular mortality (BSEM, *op cit*). However, this has not so far been studied directly.

57. The results of the American studies discussed above were confirmed in a review of air pollution under the European Commission (Clean Air for Europe: CAFÉ) assisted by the WHO. This led to the Commission declaring in the *Thematic Strategy on Air Quality*[56] that "*serious air pollution impacts persist*".

58. The European Environment Agency have emphasised the importance of particulate matter (PM) report[57] "*Air quality in Europe — 2011 report"*. They wrote:

> *Epidemiological studies attribute the most severe health effects from air pollution to PM and, to a lesser extent, ozone. For both pollutants, no safe level has been identified. Even at concentrations below current air quality guidelines they pose a health risk (WHO[58]).*
>
> *Health effects of fine particulate matter ($PM_{2.5}$) are caused after their inhalation and penetration into the lungs. Both chemical and physical interactions with lung tissues can induce irritation or damage. The smaller the particles, the further they penetrate into the lungs. PM's mortality effects are clearly associated with the $PM_{2.5}$ fraction, which in Europe*

represents 40–80 % of the PM_{10} mass concentration in ambient air. However, the coarser 2.5–10 µm fraction of PM_{10} also has health impacts and affects mortality. Although evidence is growing that $PM_{2.5}$ is perhaps a greater health concern, ambient air quality measurements and emissions data are often only available for PM_{10} at present.

The current levels of PM exposure experienced by most urban and rural populations have harmful effects on human health. Chronic exposure to particulate matter contributes to the risk of developing cardiovascular and respiratory diseases, as well as lung cancer. Mortality associated with air pollution is about 15–20 % higher in cities with high levels of pollution compared to relatively cleaner cities. In the European Union, average life expectancy is 8.6 months lower due to exposure to PM2.5 resulting from human activities (WHO[59], 2008).

59. According to Professor C. V Howard[xv] (op cit), epidemiological studies worldwide have consistently demonstrated links between ambient particulate matter exposure (PM_{10} and $PM_{2.5}$) and adverse health outcomes, including increased rates respiratory and cardiovascular illness, hospitalizations, and pre-mature mortality[60,61]. He also states that successive studies have concluded there is no threshold, i.e. no level of fine-particle pollution below which no deaths occur. The ACS researchers have found that even air pollution levels that are well within American legal limits (stricter than those in the UK) are killing people, especially older people and those with chronic heart and lung ailments.

60. The Commission also said "*currently in the EU there is a loss in statistical life expectancy of over 8 months due to PM2.5 in air, equivalent to 3.6 million life years lost annually*". The thematic strategy shows that even with effective implementation of current policies this will reduce only to around 5.5 months (equivalent to 2.5 million life years lost or 272,000 premature deaths).

4.4: Regulatory Approaches

61. It is interesting to compare the EPA National Ambient Air Quality Standards (Table 4) with proposals in the UK. The National Ambient Air Quality Standard for $PM_{2.5}$ particulates was introduced into the USA in 1997 with a mean annual limit of 15µg per cubic metre. This had measurable health benefits.

Table 4: USA National Ambient Air Quality Standards

Pollutant	Type	Standard	Averaging Times
PM_{10}	Primary and Secondary	150 µg/m³	24-hour
$PM_{2.5}$	Primary and Secondary	35 µg/m³	24-hour
$PM_{2.5}$	Primary and Secondary	15 µ g/m³	annual

Source: http://en.wikipedia.org/wiki/National_Ambient_Air_Quality_Standards
These may also be seen (in slightly different format) at http://www.epa.gov/air/criteria.html

62. The European Union air quality (AQ) management regime started in 1980 with Directive 80/779/EEC, which set air quality limit values (AQLVs) and guide values for SO2 and suspended particulates. Later Directives set limit values for lead, nitrogen dioxide, and ozone. The 1996 Air Quality Framework Directive[62] (AQFD) and its daughter Directives are aimed at establishing a harmonized structure for assessing and managing AQ throughout the EU.[xvi] .European limits are less stringent than those in the USA, as shown in Table 5.

[xv] Professor C. Vyvyan Howard MB. ChB. PhD. FRCPath.
[xvi] The role of the European Commission includes oversight of the implementation of the EU legislation, including enforcement action if a Member State has not complied with its obligations under the EC Treaty

63. Table 6 shows the EU PM_{10} and $PM_{2.5}$ limit and target values for health protection in more detail. The deadline for Member States to meet the PM_{10} limit values was 1 January 2005. The deadline for meeting the target value for $PM_{2.5}$ (25 µg/m3) was 1 January 2010, while the deadlines for meeting the other limit and 'obligation' values for $PM_{2.5}$ (20 µg/m3) are 2015 or 2020.

64. Table 7 shows the World Health Organisation (WHO guidelines for PM emissions taken from the WHO Air Quality Guidelines (AQG). Like the EPA National Ambient Air Quality Standards, they are stricter than the EU air quality standards. The WHO (2008) explains the reasoning behind its limit values as follows:

"*The 2005 AQG set for the first time a guideline value for particulate matter (PM). The aim is to achieve the lowest concentrations possible. As no threshold for PM has been identified below which no damage to health is observed, the recommended value should represent an acceptable and achievable objective to minimise health effects in the context of local constraints, capabilities and public health priorities*."

65. According to BSEM, an annual mean limit for $PM_{2.5}$ particulates was to be introduced into Scotland in 2010 of 12µg per cubic metre. Likewise, they state that an annual mean target for $PM_{2.5}$ particulates is to be introduced into the UK in 2020 and this will be 25µg per cubic metre. Why the difference is so vast when the science is the same?

Table 5: European limits - Particulate matter (PM_{10}/$PM_{2.5}$)

	24-hour average PM_{10}	Annual average PM_{10}	Annual average $PM_{2.5}$ (1)
Upper assessment threshold	70 % of limit value (35 µg/m3, not to be exceeded more than 35 times in any calendar year)	70 % of limit value (28 µg/m3)	70 % of limit value (17 µg/m3)
Lower assessment threshold	50 % of limit value (25 µg/m3, not to be exceeded more than 35 times in any calendar year)	50 % of limit value (20 µg/m3)	50 % of limit value (12 µg/m3)
(1) The upper assessment threshold and the lower assessment threshold for $PM_{2.5}$ do not apply to the measurements to assess compliance with the $PM_{2.5}$ exposure reduction target for the protection of human health.			

Source: Directive 2008/50/EC of the European Parliament and of the Council of 21 May 2008 on ambient air quality and cleaner air for Europe
http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:2008:152:0001:0044:EN:PDF

Table 6: Air Quality Limit and Target Values for PM_{10} and $PM_{2.5}$ in the Air Quality Directive

Size fraction	Averaging period	Value	Comments
PM_{10}, limit value	One day	50 µg/m³	Not to be exceeded on more than 35 days per year. To be met by 1 January 2005
PM_{10}, limit value	Calendar year	40 µg/m³	To be met by 1 January 2005
$PM_{2.5}$, target value	Calendar year	25 µg/m³	To be met by 1 January 2010
$PM_{2.5}$, limit value	Calendar year	25 µg/m³	To be met by 1 January 2015
$PM_{2.5}$, limit value (a)	Calendar year	20 µg/m³	To be met by 1 January 2020
$PM_{2.5}$, exposure concentration obligation (b)		20 µg/m³	2015
$PM_{2.5}$ exposure reduction target (b)	0–20 % reduction in exposure (depending on the average exposure indicator in the reference year) to be met by 2020		

Note: (a) Indicative limit value (Stage 2) to be reviewed by the Commission in 2013 in the light of further information on health and environmental effects, technical feasibility and experience of the target value in Member States.

(b) Based on a three-year average.

Source: EU, 2008c, Directive 2008/50/EC of the European Parliament and of the Council of 21 May 2008 on ambient air quality and cleaner air for Europe, OJ L 152, 11.6.2008 (http://eur-lex.europa.eu/LexUriServ/ LexUriServ.do?uri=OJ:L:2008:152:0001:0044:EN:PDF)

Table 7: WHO air quality guidelines for PM

µg/m3	24-hour mean	Annual mean
$PM_{2.5}$	25	10
PM_{10}	50	20

Source: European Environment Agency report Air quality in Europe — 2011 report

66. Annexes 2 and 3, drawn from DEFRA, give more details of the regulatory background and the emission standards in the UK. (These Annexes also appear in the Harmful Emissions chapter).

4.5: Conclusions - Fine Particulate Pollution

67. So far as PM_{10} and $PM_{2.5}$ particles are concerned, epidemiological evidence clearly shows that there is good scientific evidence of harm to human health. However, while harm as a result of incineration is clearly established, the extent of risk and the regulatory interpretation and response of this evidence is not agreed, with European standards falling short of best practice (EPA and WHO). Add to this the probability of synergistic effects between the particles, especially $PM_{2.5}$ and below, and other pollutants and the conditions in which the Precautionary Principle should be applied are clearly satisfied.

4.6: Health Effects of Ultrafine Particles

68. There are no set limits for particles smaller than $PM_{2.5}$ yet the evidence shows that they may be considerably more harmful than the larger particles. This is partly due to the fact that they provide a vector for other pollutants from the incinerator emissions to enter directly into the body (see Chapter on Harmful Emissions) but also due to their greater penetration into the body. Howard (op cit) states that there is no longer any serious doubt that the size of the particles is the most important issue from a public health viewpoint and that the reasons are obvious when the respiration of particles is considered in more detail:

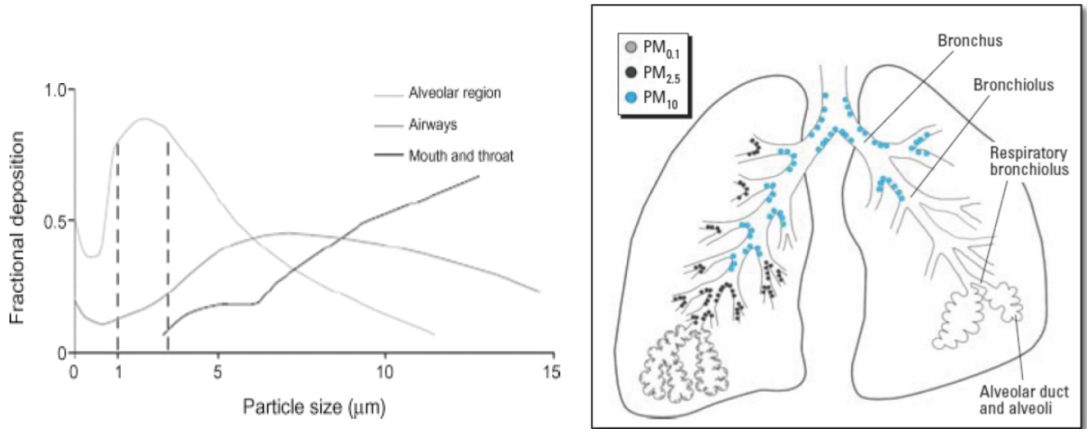
- Particles larger than 10 µm (10 millionths of a metre) generally get caught in the nose and throat, never entering the lungs.
- Particles smaller than 10 µm (PM_{10}) can get into the large upper branches just below the throat where they are caught and removed (by coughing and spitting or by swallowing).
- Particles smaller than 5 µm (PM_5) can get into the bronchial tubes, at the top of the lungs.
- Only particles smaller than 2.5µm ($PM_{2.5}$) in diameter can get down to the deepest (alveolar) portions of the lungs where gas exchange occurs between the air and the blood stream, oxygen moving in and carbon dioxide moving out[63]. Whilst PM ≥ 10µm in diameter enter the nose and mouth only the thoracic fraction, PM_{10}, passes the larynx and penetrates the trachea and bronchial regions of the lung, distributing mainly at pulmonary bifurcations. The respirable fraction, PM2.5, and ultrafine PM, $PM_{0.1}$, deposit deep within the lungs.

69. Figure 1 (drawn from Howard, *op cit*) shows their deposition graphically and illustrates where they are deposited in the lung by particle size.

70. The smallest particulates, particularly the ultrafine particulates (PM0.1) are highly chemically reactive, a property of their small size and large surface area[64]. A further danger of the smallest particulates is that there are thousands more of them per unit weight. In incinerators heavy metals, dioxins and other chemicals can adhere to their surface[65] increasing their toxicity. The body does

not have efficient mechanisms for clearing the deeper part of the lung as only a tiny fraction of natural particles will be as small as this.

Figure 1 Deposition of Particles by Size



71. Removal of the smaller particles (<2.5 μm) deposited in the alveoli is difficult. If soluble in water, they pass directly into the blood stream within minutes. Howard reports that if insoluble, they are collected by scavenging cells called macrophages, which transport them to lymph nodes where they are retained for months or years (NRC, 1979). However, lung macrophage cells seem to have difficulty in recognising the smaller UFPs (those <65 nm), so may let some of them through the lung epithelium, especially during episodes of high numbers. Once they penetrate the epithelium and enter the blood stream, UFPs may be transported around the body and potentially be absorbed into cells – a process called endocytosis. UFPs can cross biological membranes, in common with many viruses, and their mobility within the body is thought to be high.

72. This, coupled with a wide range of health-related issues identified by Howard (e.g. UFPs have access to the blood circulation; induce more oxidative stress than fine particles; cause more pro-inflammatory responses than larger particles; have greatly enhanced toxic potential due to their free location and movement within cells, which promote interactions with intracellular proteins and organelles and even the nuclear DNA; adversely affect cardiac functions and vascular homeostasis; affect the immune system) explains why interest has also focused on the fraction of ultrafine particles (UFP) with a diameter less than 0.1 μm,

73. These $PM_{0.1}$s are abundant in number but contribute little to the mass. UFPs are produced in great numbers by incinerators[66]. While they have been less studied than $PM_{2.5}$ and PM_{10} particulates, there has been enough data available for the WHO to conclude that they produce health effects immediately, after a time lag and in association with cumulative exposure[1]. BSEM[1] also report studies that show that ultrafine particles:

- Have a more marked effect on cardiovascular mortality than fine particulates, with a time lag of 4-5 days[67]. Stroke mortality has been positively associated with current and previous day levels of ultrafine particulates and this has occurred in an area of low pollution suggesting there may be no threshold for this effect.
- Are more potent than other particulates on a per mass basis in inducing oxidative stress in cells and they have the ability to cross the blood-brain barrier and lodge in brain tissue.

74. These results led BSEM to conclude that ultrafine particles represent another largely unknown and unexplored danger of incineration. There is sufficient evidence that they pose a significant threat to human health but this has yet to be quantified. This is clearly a situation in which the

Precautionary Principle must apply, especially when it is recalled that AWRP will have no requirement to monitor this fraction of the particulate emissions which are the most prone to enter the human (and animal) body and can act as vectors for other toxins,

4.7: Assessment by the World Health Organisation and Other Authorities

75. Based on the World Health Organisation Air Quality Guidelines[68], a 1µg per m^3 increase in $PM_{2.5}$ particulates (a very conservative estimate of the level of increase expected around large incinerators) would lead to a reduced life expectancy of 40 days per person over 15 years (this equals a reduction of life expectancy of 1.1 years for each 10µg per m^3 increase in $PM_{2.5}$ particulates). Although this figure appears small, BSEM (*op cit*) note that the public health implications are large and the effect on a typical population of 250,000[xvii] surrounding an incinerator would be a loss of 27,500 years of life over a 15 year time period. While this figure gives an indication of the likely loss of life from any major source of $PM_{2.5}$ particulates, incinerators normally operate for much longer periods than 15 years so this estimated loss of life is likely to be an underestimate since it is from particulates alone and not from other toxic substances.

76. The European Respiratory Society[69] has published its concern about the mismatch between European Union policy and the best scientific evidence. They state that a reduction in the yearly average $PM_{2.5}$ particulates to 15µg per m^3 would result in life expectancy gains at age 30 of between 1 month and 2 years and point out that the benefits of implementing stringent air pollution legislation would outweigh the costs. BSEM (op cit) view their recommendations as sensible and based on sound science. Manifestly, building incinerators such as the AWRP EfW (incinerator) plant would achieve the opposite: they would increase particulate pollution and reduce life expectancy.

77. Statements by leading researchers include:

- "*the magnitude of the association between fine particles and mortality suggests that controlling fine particles would result in saving thousands of early deaths each year*" (Schwartz[70]) and
- "*There is consistent evidence that fine particulates are associated with increased all cause, cardiac and respiratory mortality. These findings strengthen the case for controlling the levels of respiratory particulates in outdoor air*" (Samet et al[71]).

4.8: Particulates – A Summary

78. There is now robust scientific evidence on the dangers to health of fine and ultrafine particulates and of the substantial health costs involved. Recent studies have shown the risk to be considerably greater than previously thought. As According Dearden (op cit) says it is now established beyond reasonable doubt that particulate air pollution causes death by various means. For these reasons it is impossible to justify increasing levels of these particulates still further by building incinerators or any other major source of $PM_{2.5}$ particulates. The data makes it quite clear that attempts should be made to the reduce levels of these particulates whenever possible.

79. There are uncertainties in the data, especially for ultrafine particles and the Precautionary Principle should be applied. **In the case of the AWRP EfW (incinerator) plant, we contend that this**

[xvii] The population of York is 174,400 (http://www.cityofyork.com/econfact/stats.htm). In mid 2009 the estimated resident population of the Harrogate district was 157,900 (http://www.harrogate.gov.uk/Pages/harrogate-4072.aspx).

means refusing planning permission, especially as there is no need for this plant and cleaner and cheaper alternatives exist.

4.9: Cancer - Epidemiological Evidence

80. Carcinogenicity is a recurring theme among many of the pollutants emitted by incinerators and epidemiological studies have been carried out on this subject. Unsurprisingly, they do not provide proof but they do provide strong support for the excess of cancer deaths close to incinerators. Epidemiological interpretation needed to take account of confounding factors including that in most studies, the incinerators were situated near other sources of pollution and often in areas of deprivation, both of which are associated with higher cancer incidence. Also, many of these studies predate recent emission limits and the populations were therefore likely to be subject to greater pollution than would be the case today. However, as the French study cited above demonstrates, much can be learnt from earlier work.

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

82. Knox[73] found results for childhood cancers around municipal incinerators which were similar to those found earlier for adult cancers. They also found this around hospital incinerators and other large combustion sources. As with previous studies of proximities of childhood cancers to industrial sites (and exposures to pre-natal radiation) the excess cancers were similar for leukaemia and solid tumours of all types. Knox felt that this was to be expected with agents that have systemic access to the DNA/RNA of all types of foetal cells. The childhood cancer excess risk was greater than that for adults; this could be for a number of reasons including other risk factors such as smoking and the longer latency period of some adult cancers as well as results being more influenced by unknown migration within the adult population. The excess had only occurred during the operational period of each incinerator and was also noted round hospital incinerators but not landfill sites (with a few exceptions). This is strong evidence that the incinerators' emissions contributed to the children's cancer deaths.

83. There are, as ever, a number of possible caveats within an epidemiological study and the author considered and largely eliminated each of them. However, he was unable to go further than saying that the excess cancer effects stem from large scale combustion plants as a group of which incinerators are but one component. This academic distinction would serve merely to emphasise that incinerators are not alone in causing excess cancers but are part of a wider group of plant that does so.

84. BSEM (op cit) review a number of overseas studies:

- Biggeri et al. in 1996 compared 755 lung cancer deaths in Trieste with controls in relation to smoking, probable occupational exposure to carcinogens and air pollution (measured nearest to their homes) and the distance of their home from each of four pollution sites. The city centre carried a risk of lung cancer but the strongest correlation was with the incinerator where they found a 6.7 excess of lung cancer after allowing for individual risk factors[74].
- Examination of the incidence of soft tissue sarcoma and non-Hodgkin's lymphoma from French Cancer Registry data in two areas close to an incinerator with high emission of dioxin showed highly significant clusters of soft tissue sarcoma and of non-Hodgkins lymphoma but no clusters of Hodgkin's disease (used as negative control). This study[75] was able to check the association by looking for space time relationships and, by looking for other clusters in the wider area which contained other areas of deprivation, to eliminate deprivation as a factor and to establish the causal link.
- According to Ohta et al[76], Japan built 73% of all the municipal waste incinerators in the world and by 1997 had become very concerned about their health effects: in the village of Shintone, 42% of all deaths between 1985-95 in the area up to 1.2 km to leeward of an incinerator (built in 1971) were due to cancer, compared to 20% further away and 25% overall in the local prefecture. Their data on soil contamination reinforced the importance of considering wind directions in evaluating the health effects of incinerators[5].
- Comba found an increased incidence of soft tissue sarcoma in an Italian population living within 2 km of an incinerator[77]. Zambon et al[78] looked at cases of sarcoma from a different perspective. They calculated dioxin exposure from incinerators and other industrial sources in patients with sarcoma using a dispersion model and found the risk of sarcoma increased with the extent and duration of exposure to dioxin.
- In 1989 Gustavsson[79] reported a twofold increase in lung cancer in incinerator workers in Sweden compared to the expected local rate. In 1993 he reported a 1.5 fold increase in oesophageal cancer in combustion workers, including those working in incinerators[80].

To summarise, incinerators and cancer are found in the UK and around the world and over many years. Some of these studies are quite recent.

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

86. BSEM also feel that the studies may have underestimated the risks. At 13 years, the follow-up period of the large British study was probably too short: at Sint Niklaas adult cancer cases seemed to increase from 13 years onward (although children's cancers occurred earlier), and in Japan, Ohta noted that cancer caused 42% of all deaths in the lee of incinerators from 14 to 24 years after the incinerator was commissioned[81]. The reported risks were higher in the studies in which allowance was made for the direction of prevailing winds, possibly because of dilution elsewhere by relatively unexposed persons.

87. BSEM note that these studies apply to the older incinerators: newer incinerators may have better filters but fine particulates and metals are incompletely removed. They argue that as some of these

pollutants, notably fine particulates, do not appear to have a safe threshold, it is clearly incorrect to claim that incinerators are safe. The higher quantity of toxic fly ash produced by modern incinerators, which is easily wind-borne, represents an additional hazard. Even if incinerators were equipped with perfect filters, their huge size and tendency to faults means that the risk of intermittent high levels of pollution is a real concern.

4.10: Health Effects of Heavy Metals

88. According to the European Environment Agency (*Air quality in Europe — 2011 report, op cit*), the heavy metals arsenic (As), cadmium (Cd), lead (Pb), mercury (Hg) and nickel (Ni) are common air pollutants in Europe, mainly emitted as a result of various industrial activities and combustion of coal. Although atmospheric levels are low, they contribute to the deposition and build-up of heavy metal contents in soils, sediments and organisms. Heavy metals are persistent in the environment and some bio-accumulate in food chains.

89. Arsenic exposure is associated with increased risk of skin and lung cancer. Cadmium is associated with kidney and bone damage and has also been identified as a potential human carcinogen, causing lung cancer. Lead exposures have developmental and neuro-behavioural effects on foetuses, infants and children, and elevate blood pressure in adults. Mercury is toxic in the elemental and inorganic forms but the main concern is associated with the organic compounds, especially methylmercury. It accumulates in the food chain, for example in predatory fish in lakes and seas and reaches humans. Nickel is a known carcinogen and also has other non-cancerous effects, e.g. on the endocrine system. Annex 4 gives more details for a selection of heavy metals.

90. Air pollution is only one source of exposure to these metals but their persistence and potential for long range atmospheric transport means that atmospheric emissions of heavy metals affect even the most remote regions (WHO).

91. Howard (op cit) reports a study by Ogulei[82] which used applied multivariate data analysis methods to a combination of particle size and composition measurements in Baltimore to apportion particulate sources and found that the majority of all the observed Lead (63.4%) and most of the Zn (32.6%) could be attributed to a waste incinerator source. The closest major municipal incinerator to the monitoring site was c. 5 miles away in a direction corresponding to the direction suggested by their analysis. The contribution from this incinerator was about 7.9% which was comparable to the 9.3% contribution that was obtained in their earlier study[83]. The size distribution for this source indicated two modes at 0.02 and 0.15 mm. Whilst the incinerator made approximately the same contribution as both local petrol traffic (8.11%) and coal fired power station (10.34%) the particulate peak was smaller than each of the others and the concentration of heavy metals was much greater in the incinerator particulates.

92. BSEM cite studies that show incinerator emissions to air and ash contain over 35 metals several of which are known or suspected carcinogens. Breathing in air containing toxic heavy metals leads to bioaccumulation in the human body and we have an increasing body burden as we get older. They are difficult to excrete so can remain in the body for years: cadmium has a 30 year half-life. As incinerators emit heavy metals, they contribute to our body burden and can lead to further damage to health.

93. Mercury is a gas at incineration temperatures and cannot be removed by the filters. It is one of the most dangerous heavy metals. It is neurotoxic and has been implicated in Alzheimer's disease[84,85,86], learning disabilities and hyperactivity[87,88]. There have been significant increases in both autism and in rates of special education students around sites where mercury is released into the environment[89,90].

94. Inhalation of heavy metals such as nickel, beryllium, chromium, cadmium and arsenic increases the risk of lung cancer[91]**.** Cumulative exposure to cadmium has been correlated with lung cancer[92]. Supportive evidence comes from Blot and Fraumeni who found an excess of lung cancer in US counties where there was smelting and refining of non-ferrous metals[93]. Inhaled cadmium also correlates with ischaemic heart disease[94].

95. Toxic heavy metals have been implicated in a range of emotional and behavioural problems in children including autism[85], dyslexia[86], impulsive behaviour[87] attention deficit and hyperactivity disorder (ADHD)[95,96]as well as learning difficulties[97,98,99,100], lowered intelligence[89] and delinquency[101], although BSEM say not every study reached standard significance levels. Many of these problems were noted in the study of the population round the Sint Niklaas incinerator[102]. Exposed adults have also been shown to be affected, showing higher levels of violence[103,104], dementia[105] and depression than non-exposed individuals. Heavy metal toxicity has also been implicated in Parkinson's disease[106].

96. Heavy metals emitted from incinerators are usually monitored at 3 to 12 monthly intervals in the stack: this is clearly inadequate for substances with this degree of toxicity. Table 8 shows the limits placed on selected heavy metals by the EU and WHO.

Table 8: Air Quality Limit and Target Values for As, Cd, Ni and Pb regulated by EU, and WHO air quality guidelines

Pollutant	EU target or limit value (a)	WHO AQG
Arsenic	6 ng/m³ (b)	–
Cadmium	5 ng/m³ (b)	5 ng/m³ (d)
Nickel	20 ng/m³ (b)	–
Lead	500 ng/m³ (c)	500 ng/m³

Note: (a) Annual mean, measured as contents in PM_{10}.

(b) Target value, entering into force on 31 December 2012.

(c) Limit value to be met by 1 January 2005. The limit value to be met only by 1 January 2010 in the immediate vicinity of specific industrial sources situated on sites contaminated by decades of industrial activities. In such cases, the limit value until 1 January 2010 is 1.0 µg/m³.

(d) AQG set to prevent any further increase of cadmium in agricultural soil, likely to increase the dietary intake of future generations.

Source: European Environment Agency report Air quality in Europe — 2011 report

5: HEALTH EFFECTS – THE VULNERABLE GROUPS

97. Not all people are equal in response to pollution; some are more susceptible than others. It is important to identify these groups in assessing risk since their dose-response relationship can be far higher than that of average individuals. Also, the approach that AmeyCespa has taken to assessing whether risks are acceptable (we contend they are not) entirely ignores the fact that some groups are far more susceptible than others.

5.1: The Foetus

98. The unborn child (foetus) is uniquely susceptible to toxic damage and early exposures can have life changing consequences for two main reasons (BSEM, *op cit*):

1. Most of these chemicals are fat soluble and the foetus has virtually no protective fat stores until very late pregnancy so the chemicals are stored in the only fatty tissues it has, namely its own nervous system and particularly the brain.

2. Many pollutants are actively transported across the placenta from the mother to the foetus. This occurs with heavy metals which the body mistakes for essential minerals. This is particularly critical for mercury where one tenth of women already have body stores of mercury which can lead to neuro-developmental problems in the newborn[107].

99. Other factors that increase foetal susceptibility are higher rates of cell proliferation, lower immunological competence and decreased capacity to detoxify carcinogens and repair DNA[108]. Safety limits currently do not take into account this increased risk to the foetus; during a narrow window of time, in the first 12 weeks in utero, the foetus's body is affected by miniscule amounts of hormone measured in parts per trillion. Tiny amounts of chemicals can upset this delicate balance and chemicals that are not toxic to an adult can have devastating effects on the newborn. Porterfield[109] has shown that small amounts of chemicals such as dioxins and PCBs, at doses that are not normally regarded as toxic, can affect thyroid hormones and neurological development. A single exposure is enough and timing is critical[110].Small doses of oestrogenic chemicals can alter sexual development of the brain and the endocrine system[111].

100. Small doses of oestrogenic[xviii] chemicals (hormone disrupting chemicals) can alter sexual development of the brain and the endocrine system and that exposure to oestrogenic chemicals affects immunity, reduces the immune response to vaccines, and is associated with a high incidence of middle ear and recurrent respiratory infections[112]. The amount of chemical that the baby takes in relates to the total persistent contaminants that have built up in the mother's fat over her lifetime. This will increase in areas around incinerators. Exposure to fine particulate pollution during pregnancy can have an adverse effect on the developing foetus and lead to impaired foetal growth.

101. In 2005, two studies examined the body burden in the foetus (BSEM *op cit*). A USA study found an average of 200 industrial chemicals and pollutants (out of 413 tested) in the umbilical cord blood of 10 randomly chosen babies. These included 180 carcinogens, 217 chemicals that are toxic to the brain and nervous system and 208 that can cause birth defects and abnormal development in animals. A European study tested for the presence of 35 chemicals in the umbilical cord blood of newborns, finding five hazardous chemicals in all babies and some had as many as 14 different compounds. It is unsurprising that the report questioned the wisdom of allowing the foetus to be exposed to a complex mixture of persistent, bio-accumulative and bioactive chemicals at the most critical stage of life. Clearly incinerators would increase the foetal body burden.

102. BSEM (*op cit*) review five reports of increases in congenital abnormalities around incinerators:
- Multiple birth defects at Sint Niklaas to leeward of the incinerator[113].
- Orofacial defects and other midline defects more than doubled near an incinerator in Zeeburg, Amsterdam[114]. Most of these deformed babies were born in an area corresponding to wind-flow from the incinerator and other defects included hypospadius and spina bifida.
- In the Neerland area, Belgium, there was a 26% increase in congenital anomalies in an area situated between two incinerators[115].

[xviii] **Oestrogens** or **œstrogens**) are a group of steroid compounds, named for their importance in the estrous cycle, and functioning as the primary female sex hormone, their name comes from estrus/oistros (period of fertility for female mammals) + gen/gonos = to generate. Oestrogenic is the adjective. It can mean promoting estrus or of, relating to, caused by, or being an estrogen.

Oestrogenic chemicals are hormone disrupting chemicals and include chemicals emitted by incinerators such as dioxins and PCBs. PCBs are very persistent in the environment while dioxins are persistent and bio-accumulative.

- A study of incinerators in France has shown chromosomal defects and other major anomalies[116] (facial clefts, megacolon, and renal dysplasias).
- A recent British study looked at births in Cumbria between 1956 and 1993 and reported significantly increased lethal birth defects around incinerators after adjusting for year of birth, social class, birth order, and multiple births. The odds ratio for spina bifida was 1.17 and that for heart defects 1.12. There was also an increased risk of stillbirth and anencephalus around crematoriums[117]. The study pointed out that the figures for birth defects are likely to be substantial underestimates since they do not include spontaneous or therapeutic abortions, both increased by foetal anomalies[5].

103. In addition, several studies have noted an increase in birth defects near waste sites, particularly hazardous waste sites. The pattern of abnormalities was similar to the pattern found with incinerators, with neural tube defects often being the most frequent abnormality found, with cardiac defects second. The pattern of damage may be explained by harmful chemicals normally stored in fatty tissue being stored in the only available fatty tissue in the foetus: the brain and nervous system (BSEM, *op cit*).

104. Even very low intakes of dioxins have significant adverse health effects, especially if the intake is in utero or in infancy. Dearden (op cit) has commented that it is the practice of those who support incineration to point out that although breast-fed infants can take in high doses of dioxins, this is only for a few months until the infant is weaned. That argument completely misses the important point that it is in the early months in life (including gestation) that much damage is done by dioxins and other endocrine disruptors, and by neurotoxins such as mercury[118]

5.2: Children's Health

105. Children face a higher health risk from incinerator emissions than adults. Two main reasons for this are:
    1. Children have a relatively faster metabolism than adults and, for example, breathe more rapidly. Thus they take in a greater pollution load relative to body weight than do adults
    2. Children's tissues are developing and are therefore more affected by the same pollutant load than the "static" tissues of adults. Developing systems are very delicate and in many instances are not able to repair damage done by environmental toxicants[119] and there is an age-related difference in neurotoxicity for many substances including heavy metals[120].

106. Children and especially babies are growing rapidly (e.g. a baby doubles its weight in roughly the first four months of life). If cumulative toxins such as heavy metals and dioxins are present, they will be incorporated in the child's body.

107. Breast fed babies take on dioxins and other toxic chemicals through the mother's milk. Indeed, scientists wanting to know absorption of Persistent Organic Pollutants (POPs) in fatty tissues (most POPs are fat-soluble) look at the fat in breast milk[121]. Breast milk carries in it the "body burden" of chemicals a mother has been exposed to and has stored over her lifetime; including pollutants and dioxins which are known to disrupt the endocrine (hormone) system. Six months of breast feeding will transfer 20% of the mother's lifetime accumulation of organochlorines to the child[122]

108. Breast milk also contains PCBs (polychlorinated biphenyls), which along with dioxins affect the nervous, endocrine (hormone) and reproductive systems of animals, and may be carcinogenic. They are found in many food sources, particularly fatty foods such as meat and milk: and of course breast milk Also, babies are exposed to their mothers' toxins *in utero*, i.e. through the

placenta. Recent research found that this prenatal exposure to PCBs has a subtle negative effect on the neurological and cognitive development of children right up to school age.

109. Breastfeeding can counteract any adverse developmental effects caused in the womb, despite current PCB levels in breast milk. That is because breast milk contains antioxidants, which seem to compensate for the toxic effects of the environment. Breast milk also helps babies develop stronger immune systems. So the most harmful effects of toxins are from exposure in the womb, not breastfeeding, and government bodies such as the Ministry of Agriculture, Food and Fisheries conclude that breastfeeding should continue to be promoted and supported. Their view is that "*The potential risk as a result of residual contaminants is far, far outweighed by the clear and proven nutritional, health and other benefits of breastfeeding"*[123]

110. Particulates carry various chemicals including PAHs into the human body. Perera from the Columbia Center for Children's Environmental Health has found that the foetus is 10 times more vulnerable to damage by these substances[124]. Also, PM2.5 particulates have an adverse effect on the developing foetus with significant reductions in weight, length and head circumference and reiterated the importance of reducing ambient fine particulate concentrations[125]. In addition further studies have shown an adverse effect on foetal development at levels currently found in cities today, such as New York[126]. Studies of air pollution in mice have found that it causes irreversible genetic mutations whereas if the mice breathed air which had been freed of particulates by filtration they developed only background levels of genetic mutations, confirming that particulates were causative[5]. At the fourth Ministerial Conference of Environment and Health in June 2004, the WHO announced that between 1.8 and 6.4% of deaths in the age group from 0 to 4 could be attributed to air pollution[127].

- A number of studies show that toxic and carcinogenic exposures in early life, including prenatal exposures, are more likely to lead to cancer than similar exposures later (BSEM *op cit*). At the First International Scientific Conference of Childhood Leukaemia (Sept 2004), Professor Alan Preece suggested that pollutants crossing the placenta, were damaging the immune system and could be linked with soaring rates of leukaemia, which were being initiated in utero. Knox's recent study[128] found that children born in "pollution hotspots" were two to four times more likely to die from childhood cancer. The "hotspots" included sites of industrial combustion, and sites with higher levels of particulates, VOCs, nitrogen dioxides, dioxins and benz(a)pyrenes – just what would be found around incinerators. In most cases, the mother had inhaled these toxic substances and they were then passed on to the foetus through the placenta.

111. BSEM (*op cit*) describe recent studies that found associations between the body burden of mercury and the risk of autism and point out that the study of the Sint Niklaas incinerator found a multitude of problems in children, including learning defects, hyperactivity, autism, mental retardation and allergies and that this is exactly what would be anticipated from the above and research already done on the health effects of heavy metals, PCBs and dioxins on children.

112. Lead can cause decreases in intelligence and alteration of behaviour in the absence of clinically visible signs of toxicity. This is also true of PCBs and methyl mercury. These effects are all the more likely when children are exposed to multiple pollutants, notably the heavy metals, which will be found in the cocktail of chemicals released by incinerators. While this may have only minor implications for an individual it can have major implications for a population[5]. For instance a 5 point drop of IQ in the population reduces by 50% the number of gifted children (IQ above 120) and increases by 50% the number with borderline IQ (below 80)[129]. This can have profound consequences for a society, especially if the drop in IQ is accompanied by behavioural changes.

5.3: Scientific Consensus on Vulnerability of Children

113. Howard (op cit) reports that the WHO and European Commission have recognised that children are specially affected by PM pollution. The WHO *Monograph: the Effects of Air Pollution on Children's health and development: a review of the evidence* [130] reviewed factors affecting children's susceptibility, effects on pregnancy outcomes, infant and childhood mortality, lung function development, asthma and allergies, neuro-behavioural development and childhood cancer. It declared that "*the amount of ill-health attributable to air pollution among European children is high*". The *Children's Environment and Health Action Plan for Europe* (CEHAPE), adopted at the *Budapest Ministerial conference* in June 2004[131] included air pollution in increasing concern about environmental effects on children's health. It agreed that developing organisms, especially during embryonic and foetal periods and early years of life, are often particularly susceptible. Howard states that it is now recognised that the inhibition of children's lung development can be very serious, potentially meaning long term harm to their respiratory health. He stated that evidently air pollutants, most probably including particulates, cause harm to children differently to adults.

114. The expert science view, summarised by Joel Schwartz[132] is that children's exposure to air pollution is of special concern because their immune system and lungs are not fully developed, so many of the epidemiological associations are likely to be causal. Howard cites a review by Heinrich and Slama[133] which found that ambient fine PM is associated with intra-uterine growth retardation, infant mortality; impaired lung function and postneonatal respiratory mortality, but less consistently with sudden infant death syndrome. Hertz-Picciotto et al.[134] [64] found bronchitis in early childhood correlates with $PM_{2.5}$ and PAH levels (Howard points out that UFPs may be a carrier for PAH). In Howard's view, while these findings may not all be conclusive, there can be no doubt that children and even the foetus are particularly vulnerable to particulate air pollutants, This has largely been overlooked in setting current standards and controls.

115. While supporters of incineration typically say that although breast-fed infants can take in high doses of dioxins, this is only for a few months until the infant is weaned, this argument completely misses the important point that it is in the early months in life (including gestation) that much damage is done by dioxins and other endocrine disruptors, and by neurotoxins such as mercury[135] [Newland & Rasmussen 2003].

116. An American review of health effects of poor air quality on children's health[136] emphasised the hazards associated with the siting of major particle-emitting plants and roads in the vicinity of schools or communities containing children.

5.4: The Chemically Sensitive

117. A proportion of the population react to chemicals (e.g. lead) and pollutants (e.g. benzene) at several orders of magnitude below that normally thought to be toxic (Ashford and Miller[137]). BSEM (*op cit*) report studies showing a tenfold difference between different individuals in the metabolism of the carcinogenic PAH benz(a)pyrene. Ashford and Miller also noted that studies in both toxicology and epidemiology have recognised that chemicals are harmful at lower and lower doses and that an increasing number of people are having problems. A significant percentage of the population have been found to react this way (15 to 30% in several surveys with 5% having daily symptoms). Research has shown 150 to 450 fold variability in response to airborne particles[138]. Chemical sensitivity is typically triggered by an acute exposure after which symptoms start to occur at very low levels of exposure. BSEM[5] believe that faults are all too common with modern incinerators leading to discharges of pollutants at levels that endanger

health – giving a very real risk of long-term sensitisation and that certain susceptible individuals will be highly affected by these pollutants. They state that these effects will be difficult to anticipate and that people affected this way are extremely difficult to treat.

5.5 Additional objection: justice for the most vulnerable

118. Waste incineration is unjust because its maximum toxic impact is on the most vulnerable members of our society, the unborn child, children and the chemically sensitive. It gratuitously imposes major risks on these vulnerable groups, far above those of other members of society, and we find this thoroughly objectionable.

6: HEALTH EFFECTS – NEUROLOGICAL AND BEHAVIOURAL EFFECTS

119. Most toxic compounds are preferentially stored in fatty tissue and this includes the brain – making the brain a key target organ for pollutants while ultrafine particulates can carry pollutants across the blood-brain barrier[5]. Heavy metals and compounds such as PCBs and dioxins cause cognitive defects, learning problems and behavioural disturbances in children and these effects occur at levels previously thought to be safe; this suggests that these pollutants also impact on adult brain function[5]. Indeed, some organochlorines, especially those with toxic metabolites and those that dissolve in the cell membranes are known to kill brain cells and even an undetectable annual loss rate of 0.1% of neurones would lead to a major decline in brain function by middle age[5]. A recent study has noted substantial increases in neurological diseases (Alzheimer's disease, Parkinson's disease and motor neurone disease) in the last two decades coupled with earlier onset of these illnesses[139]. Similarly diseases affecting the brain (including ADHD, autism and learning difficulties) have also shown large increases in the young[140] and it is very likely that these diseases have aetiological[xix] factors in common[5]. BSEM[5] note that heavy metal exposure is known to correlate with both Parkinson's disease and Alzheimer's disease and that both diseases have increased dramatically over the last 30 years.

120. Diseases such as Alzheimer's have high care costs and dire effect on both patients and carers. Although multiple factors are probably involved in its causation, there is evidence of a link to heavy metal exposure, making it imperative to reduce our exposure to these toxic metals and other neurotoxic chemicals (BSEM, op cit). To gratuitously and deliberately increase exposure to these pollutants, for example by building incinerators, is unwise.

121. BSEM note that many pollutants pass straight from the nose to the brain where they affect brain function and that air pollution correlates with inpatient admissions with organic brain syndrome, schizophrenia, major affective disorders, neurosis, behavioural disorder of childhood and adolescence, personality disorder, depression and alcoholism and that increases in the total number of psychiatric emergency room visits and in schizophrenia correlate to high air pollution. Additionally, BSEM[5] note studies relating violence and crime to heavy metals and these include lead, cadmium and manganese, with most studies focussing on lead including raised levels of lead in the air. This growing literature should serve as a warning about the dangers of allowing heavy metals to be emitted into the environment and BSEM therefore note that we need to consider the effect of incinerators, not only on health, but on education and on quality of life, including the impact of violence and crime.

[xix] **Aetiology:** The study of the causes (e.g. of a disorder). The word "aetiology" is mainly used in medicine, where it is the science that deals with the causes or origin of disease, the factors which produce or predispose toward a certain disease or disorder.

7: THE SPREAD OF POLLUTION

122. The US National Research Council (an arm of the National Academy of Sciences that was established to advise the US government) found that it was not just the health of workers and local populations that would be affected by incinerators. They reported[141] that populations living more distantly are also likely to be exposed to incinerator pollutants and stated:

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

123. The US National Research Council later commented that the incremental burden from all incinerators deserves serious consideration beyond a local level. This is clearly relevant in the UK context and to AWRP in particular. Crucially, the more toxic smaller particulates, which typically have more toxic chemicals and carcinogens attached, will travel the furthest[142] so pollution from incineration is not just a local issue.

124. A striking example of the unforeseen consequences of releasing pollutants into the air was seen in Nunavut, in the far North of Canada in the Polar Regions. The Inuit mothers here have twice the level of dioxins in their breast milk as Canadians living in the South, although there is no source of dioxin within 300 miles. At the centre of Biology of Natural Systems in Queen's College, New York, Dr Commoner and his team used a computer programme to track emissions from 44,000 sources of dioxin in North America. This system combined data on toxic releases and meteorological records. Among the leading contributors to the pollution in Nunavut were three municipal incinerators in the USA[143,144].

125. The obvious lesson from this American work is that pollution from the proposed AWRP EfW (incinerator) plant could affect people over a much wider area than covered in the AmeyCespa application. **We find this unacceptable and believe it adds to the case for rejecting the planning application.**

8: HEALTH COSTS OF INCINERATION

126. Incineration has both direct costs (e.g. building and operating the plant and the infrastructure to support it) and indirect costs which include the costs of alleviating or treating the harm that it causes. Not least amongst these are the health costs. Unfortunately the environmental, human and health costs of incineration tend to be overlooked because they come out of another budget and are therefore not accounted for in NYCC's business case and are irrelevant to AmeyCespa. Nevertheless these costs will have to be paid for and should therefore be included in the equation; after all it is the taxpayer who ultimately faces them.

127. Incineration creates substantial costs. A 1996 ETSU (Harwell) report for the European Commission suggested that for every tonne of waste burnt there would be between £21 and £126 of health and environmental damage, thus a 400,000 tonnes per year incinerator would cost the tax-payer between £9m and £57m / year[145]. Better emission control means these costs would now be lower but this is offset by the corresponding increase in costs that is now needed to make fly ash safe.

128. Estimates of the health costs of incineration are surprisingly high. DEFRA's report in 2004 found that the health costs from PM_{10} particulates from incinerators alone, using a central to high estimate, would be £39,245 per tonne of particulates emitted[146]. A 400,000 tonne per year incinerator would produce about 24,000kg (24 tonnes) of particulates per year and the DEFRA estimate of health costs would be £941,000 per annum; equivalent cost figures for the EfW (incinerator) plant at AWRP would be around £750,000 per annum.

129. There is, however, a wide range of estimates. DEFRA looked at 13 studies of $PM_{2.5}$ and PM_{10} particulates and noted that the health costs ranged from £2,000-£300,000 per tonne for $PM_{2.5}$ and £1,800-£226,700 for PM_{10}. These estimates were based on modelling data which fail to take account of all the risks and do not take into account recent data demonstrating high levels of pollutants emitted during start-up and shut-down. Thus they are likely to underestimate particulate emissions and the costs are probably towards the upper end of the range. BSEM suggest a total health cost per annum for particulates alone for a 400, 000 per year incinerator of £6.5 million[xx], though this could be on regarded as a maximum on the evidence available The equivalent figure for the EfW (incinerator) plant at AWRP would be £5.3 million. To give a realistic estimate of the health costs of incineration, the additional costs from the other pollutants must be added to this.

130. Eshet's review of health costs of incineration noted the complexity and difficulty of these calculations, with estimates varying between $1.3 and $171 per tonne of waste burnt[147]. A study of British incinerators estimated the cost to be between $2.42 and $13.16 per tonne of waste burnt[148]. As BSEM note, most of these studies do not take into account the cost of ash, the cost of clean-up of accidents or water contamination or the more subtle health effects such as behavioural changes, reduction in IQ, reproductive and hormonal effects which have become apparent in recent years with many pollutants such as lead and organochlorines and suggest that the costs could be considerably higher than estimated. What is clear is that the EfW (incinerator) facility at AWRP would cause millions of pounds worth of health damage annually/ By contrast the health costs of alternative waste technologies such as mechanical biological treatment (MBT), aerobic digestion and plasma gasification have low environmental and health costs. **We therefore believe that makes incinerators a poor choice for waste management**

131. Reducing pollution reduces health costs and this has been demonstrated in a variety of industries in the UK and USA[149,150] For example, a White House study by the Office of Management and Budget in 2003 concluded that enforcing clean air regulations led to reductions in hospitalisations, emergency room visits, premature deaths and lost workdays which led to a saving of between $120 and $193 billion between October 1992 and September 2002 (and this excluded prescription costs and primary care costs**).**

132. This illustrates the scale of costs of health problems arising from the AWRP EfW (incinerator) plant. **In the case of incineration, the best choice is to use an alternative, safer technology;** gratuitously adding to health care costs is unwise, especially where safer alternatives exist.

[xx] BSEM calculate as follows. The Quality of Urban Air Review Group has estimated that the PM~~2.5~~ fraction of total particulates is between 28% and 100%. Leaving aside the likelihood that the PM2.5 fraction is higher from incinerator emissions an average figure of 60% $PM_{2.5}$s would be likely. This calculation therefore estimates that a 400,000 tonne incinerator would produce 24 tonnes of particulates, that 60% would be $PM_{2.5}$ particulates at a cost of £4.32 million per annum and 40% would be at the lower cost for other PM_{10}s costing £2.18 million per annum. The total cost in health damage from particulates would therefore be £6.5 million per annum

9: QUANTIFYING THE HEALTH RISKS FROM INCINERATION

133. Many of the numerous publications on health risks from incineration were reviewed by Thompson & Anthony[151]. Some studies have found significant adverse health effects whilst others have not. Dearden (op cit) explains that there are two acknowledged difficulties here:
    - incinerators are almost invariably built in the vicinity of other polluting industry, so that it becomes very difficult to isolate the contribution of the incinerator.
    - such studies are inevitably retrospective, and deal with plant that has been operating for a considerable time; new plant would be expected to have considerably lower emissions of potential pollutants.

    This explains why not all studies are able to find the same health effects; the situation is quite unlike a laboratory experiment in which confounding variable are controlled for.

134. So the crucial question is what are the health risks associated with a modern MSW incinerator. This question has been answered in a paper by Roberts and Chen[152]. They derived a quantitative measure of risk from a modern waste incinerator, based on current allowed emission levels. The authors calculate that the overall risk of dying in any one year from incinerator emissions is 2.49×10^{-7}, with the main contributors to that risk being cadmium (72%), dioxins (17%), arsenic (10%) and PAHs (1%). The risk of dying from incinerator emissions over the 25 year operating life of an incinerator is 25 times the annual risk, or 6.23×10^{-6}, and the 70-year lifetime risk is 1.74×10^{-5}. As Dearden (op cit) states, both of these values are well above the *de minimis* acceptable lifetime target level of 10^{-6} (i.e. 1 in a million) used by the US Environmental Protection Agency[153] and recommended by the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment[154].

135. While the US EPA target level is for cancer risk alone, the four toxins found by Roberts and Chen to contribute most to mortality from incinerator emissions are all known carcinogens, so the comparison is valid. Dearden points out that for better protection of local communities[155] and through the use of the Precautionary Principle[156], a target level of 1 in a million (10^{-6}) should be used, rather than the 1 in 100,000 (10^{-5}) suggested by those in favour of incineration.

136. These figures demonstrate that even new incineration plant such as the proposed EfW (incinerator) plant at AWRP has the potential to cause serious health risks. This main reasons for this (as stated by Dearden, op cit) are:

    - It is likely that waste incinerators will have dioxin and other emissions much higher than is claimed.
    - Even very low intakes of dioxins have significant adverse health effects, especially if the intake is *in utero* or in infancy. While those who support incineration typically say that although breast-fed infants can take in high doses of dioxins, this is only for a few months until the infant is weaned, this argument completely misses the important point that it is in the early months in life (including gestation) that much damage is done by dioxins and other endocrine disruptors, and by neurotoxins such as mercury (see section 5.3).
    - Compounds such as PCBs often possess dramatically different toxicities at low dose than at high dose[157] because of their potency as enzyme inducers.

137. In addition, recent research is showing the significant damage that can be done by ultrafine particulates, which cannot be effectively removed from incinerator flue gases, and which can carry sorbed toxic pollutants into human and animal lungs and bodies, and on to growing vegetables and other crops (Dearden, op cit).

10: THE DOSE RESPONSE RELATIONSHIP

138. As discussed in the Harmful Emissions chapter (Section 3), there are hundreds, if not thousands, of chemicals emitted by incinerators and some of these can act synergistically (this can involve particulates) and that very little is known of the coalitive effect, cosynergism and potentiation (when one agent with no toxicity enhances the toxicity of the other). Virtually nothing is known of such effects, and they cannot, with our present knowledge, be predicted.

139. One of the central planks in toxicology is the rectilinear or sigmoidal dose-response relationship, with its assumption that higher doses produce greater toxic responses. A corollary to the dose-response relationship is there is usually (although not always) a threshold below which no observable adverse effect occurs. This is sometimes used by proponents of incineration to imply that even if there is no known threshold very low doses will have very little effect.

140. However, as Dearden (op cit) points out, it has been known for many years that some toxicants could exhibit high toxicity at very low doses. In such cases a graph of toxicity (on the vertical axis) versus dose (on the horizontal axis) passes through a minimum. This phenomenon is termed hormesis. Calabrese[158] has stated that the hormetic dose-response is far more common and fundamental than the dose-response models (threshold/linear no threshold) used in toxicology and risk assessment. In some cases the response at very low doses is qualitatively the opposite of that at higher doses; e.g. a toxin can promote health. However, Tuomisto et al[159],[160] found that the cancer risk from dioxins appeared to follow a hormetic pattern, with toxicity increasing at very low doses. Lippmann[161] has also suggested that hormesis occurs in the cancer risks from dioxins and related chemicals while Kaiser[162] showed that endocrine disruptors such as dioxins follow this pattern. Irigaray et al[163] have suggested that very lipophilic pollutants may also have a role in obesity.

141. This led Dearden to state that "*evidence is accumulating that low, perhaps even very low, levels of dioxins and other toxicants can carry very serious health risks. THE IMPORTANCE OF THIS CANNOT BE OVER-EMPHASISED, for it turns environmental toxicology on its head. It means, in my view, that not a single new incinerator should be built, and existing incinerators should be closed down as quickly as possible*".

142. We echo Dearden's views and contend that they are yet another reason to refuse planning permission for the EfW (incinerator) at AWRP.

11: TOWARDS A CONSENSUS?

143. Taken together, the studies reported above, coupled with the views of respected experts such as Professor C. Vyvyan Howard MB. ChB. PhD. FRCPath[xxi] and Professor J C Dearden[xxii]

[xxi] **Professor Vyvyan Howard** is a medically qualified toxico-pathologist specialising in the problems associated with the action of toxic substances on the foetus and the infant. He is Professor of Bioimaging at the University of Ulster and has written a number of papers and book chapters and spoken in a variety of forums to draw attention to the threat posed by environmental pollutants to the developing foetus. He is a Fellow of the Royal College of Pathologists, Past President of the Royal Microscopical Society, Member of the British Society of Toxico-Pathologists, Immediate Past President of the International Society of Doctors for the Environment and Member of the European Teratology Society. He has just completed 6 years as a toxicologist on the UK Government DEFRA Advisory Committee on Pesticides

[xxii] **Professor Dearden** (BSc, MSc, PHD) is Emeritus Professor of Medicinal Chemistry at Liverpool John Moores University. He is an honorary member of the Royal Pharmaceutical Society of Great Britain, for contributions to pharmaceutical research. In 2004 he received the biennial International QSAR Award for Research in Environmental Toxicology and has written about 250 scientific publications in computational toxicology and related fields. He served on a European Commission working party in connection with the recent REACH (Registration, Evaluation and Authorisation of Chemicals) legislation, and was invited to give evidence to the Royal Commission on Environmental Pollution in 2001.

demonstrate that objective scientific evaluation indicates that there are reasonable grounds for concern about potentially dangerous effects of incinerator emissions on human health, with babies and young children being amongst the most vulnerable. These views are supported by statements cited above from the World Health Organisation, the European Commission and the European Respiratory Society cited above.

144. While we believe that the evidence above is sufficiently robust, when coupled with the Precautionary Principle, to refuse planning permission for the AWRP EfW (incinerator) facility, we are aware that official UK bodies do not share the scientific consensus described above. Thus there is a significant body of opinion that does not support the emerging consensus and AmeyCespa have used this to claim that their proposed EfW (incinerator) plant is safe. This is discussed in the next section.

11.1: Supporting Policy Statements

145. Howard has pointed out that the health evidence is supported by strong policy. A 2007 WHO report[164] says

> "*the evidence of adverse health effects related to landfills and incinerators, although not conclusive, adds to other environmental concerns in directing waste management strategic choices towards reduction of waste production, re-use and recycling schemes, as prescribed by EU Directives*".

146. Howard states that the Irish Health Research Board review (*op cit*) includes similar commentary and says that one submission "*included a letter from the EU Environment Commissioner, which stressed that 'incinerators are not the answer to waste management .... Incinerators only reduce the volume of waste but the environmental impact of incineration is significant.'*" According to Howard, the same contributor quoted the Head of EU Waste Management, who stated that incinerators need enormous input in order to be economic and that in many countries they are now considered similar to nuclear power stations and should be avoided. Howard extracted the following quote:

'The Commission does not support incineration. We do not consider this technique is favourable to the environment or that it is necessary to ensure a stable supply of waste for promoting combustion over the long term. Such a strategy would only slow innovation. We should be promoting prevention and recycling above all. ***Those countries who are in the process of drafting their planning should not base it upon incineration****.'*

12: THE OFFICIAL VIEW ON HEALTH RISKS

147. The official view is that health risks, at least from a modern well-managed incinerator are small or negligible. In essence they take the caveat that observed health risks are associated with old plant further than seems justified. It is unfortunate that bodies such as DEFRA, the Environment Agency and the Health Protection Agency (HPA) take this view as this is likely to influence those in local in councils taking decisions on future waste strategy, including incineration.

148. Howard points out that behind these differences of opinion there lays a practical issue, and one of significant policy importance, in that the majority of published epidemiological studies relate to these older plants. Following the introduction of the European Union regulations (see above) many older plants have closed, or been fitted with more stringent emission controls. While clearly

desirable from a public health perspective, it does raise issues of the relevance of studies around older plants, to populations affected by more modern facilities. Thus Howard points out that:

- **Proponents of new facilities tend to dismiss the older research as irrelevant.** This seems to be reflected in the views of DEFRA, the HPA and the Environment Agency and in studies by the relevant advisory committees COC. COC emphasises the need to be careful in interpreting mortality and morbidity data and the need for proper epidemiological studies. They reviewed cancer incidence between the mid 1970s and mid 1980s in 14 million people living within 7.5 km of 72 British MSW incinerators (Elliott *et al*[165], COC[166]) and concluded that: "*any potential risk of cancer due to residency (for periods in excess of 10 years) near to MSW incinerators was exceedingly low, and probably not measurable by the most modern techniques"*. Their review seven studies on cancer incidence near old generation MSW incinerators published since 2000 (Comba et al, [167]; Floret et al[168], 2003; Knox[169], 2000; Viel et al[170], Zambon et al[171]) cited weaknesses in the studies due to confounding factors before concluding that *"Although the studies indicate some evidence of a positive association between two of the less common cancers ..... and residence near to incinerators in the past, the results cannot be extrapolated to current incinerators, which emit lower amounts of pollutants"*

- **Opponents take a contrary view** arguing that similar claims of safety were made in relation to those older facilities when they were operating; that the risk assessments relied upon to show new incinerators are safe would not, if applied to the older plants, reveal the levels of impacts reported in the literature thus indicating that the risk assessments do not validate in real-world situations; and that epidemiology, by its nature, involves retrospective studies. Furthermore the modern incinerators tend to be much larger than those operated historically so that although the emissions concentrations have reduced the total mass of pollutant emissions may even increase.

149. Howard cites a comprehensive review by the Irish Health Research Board[172] which was commissioned by Ireland's Department of Environment and Local Government, which clearly recognised these arguments and concluded that "*there is some evidence that incinerator emissions may be associated with respiratory morbidity*" and that "*acute and chronic respiratory symptoms are associated with incinerator emissions*". The review also confirmed that "*a number of well-designed studies have reported associations between developing certain cancers and living close to incinerator sites. Specific cancers identified include primary liver cancer, laryngeal cancer, soft-tissue sarcoma and lung cancer*".

150. The following extracts illustrate the views of DEFRA, the HPA and the Environment Agency.

12.1: DEFRA

151. DEFRA, in deriving the 2007 waste strategy for England stated:

> *Concern over health effects is most frequently cited in connection with incinerators. Research carried out to date shows no credible evidence of adverse health outcomes for those living near incinerators. The relevant health effects – primarily cancers – have long incubation times, but the available research demonstrates an absence of symptoms relating to exposures twenty or more years ago, when emissions from incineration were much greater than they are now. Very demanding EU standards for dioxin emissions now apply. The Health Protection Agency has published a short position statement on the health impacts for municipal waste incineration which reaches similar conclusions*.

152. DEFRA clearly see no health risks from incinerators. In deriving these views they reviewed studies into cancer cases close to incinerators and failed to find any consistent evidence of a link between exposure to emissions from incinerators and an increased rate of cancer. They also dismissed cases where apparently significant effects had been observed in relation to incinerators close to other sources of potentially hazardous emissions. This approach appears to ignore the work cited above and give undue weight to confounding factors.

12.2: Environment Agency

153. Similarly, the Environment Agency[173] state that:

> *"Studies into the health of communities living near to incinerators have not found any convincing links between incinerator emissions and adverse effects on public health. We work with health authorities and the Health Protection Agency to investigate local concerns."*

We presume that in making this statement, the Environment Agency is largely reflecting HPA views. These are discussed below.

154. However, the Environment Agency also state that: "*We regulate all waste facilities, including energy from waste incinerators, to prevent or minimize any risks to the environment or health*."

12.3: Health Protection Agency

155. The HPA report *The Impact on Health of Emissions to Air from Municipal Waste Incinerators*[174] sets out their most recent position. Its summary states that:

> *"The Health Protection Agency has reviewed research undertaken to examine the suggested links between emissions from municipal waste incinerators and effects on health. While it is not possible to rule out adverse health effects from modern, well regulated municipal waste incinerators with complete certainty, any potential damage to the health of those living close-by is likely to be very small, if detectable. This view is based on detailed assessments of the effects of air pollutants on health and on the fact that modern and well managed municipal waste incinerators make only a very small contribution to local concentrations of air pollutants. The Committee on Carcinogenicity of Chemicals in Food, Consumer Products and the Environment has reviewed recent data and has concluded that there is no need to change its previous advice, namely that any potential risk of cancer due to residency near to municipal waste incinerators is exceedingly low and probably not measurable by the most modern techniques. Since any possible health effects are likely to be very small, if detectable, studies of public health around modern, well managed municipal waste incinerators are not recommended".*

156. AmeyCespa in their Chapter 12 – Human Health quote part of this statement, saying:

> *"*It should be noted that in its position statement on the Impact of Health of Emissions to Air from Municipal Waste Incinerators (September 2009), the HPA concluded that *'while it is not possible to rule out adverse health effects completely, any potential damage from modern, well run and regulated incinerators is likely to be so small that it would be undetectable'."*

This quote does not fully reflect the caveats that the HPA attached in the full statement given above.

157. The HPA stress that these conclusions apply only to "*modern well-managed*" incinerators and their report outlines the relevant regulations. They obviously exclude poorly run incinerators and the older variety of incinerator that operated before pollution regulations came into force. Thus, they have dismissed the evidence that can be derived from epidemiological evidence

158. The phrase "*modern well-managed*" begs some questions.

- The term "*modern*" in normal parlance implies something that is well-designed, perhaps even "state of the art". Such an imprimatur of quality does not necessarily apply. For example, according to a September 2010 Site Status Report by the Scottish Environmental Protection Agency (SEPA) the Dargavel plant at Dumfries has experienced problems: "*Since commisioning re-started in March 2010… there have been 17 recorded noise complaints, 15 activations of the by-pass stack, 2 failures of the Continuous Emissions Monitoring System and 172 short term ELV (Emissions Limit Values) breaches"* (SEPA[175]). In September 2010 SEPA named the Dargavel plant as one of the 20 worst polluters in Scotland and categorised it as having a "very poor" pollution record[176].
- The term "*well-managed"*, or for that matter "*well run and regulated"* presumably include the idea that the plant in question manages its emissions in line with the requirements of the regulators and keeps them as low as possible. However, the reality is that many incinerators breach the regulations, as discussed in Section 6.1 of the Harmful Emissions Chapter.

159. Even for "*modern well-managed*" incinerators the HPA do not rule out the possibility of damage to health. Moreover, there are many examples where emission regulations are breached and the Dumfries plant is not alone in experiencing problems. The possibility of such problems at any new EfW / incineration plant cannot be ruled out.

160. HPA felt that provided solid ash residues and cooling water are handled and disposed of appropriately (a significant proviso), atmospheric emissions are the only significant route of exposure to people. They considered the effect of the following types of air-borne pollutant:
    I. Particles (i.e. particulates)
    II. Polychlorinated dibenzo-*p*dioxins and polychlorinated dibenzo-*p*-furans (commonly referred to as "dioxins")
    III. Other carcinogens such as the polycyclic aromatic hydrocarbons (PAH).

    These are discussed in turn below.

161. (1) **Particles:** The report says that "*both long-term exposure and short-term increases in exposure to particles can damage health*". It recognises that short-term increases in concentrations cause cardio-respiratory effects including an increase in deaths from heart attacks and from respiratory disease, increased hospital admissions for treatment of these disorders and increases in related symptoms. It admits that no "*thresholds of effect can be identified for either the effects of long-term exposure or for the effects of short-term increases in concentrations. Thus, any increase in particle concentrations should be assumed to be associated with some effect on health. The critical step in assessment of effects on health is not simply making the correct assertion that some effect is possible but in estimating the size of that effect.*" **Such statements should lead naturally to the Precautionary Principle**.

162. This is where uncertainty creeps in. At present, the regulations relate to the mass of particles in a given volume of air. So, PM_{10} is defined as the mass of particles of less than (about) 10 microns in diameter per m^3 of air and $PM_{2.5}$ is the mass of particles of less than about 2.5 microns in diameter per m^3 of air. Based on the mass of particles per m^3 of air generated by a modern well regulated incinerator, the report does not raise any major health concerns but it also says that:

"*… it is worth noting that PM_{10} and $PM_{2.5}$ samples from around the world can vary substantially in their chemical composition and size distribution but nonetheless exhibit similar concentration-response coefficients in time-series epidemiological studies.*

…. monitoring of chemical characteristics of the ambient aerosol (for example, its metallic components), the number of particles per unit of volume of air, the total surface area of particles per unit volume of air, or the capacity of particles to generate free radicals could prove more valuable than measurements of mass concentrations (PM_{10} and $PM_{2.5}$). But none of this is yet well established and international and national regulations are currently framed in terms of mass concentrations."

In other words, there may be health effects that are being missed because the regulations are based on mass concentration measurements rather than on these other measures such as particle numbers. This is all the more likely when one recalls that the smallest particles are also the most dangerous (see above). **Even on the evidence of the HPA report, there is need for caution and it is therefore essential to apply the Precautionary Principle.**

163. **(II) Carcinogens:** The HPA report distinguishes between genotoxic carcinogens (compounds that induce cancer by reacting directly with DNA to cause a mutation) and non-genotoxic carcinogens (compounds that induce cancer by a range of mechanisms not based on creating direct mutation). Most known human chemical carcinogens are genotoxic, e.g. aflatoxins, benzene, 1,3- butadiene, 2-naphthylamine, polycyclic aromatic hydrocarbon (PAH) compounds. Examples of non-genotoxic carcinogens are oestrogens and 2,3,7,8-tetrachlorodibenzo-para-dioxin (TCDD, a strongly carcinogenic dioxin). The terms carcinogen in the rest of the HPA report refers only to the genotoxic carcinogens and therefore ignores dioxins. The report says that:

"*If it is found that incinerators emit the carcinogens considered by EPAQS (the Expert Panel on Air Quality Standards),* [then] *it is reasonable to compare the augmented local concentration (i.e. the local background concentration plus the increment contributed by the incinerator) with the EPAQS standard. If this is not exceeded it may be reasonably assumed that the additional risk imposed by the emissions is minimal. If, on the other hand, the emissions cause the local concentrations to exceed the EPAQS standard(s), the appropriate regulator would need to decide whether the additional risk posed by the incinerator was a cause for concern and what further reductions may be necessary*".

164. So, if the emission of carcinogens is within the EPAQs standards(s) the report says the additional risk is minimal BUT if the emissions when added to the existing local concentrations exceeds the EPAQS standard(s), then there could be cause for concern. Much then depends on how well these standards deal with the lack of any known threshold for damaging effects and the great variability of individual people in their response to such environmental and health challenges. **Even on the evidence of the HPA report and bearing in mind the exclusion of non-genotoxic carcinogens, there is need for caution and it is therefore essential to apply the Precautionary Principle.**

165. **(III) Dioxins:** The report says that:

"*The majority (more than 90%) of non-occupational human exposure to dioxins occurs via the diet, with animal-based foodstuffs like meat, fish, eggs, and dairy products being particularly important. Limited exposure may also occur via inhalation of air or ingestion of soil depending on circumstances. Inhalation is a minor route of exposure and, given that DEFRA has calculated that incineration of MSW accounts for less than 1% of UK emissions of dioxins, the contribution of incinerator emissions to direct respiratory exposure of dioxins is a negligible component of the average human intake*".

It concludes that:

"*These calculations show that, even for people consuming a significant proportion of locally produced foodstuffs, the contribution of incinerator emissions to their intake of dioxins is small and well below the tolerable daily intake (TDI) for dioxins recommended by the relevant expert advisory committee".*

166. This conclusion presumably applies to modern well-managed incinerators and therefore begs the question of what happens even with a modern incinerator during start-up or fault periods. Dioxins may make a larger contribution to human exposure via the food chain, particularly fatty foods through the process of bio-accumulation. Dioxins from emissions could also be deposited on soil and crops and accumulate in the food chain via animals that graze on the pastures, though dioxins are not generally taken up by plants. Much therefore depends on the extent to which local people eat, or are willing to eat foodstuffs subject to the incinerator plume as well as on the rate of deposition. Thus the impact of emissions on locally produced foods such as milk and eggs (and perhaps the potential impact on local agricultural enterprises) should be considered in deciding whether to grant a planning permission.

13: POLICY CONTEXT

167. Emissions to air are covered by the Waste Incineration Directive (2000/76/EC), which requires adherence to emission limits for a range of pollutants, including dioxins/furans and trace metals. These limits have been discussed above and compared with the generally more stringent limits set by the US Environmental Protection Agency. In addition, the UK is bound by the Stockholm Convention on POPs which is crucial since emission of POPs could be avoided altogether since cheaper and cleaner alternatives exist.

168. AmeyCespa tabulate what they call "the most stringent SGV" and the Tolerable Daily Intake for Dioxins/Furans set by the WHO[177] and by the UK Committee on Toxicity (COT)[178]. The two are comparable and both are a decade old.

169. AmeyCespa quote a number of statements from PPS10. The first of these is that *"the overall objective of Government policy on waste … is to protect human health and the environment by producing less waste and by using it as a resource wherever possible."* (para.1, p.5). Moving towards a much higher rate of recycling (see our Chapter on need and Technology Choices) would be much more consistent with this aim than the AWRP configuration (the EfW (incinerator) plant is significantly oversized while burning waste that could have been reused or recycled destroys resources which must then be replaced from virgin resources). Finally, this chapter establishes that there is a substantial body of evidence that health risks from incineration are unacceptably high.

170. PPS10 Paragraph 3 states that a 'key planning objective' is to *"help secure the recovery or disposal of waste without endangering human health and without harming the environment, and enable waste to be disposed of in one of the nearest appropriate installations".* Again, this chapter shows, there is a substantial body of evidence that health risks from incineration are unacceptably high.

171. **For the reasons stated in the foregoing two paragraphs, granting planning permission to the EfW (incinerator) would be contrary to PPS10. It should therefore be refused.**

172. Planning Policy Statement 23: Planning and Pollution Control is important, especially when linked to the Precautionary Principle. This Statement advises that "*any consideration of the quality of land, air or water and potential impacts arising from development, possibly leading to impacts on health, is capable of being a material planning consideration, in so far as it arises or may arise from, or may affect, any land use*" and that "*the planning system should focus on whether the development itself is an acceptable use of land, and the impacts of those uses, rather than the control of processes or emissions themselves*". Bearing in mind the Precautionary Principle, the potential impacts the quality on land, air or water (see our Harmful Emissions chapter) and the impacts on health (as backed by epidemiological evidence) discussed above, this is clearly a material planning consideration. **We contend that constructing a visually intrusive and polluting facility of inappropriate design for a rural setting in an open location where it can be seen for miles around is <u>not</u> an appropriate use of land**.

173. According to AmeyCespa, the Waste Local Plan includes the following *Policy 4/1 Waste management Proposals* which states that *Proposals for waste management facilities will be permitted provided that* (among other things)*:*

b. the proposed method and scheme of working would minimise the impact of the proposal; the opposite is true since incineration is the worst technology other than landfill from a global warming standpoint (see our Climate Change chapter) and has more adverse environmental and health impact than any other waste management technology (as shown above and in our Harmful Emissions chapter).

c. "there would not be an unacceptable environmental impact"; incineration is the worst technology other than landfill from a global warming standpoint and has greater adverse health and environmental impact than any other waste management technology (as shown above and in our Harmful Emissions chapter).

d. "there would not be an unacceptable cumulative impact on the local area"; many of the pollutants emitted by the EfW (incinerator) plant are bio-accumulative so that adverse environmental and health impacts will build up over time – as discussed above.

h. "other environmental and amenity safeguards would effectively mitigate the impact of the proposal"; the air pollution control system is not adequate fully to mitigate the risk to health and the environment.

174. Far from supporting AmeyCespa's case, the Policy 4/1 Waste management Proposals militate strongly against it. Planning permission for the EfW (incinerator) should be refused.

175. There is a broader policy issue, that of **Sustainable development**. It is a pattern of resource use that aims to meet human needs while preserving the environment so that these needs can be met not only in the present, but also for generations to come[xxiii]. The term was used by the World Commission on Environment and Development[179] convened by the United Nations in 1983 – perhaps more commonly known as the Brundtland Commission. They coined what has become the most often-quoted definition of sustainable development as

> "*development that meets the needs of the present without compromising the ability of future generations to meet their own needs*".

176. Today's environmental problems, like air pollution, are largely a consequence of the unsustainable consumption of natural resources and the mismanagement of waste products. Sustainability is about environmental protection, sustained economic growth and social equity. Health risks link directly to failures in environmental protection and to social equity (social factors play a role in morbidity and mortality).This chapter shows that a wide range of pollutants are emitted into the

[xxiii] This is sometimes taught as **ELF**-Environment, Local people, Future.

atmosphere by incinerators and that there is a large body of evidence that there are significant and unacceptable health risks. Thus the EfW plant at AWRP would not be a sustainable development/

14: CONCLUSIONS

177. The evidence discussed in Sections 3 to 10 shows that there is considerable evidence from a wide variety of sources that incineration is linked directly to a wide range of adverse health impacts. While there may be defects in some of the individual studies, the overall message that incinerators harm the health of individuals in the population surrounding incinerators and even those living further afield is clear. Moreover, calculations of the risks of modern incinerators that meet current emission standards show that they exceed the generally accepted risk criterion of 1 in a million.

178. As revealed in Section 11 there is a considerable body of opinion that holds that objective scientific evaluation indicates that there are reasonable grounds for concern about potentially dangerous effects of incinerator emissions on human health, with babies and young children being amongst the most vulnerable. These views are supported both by eminent scientists and by statements from the World Health Organisation, the European Commission and the European Respiratory Society and others.

179. On the other hand official UK bodies do not share the scientific consensus described above (see Section 12). Thus there is a significant body of opinion that does not support the emerging consensus and AmeyCespa have used this to claim that their proposed EfW (incinerator) plant is safe.

180. There is therefore evidence of a range of adverse health effects but that it is of measurable extent is not accepted by all – the HPA for one do not accept this. We believe that all parties accept that the causal pathways between various categories of harm are known for many substances and that most would also accept that harm may also be caused by other, as yet undiscovered effects from substances now believed to be safe.

181. There are difficulties at the next stage – the dose-response relationship at low doses and this is compounded by the phenomenon of hormesis which is likely to be important. Other confounding factors are bio-accumulation, the greater sensitivity of babies (including those *in utero*), young children and the chemically sensitive and the long lead time or latency period of cancers. While the phenomenon of synergy is known, how far it influences the dose-response relationship is probably not universally agreed.

182. The current regulatory regime in the UK (and EU) falls short of best practice, though it has led to improved air quality as pollution generally has been lowered (this includes incinerators). However, the HPA appear not to have taken account of the fact that the smaller particles (PM_1 and ultrafine particles, $PM_{0.1}$) are more dangerous than the regulated PM_{10} and $PM_{2.5}$s; certainly regulatory limits do not take account of this.

183. We understand that it is necessary to balance the claimed "benefits" of incinerating NYCC's and York's MSW in the EfW (incinerator) plant at AWRP with the clear interest of local people to avoid the various categories of environmental and health harm that will befall them. As explained in Section 2, Faced with the uncertainties summarised above, it is essential to invoke the Precautionary Principle (as explained in Section 2).

184. Under the law of the European Union, the application of the Precautionary Principle has been made a statutory requirement (see Section 2). It presupposes that potentially dangerous effects deriving from a phenomenon, product or process have been identified, and that scientific

evaluation does not allow the risk to be determined with sufficient certainty. This is the situation regarding the health risks associated with incineration in general and therefore the health risks associated with emissions from the AWRP EfW (incinerator) plant.

185. Recall that EU Treaty Article 174(2) as amended at Nice 2004 recognized that scientific evaluation can be inconclusive and accorded priority to public health:

> "*a precautionary approach must be paramount, as opposed to acting only where proof or very strong suspicion of harm can be demonstrated".* And
>
> *"the protection of public health, including the effects of the environment on public health, must be given priority."*

186. We therefore contend that the Precautionary Principle must be applied to your consideration of the proposed EfW (incinerator) plant at AWRP. We very strongly object both to the harm that this plant would do to the health of the local area and beyond and to the health costs it would burden the NHS with.

187. Given that

- *"the protection of public health, including the effects of the environment on public health, must be given priority"* [EU Treaty Article 174(2)]
- There is a large body of scientific evidence and opinion that indicates there are reasonable grounds for concern about potentially dangerous effects of incinerator emissions on human health, with babies and young children being amongst the most vulnerable.
- Incineration is linked directly to a wide range of adverse health impacts including cancers, heart disease, diseases of the respiratory tract, endocrine system disorders and the effects of toxic heavy metals.
- Modern incinerators that meet current emission standards exceed the generally accepted risk criterion for cancer.
- Local planning policies and the UK's obligations under the Stockholm convention would be run counter to granting planning permission for the EfW (incineration) plant.
- Incineration uis not a sustainable development.

and that

- there are cheaper and cleaner alternatives which are free of the health risks associated with incineration

We urge you to refuse planning permission for the AWRP EfW (incinerator) plant

ANNEX 1: THE PRECAUTIONARY PRINCIPLE – FURTHER DETAILS

1. This Annex gives further details of the precautionary principle including its application to areas beyond the environment.

2. The precautionary principle may be invoked where urgent measures are needed in the face of a possible danger to human, animal or plant health, or to protect the environment where scientific data do not permit a complete evaluation of the risk. It may not be used as a pretext for protectionist measures. This principle is applied mainly where there is a danger to public health. For example, it may be used to stop distribution or order withdrawal from the market of products likely to constitute a health hazard.

ACT: Communication from the Commission of 2 February 2000 on the precautionary principle [COM(2000) 1 final - Not published in the Official Journal[180]].

SUMMARY

3. The EC Treaty contains only one explicit reference to the precautionary principle, namely in the title on environmental protection. However, in practice, the scope of this principle is far wider and also covers consumer policy and human, animal and plant health.

4. Since the precautionary principle is not defined in the Treaty or in other Community instruments, the Council in its Resolution of 13 April 1999 requested the Commission to develop clear and effective guidelines for the application of the principle. The Commission's Communication is a response to this request.

5. The establishment of common guidelines on the application of the precautionary principle will also have positive repercussions at international level. The principle has been recognised in various international agreements, notably in the Sanitary and Phytosanitary Agreement (SPS) concluded in the framework of the World Trade Organisation (WTO).

6. A clear definition as to how the Community intends to use the precautionary principle with a view to ensuring an appropriate level of environmental and health protection can contribute to the discussions already launched in these international arenas.

7. In its Communication, the Commission analyses the factors that trigger use of the precautionary principle and the associated measures. It then proposes guidelines for applying the principle.

The factors triggering use of the precautionary principle

8. According to the Commission the precautionary principle may be invoked when the potentially dangerous effects of a phenomenon, product or process have been identified by a scientific and objective evaluation, and this evaluation does not allow the risk to be determined with sufficient certainty. Hence use of the principle belongs in the general framework of risk analysis (which, besides risk evaluation, includes risk management and risk communication), and more particularly in the context of risk management which corresponds to decision-making.

9. The Commission stresses that the precautionary principle may only be invoked in the event of a potential risk and that it can never justify arbitrary decisions. Hence the precautionary principle may only be invoked when the three preliminary conditions are met - identification of potentially adverse effects, evaluation of the scientific data available and the extent of scientific uncertainty.

The measures resulting from use of the precautionary principle

10. As regards the measures resulting from use of the precautionary principle, they may take the form of a decision to act or not to act. The response depends on a political decision and is a function of the level of risk considered "acceptable" by the society on which the risk is imposed.

11. When action without awaiting further scientific information seems to be the appropriate response to the risk in application of the precautionary principle, a decision still has to be taken as to the nature of this action. Besides the adoption of legal instruments subject to review by the courts, there are a whole raft of measures for decision-makers to choose from (funding of a research programme, informing the public as to the adverse effects of a product or procedure, etc.).
Under no circumstances may the measure be selected on the basis of an arbitrary decision.

Guidelines for use of the precautionary principle

12. The precautionary principle should be informed by three specific principles:

- implementation of the principle should be based on the fullest possible scientific evaluation. As far as possible this evaluation should determine the degree of scientific uncertainty at each stage;
- any decision to act or not to act pursuant to the precautionary principle must be preceded by a risk evaluation and an evaluation of the potential consequences of inaction;
- once the results of the scientific evaluation and/or the risk evaluation are available, all the interested parties must be given the opportunity to study of the various options available, while ensuring the greatest possible transparency.

13. Besides these specific principles, the general principles of good risk management remain applicable when the precautionary principle is invoked. These are the following five principles:
    - proportionality between the measures taken and the chosen level of protection;
    - non-discrimination in application of the measures;
    - consistency of the measures with similar measures already taken in similar situations or using similar approaches;
    - examination of the benefits and costs of action or lack of action;
    - review of the measures in the light of scientific developments

The burden of proof

14. Apart from the rules applicable to products such as drugs, pesticides or food additives, Community legislation does not prescribe a prior authorisation system for placing products on the market. Thus in most cases it is for the users, the citizens or consumer associations to demonstrate the danger associated with a procedure or a product after it has been placed on the market.

15. According to the Commission, an action taken under the precautionary principle may in certain cases include a clause shifting the burden of proof to the producer, manufacturer or importer. This possibility should be examined on a case-by-case basis; the Commission does not recommend the general extension of such an obligation to all products.

RELATED ACTS

Regulation (EC) No 178/2002**of the European Parliament and of the Council of 28 January 2002 laying down the general principles and requirements of food law, establishing the European Food Safety Authority and laying down procedures in matters of food safety [Official Journal L 031 of 01.02.2002].**

16. The precautionary principle may be invoked where a food might have harmful effects on health (Article 7), in order to be able to react quickly and take appropriate measures. This principle is implemented in particular where there is uncertainty or where comprehensive scientific information on the potential risk is not available.

17. Measures must be proportionate to the risk and must be reviewed within a reasonable period of time.

ANNEX 2: AIR QUALITY AND THE LAW

1. The material presented in this Annex is taken from DEFRA at http://uk-air.defra.gov.uk/air-pollution/

UK and EU Air Quality Policy Context

EU Level

2. Action to manage and improve air quality is largely driven by European (EU) legislation. The 2008 ambient air quality directive (2008/50/EC) sets legally binding limits for concentrations in outdoor air of major air pollutants that impact public health such as particulate matter (PM_{10} and $PM_{2.5}$) and nitrogen dioxide (NO_2). As well as having direct effects, these pollutants can combine in the atmosphere to form ozone, a harmful air pollutant (and potent greenhouse gas) which can be transported great distances by weather systems.

3. The 2008 directive replaced nearly all the previous EU air quality legislation and was made law in England through the Air Quality Standards Regulations 2010, which also incorporates the 4th air quality daughter directive (2004/107/EC) that sets targets for levels in outdoor air of certain toxic heavy metals and polycyclic aromatic hydrocarbons. Equivalent regulations exist in Scotland, Wales and Northern Ireland.

4. Separate legislation exists for emissions of air pollutants with the main legislation being the UNECE Gothenburg Protocol which sets national emission limits (ceilings) for SO2, NOX, NH3 and volatile organic compounds for countries to meet from 2010 onwards. Similar ceilings have since been set in European law under the 2001 National Emission Ceilings Directive (2001/81/EC), which was subsequently made into UK law as the National Emission Ceilings Regulations 2002.

5. The European Commission is required to review the directive in 2013 and it is expected that they will initiate work with stakeholders and Member States later in 2011. The review is expected to look at strengthening provisions for fine particulate matter (PM2.5) and consolidate the 4th Air Quality Daughter Directive.

UK Level: National and Local Authorities

6. In the UK, responsibility for meeting air quality limit values is devolved to the national administrations in Scotland, Wales and Northern Ireland. The Secretary of State for Environment, Food and Rural Affairs has responsibility for meeting the limit values in England and the Department for Environment, Food and Rural Affairs (Defra) co-ordinates assessment and air quality plans for the UK as a whole.

7. The UK Government and the devolved administrations are required under the Environment Act 1995 to produce a national air quality strategy. This was last reviewed and published in 2007. The strategy sets out the UK's air quality objectives and recognises that action at national, regional and local level may be needed, depending on the scale and nature of the air quality problem.

8. Part IV of the Environment Act 1995 and Part II of the Environment (Northern Ireland) Order 2002 requires local authorities in the UK to review air quality in their area and designate air quality management areas if improvements are necessary. Where an air quality management area is designated, local authorities are also required to work towards the Strategy's objectives prescribed in regulations for that purpose. An air quality action plan describing the pollution

reduction measures must then be put in place. These plans contribute to the achievement of air quality limit values at local level.

Index and Bands

9. In the UK, most air pollution information services use the index and banding system approved by the Committee on Medical Effects of Air Pollutants (COMEAP). The system uses an index numbered 1-10, divided into four bands to provide more detail about air pollution levels in a simple way, similar to the sun index or pollen index.
    - 1-3 (Low)
    - 4-6 (Moderate)
    - 7-9 (High)
    - 10 (Very High)

10. The overall air pollution index for a site or region is determined by the highest concentration of five pollutants:
    - Nitrogen Dioxide
    - Sulphur Dioxide
    - Ozone
    - Carbon Monoxide
    - Particles < 10µm (PM_{10})

The table below outlines the air pollution health bandings and the potential impact on the health of people who are sensitive to air pollution.

Banding	Index	Health Descriptor
Low	1, 2, or 3	Effects are unlikely to be noticed even by individuals who know they are sensitive to air pollutants
Moderate	4, 5, or 6	Mild effects, unlikely to require action, may be noticed amongst sensitive individuals.
High	7, 8, or 9	Significant effects may be noticed by sensitive individuals and action to avoid or reduce these effects may be needed (e.g. reducing exposure by spending less time in polluted areas outdoors). Asthmatics will find that their 'reliever' inhaler is likely to reverse the effects on the lung.
Very High	10	The effects on sensitive individuals described for 'High' levels of pollution may worsen.

Boundaries Between Index Points for Each Pollutant

Nitrogen Dioxide

Based on the hourly mean concentration

Index	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
Band	Low	Low	Low	Moderate	Moderate	Moderate	High	High	High	Very High
µg/m³	0-95	96-190	191-286	287-381	382-477	478-572	573-635	636-700	701-763	764+
ppb	0-49	50-99	100-149	150-199	200-249	250-299	300-332	333-366	367-399	400+

Ozone

Based on the running 8-hourly or hourly mean. For ozone, the maximum of the 8-hourly and hourly mean is used to calculate the index value.

Index	1	2	3	4	5	6	7	8	9	10
Band	Low	Low	Low	Moderate	Moderate	Moderate	High	High	High	Very High
µg/m³	0-33	34-65	66-99	100-125	126-153	154-179	180-239	240-299	300-359	360+
ppb	0-16	17-32	33-49	50-62	63-76	77-89	90-119	120-149	150-179	180+

Sulphur Dioxide

Based on the 15-minute mean concentration.

Index	1	2	3	4	5	6	7	8	9	10
Band	Low	Low	Low	Moderate	Moderate	Moderate	High	High	High	Very High
µg/m³	0-88	89-176	177-265	266-354	355-442	443-531	532-708	709-886	887-1063	1064+
ppb	0-32	33-66	67-99	100-132	133-166	167-199	200-266	267-332	333-399	400+

Carbon Monoxide

Based on the running 8-hourly mean concentration.

Index	1	2	3	4	5	6	7	8	9	10
Band	Low	Low	Low	Moderate	Moderate	Moderate	High	High	High	Very High
mg/m³	0-3.8	3.9-7.6	7.7-11.5	11.6-13.4	13.5-15.4	15.5-17.3	17.4-19.2	19.3-21.2	21.3-23.1	23.2+
ppm	0.0-3.2	3.3-6.6	6.7-9.9	10.0-11.5	11.6-13.2	13.3-14.9	15.0-16.5	16.6-18.2	18.3-19.9	20+

PM_{10} Particles

Based on the running 24-hourly mean concentration.

Index	1	2	3	4	5	6	7	8	9	10
Band	Low	Low	Low	Moderate	Moderate	Moderate	High	High	High	Very High
µg/m³ (Grav. Equiv.)	0-21	22-42	43-64	65-74	75-86	87-96	97-107	108-118	119-129	130+
µg/m³ (Ref. Equiv.)	0-19	20-40	41-62	63-72	73-84	85-94	95-105	106-116	117-127	128+

ANNEX 3: UK EMISSIONS STANDARDS

National air quality objectives and European Directive limit and target values for the protection of human health							
Pollutant	**Applies**	**Objective**	**Concentration measured as[10]**	**Date to be achieved by and maintained thereafter**	**European obligations**	**Date to be achieved by and maintained thereafter**	**New or existing**
Particles (PM_{10})	UK	50µg.m^{-3} not to be exceeded more than 35 times a year	24 hour mean	31 December 2004	50µg.m^{-3} not to be exceeded more than 35 times a year	1 January 2005	Retain existing
	UK	40µg.m^{-3}	annual mean	31 December 2004	40µg.m^{-3}	1 January 2005	
	Indicative 2010 objectives for PM_{10} (from the 2000 Strategy and 2003 Addendum) have been replaced by an exposure reduction approach for $PM_{2.5}$ (except in Scotland – see below)						
	Scotland	50µg.m^{-3} not to be exceeded more than 7 times a year	24 hour mean	31 December 2010			Retain existing
	Scotland	18µg.m^{-3}	annual mean	31 December 2010			
Particles ($PM_{2.5}$) Exposure Reduction	UK (except Scotland)	25µg.m^{-3}	annual mean	2020	Target value 25µg.m^{-3} [12]	2010	New (European obligations still under negotiation)
	Scotland	12µg.m^{-3}		2020	Limit value 25µg.m^{-3}	2015	
	UK urban areas	Target of 15% reduction in concentrations at urban background[11]		Between 2010 and 2020	Target of 20% reduction in concentrations at urban background	Between 2010 and 2020	
Nitrogen dioxide	UK	200µg.m^{-3} not to be exceeded more than 18 times a year	1 hour mean	31 December 2005	200µg.m^{-3} not to be exceeded more than 18 times a year	1 January 2010	Retain existing
	UK	40µg.m^{-3}	annual mean	31 December 2005	40µg.m^{-3}	1 January 2010	
Ozone	UK	100µg.m^{-3} not to be exceeded more than 10 times a year	8 hour mean	31 December 2005	Target of 120µg.m^{-3} not to be exceeded more than 25 times a year averaged over 3 years	31 December 2010	Retain existing

National air quality objectives and European Directive limit and target values for the protection of human health							
Pollutant	**Applies**	**Objective**	**Concentration measured as**	**Date to be achieved by and maintained thereafter**	**European obligations**	**Date to be achieved by and maintained thereafter**	**New or existing**
Sulphur dioxide	UK	266µg.m^{-3} not to be exceeded more than 35 times a year	15 minute mean	31 December 2005			Retain existing
	UK	350µg.m^{-3} not to be exceeded more than 24 times a year	1 hour mean	31 December 2004	350µg.m^{-3} not to be exceeded more than 24 times a year	1 January 2005	
	UK	125µg.m^{-3} not to be exceeded more than 3 times a year	24 hour mean	31 December 2004	125µg.m^{-3} not to be exceeded more than 3 times a year	1 January 2005	
Polycyclic aromatic hydrocarbons	UK	0.25ng.m^{-3} B[a]P	as annual average	31 December 2010	Target of 1ng.m^{-3}	31 December 2012	Retain existing
Benzene	UK	16.25µg.m^{-3}	running annual mean	31 December 2003			Retain existing
	England and Wales	5µg.m^{-3}	annual average	31 December 2010	5µg.m^{-3}	1 January 2010	
	Scotland, Northern Ireland	3.25µg.m^{-3}	running annual mean	31 December 2010			
1,3- butadiene	UK	2.25µg.m^{-3}	running annual mean	31 December 2003			Retain existing
Carbon monoxide	UK	10mg.m^{-3}	maximum daily running 8 hour mean/in Scotland as running 8 hour mean	31 December 2003	10mg.m^{-3}	1 January 2005	Retain existing
Lead	UK	0.5µg.m^{-3}	annual mean	31 December 2004	0.5µg.m^{-3}	1 January 2005	Retain existing
		0.25µg.m^{-3}	annual mean	31 December 2008			

National air quality objectives and European Directive limit and target values for the protection of human health							
Pollutant	**Applies**	**Objective**	**Concentration measured as**	**Date to be achieved by and maintained thereafter**	**European obligations**	**Date to be achieved by and maintained thereafter**	**New or existing**
National air quality objectives and European Directive limit and target values for the protection of vegetation and ecosystems							
Nitrogen oxides	UK	30µg.m^{-3}	annual mean	31 December 2000	30µg.m^{-3}	19 July 2001	Retain existing in accordance with 1st Daughter Directive
Sulphur dioxide	UK	20µg.m^{-3}	annual mean	31 December 2000	20µg.m^{-3}	19 July 2001	Retain existing in accordance with 1st Daughter Directive
	UK	20µg.m^{-3}	winter average	31 December 2000	20µg.m^{-3}	19 July 2001	
Ozone: protection of vegetation & ecosystems	UK	Target value of 18,000µg m^{-3} based on AOT40 to be calculated from 1 hour values from May to July, and to be achieved, so far as possible, by 2010	Average over 5 years	1 January 2010	Target value of 18,000µg m^{-3} based on AOT40 to be calculated from 1 hour values from May to July, and to be achieved, so far as possible, by 2010	1 January 2010	New EU target

Source: http://uk-air.defra.gov.uk/documents/National_air_quality_objectives.pdf

ANNEX 4: SOURCES AND EFFECTS OF SOME HEAVY METALS

Metal	*Origins in Air*	*Effects*
Arsenic	Released into the air from both natural and anthropogenic sources. Most man-made emissions are released from metal smelters and the combustion of fuels. Tobacco smoke may contain arsenic, thereby being a source of exposure in ambient air. Arsenic in air is usually a mixture of arsenite and arsenate. Most As in the air is found in the fine particle fraction.	The oral uptake of arsenic, through food and drinking water, is generally the most important route of exposure, whereas inhalation normally contributes less than 1 % to the total dose. The non-cancerous effects of inhaling air with high arsenic levels are increased mortality from cardiovascular diseases, neuropathy and gangrene of the extremities. There is evidence that inorganic arsenic compounds are skin and lung carcinogens in humans. Lung cancer is the critical effect following exposure by inhalation. Arsenic is highly toxic to aquatic life and also very toxic to animals in general. Plant growth and crop yields may be reduced where soil arsenic content is high. Organic arsenic compounds are very persistent in the environment and bioaccumulate in the food chain.
Cadmium	Cadmium is released into the air from natural and man-made sources. Volcanoes, windborne particles and biogenic emissions are considered the main natural sources of cadmium in the atmosphere. Man-made sources of cadmium include non-ferrous metal production, stationary fossil fuel combustion, **waste incineration**, iron and steel production and cement production.	Food is the main source of cadmium exposure in the general population, > 90 % of the total intake in non-smokers. In heavily contaminated areas, dust re-suspension can constitute a substantial part of the exposure for the local population. In Europe, air pollution and mineral and organic fertilisers contribute roughly equally to annual exposure. Each continues to augment the relatively large accumulations of cadmium in topsoil, increasing the risk of future exposure through food. The levels of cadmium in non-smokers have not decreased over the last decade. Kidney and bones are the critical organs affected by chronic environmental exposure to cadmium. The main effects include an increased excretion of low-molecular-weight proteins in urine and increased risk of osteoporosis. An increased risk of lung cancer has also been reported following inhalation exposure in occupational settings. Cadmium is toxic to aquatic life as it is directly absorbed by organisms in water. It interacts with cytoplasmatic components such as enzymes, causing toxic effects in the cells. It can also produce lung cancers in humans and animals exposed via inhalation. Cadmium is highly persistent in the environment and bio-accumulates.
Lead	Lead is released into the air from natural and man-made sources. Natural emissions are soil suspension by wind, sea salt, volcanoes, forest fires and biogenic sources. These emissions are not entirely natural but contain some contributions from past depositions of anthropogenic lead. Major anthropogenic emission sources of lead on a global scale include the combustion of fossil fuels from, for example, traffic**, waste disposal** and production of non-ferrous metals, iron, steel and cement. The lead inputs through atmospheric deposition and the application of mineral and organic fertilisers to top-soils are of roughly the same magnitude. Those inputs are relatively small in comparison to lead stores that have already accumulated and inputs from natural	Lead is a neurotoxic metal that also accumulates in the body and damages organs (kidneys, liver, brain) and nerves. Exposure to high levels causes serious brain damage, including mental retardation, behavioural disorders, memory problems and mood changes. Impairment of neurodevelopment in children is the most critical effect. Exposure in utero, during breastfeeding or in early childhood may all be responsible for these effects. Lead accumulates in the skeleton and its mobilisation from the bones during pregnancy and lactation exposes the foetus or the breastfed infant. Hence, the lifetime exposure of a woman before pregnancy is important. Inhalation exposure may be significant when lead levels in the air are high. Elevated exposures are generally due to local sources rather than being the result of long-range transport. Most often, food is the predominant source of lead uptake in the general population. However, air pollution may contribute significantly to the lead content of crops, through direct deposition. Although uptake via plant roots is relatively limited, rising lead levels in soils over the long term are a matter for concern and should be addressed because of the

	sources.	possible health risks of low-level exposure to lead. Lead bioaccumulates and adversely impacts both terrestrial and aquatic systems. As with humans, the effects on animal life include reproductive problems and changes in appearance or behaviour.
Mercury	The largest man-made source of mercury on a global scale is the combustion of coal and other fossil fuels. Others sources include metal production, cement production, **waste disposal** and cremation. In addition, gold production makes a significant contribution to global emissions of mercury. The main natural sources of mercury are diffusion from the Earth's mantle though the lithosphere, evaporation from the sea surface and geothermal activity. Natural sources of mercury contribute about one-third of total global emissions, with anthropogenic emissions accounting for about two-thirds. Mercury emitted in inorganic forms is converted biologically to methylmercury in soil and water.	Mercury can damage the liver, the kidneys and the digestive and respiratory systems. It can also cause brain and neurological damage and impair growth. Methylmercury is a potent neurotoxin. Unborn children are the most susceptible population group. Mercury bioaccumulates and adversely impacts both terrestrial and aquatic systems. It can affect animals in the same way as humans and is very toxic to aquatic life. In several species of (mainly large predatory) fish and mammals the mercury guideline level (0.5 mg/kg) is often exceeded in many countries.
Nickel	Nickel is a ubiquitous trace metal, which occurs in soil, water, air and in the biosphere. Nickel emissions to the atmosphere may occur from natural sources such as wind-blown dust, volcanoes and vegetation. The main anthropogenic sources of nickel emissions into the air are combustion of oil for heat or power generation, nickel mining and primary production, incineration of waste and sewage sludge, steel manufacture, electroplating and coal combustion.	Food is the major source of exposure to nickel but exposure can also result from breathing ambient air, drinking water or inhaling tobacco smoke containing nickel. Skin contact with soil, bath or shower water, or metals containing nickel, as well as metals plated with nickel can also result in exposure. In very small quantities nickel is essential to humans. However, a large uptake can be a danger for human health as several nickel compounds are carcinogenic, increasing the risk of developing, for example, lung, nose, larynx or prostate cancers. Non-cancerous effects on health are allergic skin reactions (generally not caused by inhalation) and effects on the respiratory tract, the immune and defence systems and on endocrine regulation. The most common harmful health effect of nickel in humans is an allergic reaction. Approximately 10–20 % of the population is sensitive to nickel. As is the case for humans, nickel is an essential element for animals in small amounts. In high concentrations, nickel and its compounds can be acutely and chronically toxic to aquatic life and may affect animals in the same way as humans. It is known that high nickel concentrations in sandy soils can damage plants and high concentrations in surface waters can diminish the growth rates of algae. Microorganisms can also suffer from growth decline. Nickel is not known to accumulate in plants or animals. As a result nickel will not bio-magnify at higher levels in the food chain.

Source: European Environment Agency report Air quality in Europe — 2011 report

GLOSSARY

ACS	American Cancer Society
ADD	Attention Deficit Disorder
ADHD	Attention Deficit Hyperactivity Disorder
AQ	Air quality
AQFD	Air Quality Framework Directive
AQG	Air Quality Guidelines
AQLVs	Air quality limit values
AWRP	Allerton Waste Recovery Park
BSEM	British Society for Ecological Medicine
CAFE	Clean Air for Europe: CAFÉ
CFS	Chronic fatigue syndrome
COC	Committee on Toxicity of Chemicals in Food, Consumer
COMEAP	Committee on the Medical Effects of Air Pollutants
COPD	Chronic obstructive pulmonary disease
CYP	The cytochrome P450 superfamily
DEFRA	Department for Environment, Food and Rural Affairs
DNA	Deoxyribonucleic acid
DVT	Deep Vein Thrombosis
EfW	Energy from Waste
EPA	Environmental Protection Agency – as used here = incineration plus electricity generation
ETSU	Energy Technology Support Unit
EU	European Union
HPA	Health Protection Agency
IHD	[l]Ischaemic or ischemic heart disease or myocardial ischaemia
IQ	Intelligence Quotient
IRIS	*Ilots Regroupés pour l'Information Statistique*
MBT	Mechanical biological treatment
ME	Myalgic encephalomyelitis (ME), also referred to as CFS, or as post-viral fatigue syndrome (PVFS), or chronic fatigue immune dysfunction syndrome (CFIDS)
MS	Multiple Sclerosis
MSW	Municipal Solid Waste
MSWI	MSW Incinerators
NO_x	Oxides of nitrogen
P450	The cytochrome P450 superfamily (officially abbreviated as CYP)
PAH	Polycyclic Aromatic Hydrocarbons
PBBs	Polybrominated biphenyls
PBDE	Polybrominated diphenyl ethers
PCB	Polychlorinated biphenyls
PCDDs	Polychlorinated dibenzodioxins (commonly called *dioxins*)
PCDFs	Polychlorinated dibenzofurans (commonly called *furans*)
PDD	Pervasive developmental disorder
POPs	Persistent Organic Pollutants
SADS	Cardiac arrhythmia, also known as "Sudden Adult Death Syndrome" and "Sudden Arrhythmia Death Syndrome"
SAB-	International Joint Commission's Science Advisory Board, the Workgroup on

WGEH	Ecosystem Health
SEPA	Scottish Environmental Protection Agency
SIDS	Sudden Infant Death Syndrome
TCDD	2,3,7,8-tetrachlorodibenzo-para-dioxin
VOCs	Volatile Organic Compounds
UFP	Ultrafine Particles
UK	United Kingdom
US	United States
USA	United States of America
VOCs	Volatile organic compounds
WHO	World Health Organisation
WHI	Women' Health Initiative

REFERENCES

[1] http://archive.defra.gov.uk/environment/waste/residual/newtech/documents/incineration.pdf page 25

[2] Michaelson J. Rethinking regulatory reform: toxics, politics, and ethics. *Yale Law Journal* 105 (1996) 1891-1925.

[3] Science & Environmental Health Network. Wingspread Statement on the Precautionary Principle, 2004. www.sehn.org/web2printer4.php?img=0&Ink=0&page=precaution.html

[4] Recuerda, Miguel A. (2006). "Risk and Reason in the European Union Law". *European Food and Feed Law Review* **5**.

[5] Europa, Summary of EU Legislation, The Precautionary Principle http://europa.eu/legislation_summaries/consumers/consumer_safety/l32042_en.htm

[6] http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=CELEX:52000DC0001:EN:NOT

[7] Consolidated Version of the Treaty on the Functioning of the European Union article 191, paragraph 2

[8]Nitroarenes http://ntp.niehs.nih.gov/ntp/roc/eleventh/profiles/s121nitr.pdf

[9] Nitrosamines and Cancer, Richard A. Scanlan, Ph.D, Dean of Research Emeritus and Professor of Food Science, Linus Pauling Institute http://lpi.oregonstate.edu/f-w00/nitrosamine.html

[10] http://en.wikipedia.org/wiki/Nitrosamines

[11] Endocrine disruptor http://en.wikipedia.org/wiki/Endocrine_disruptor

[12] Harrison PTC, Humfrey CDN, Litchfield M, Peakall D, Shuker LK (1995). "Environmental oestrogens: consequences to human health and wildlife". *IEH assessment*. Medical Research Council, Institute for Environment and Health.

[13] "EDC Human Effects". *e.hormone*. Center for Bioenvironmental Research at Tulane and Xavier Universities. http://e.hormone.tulane.edu/learning/human-effects.html.

[14] Kodavanti PR (2006). "Neurotoxicity of Persistent Organic Pollutants: Possible Mode(s) of Action and Further Considerations". *Dose Response* **3** (3): 273–305. doi:10.2203/dose-response.003.03.002. PMID 18648619."

[15] Verhulst SL, Nelen V, Hond ED, Koppen G, Beunckens C, Vael C, Schoeters G, Desager K (January 2009). "Intrauterine exposure to environmental pollutants and body mass index during the first 3 years of life". *Environ. Health Perspect.* **117** (1): 122–6. doi:10.1289/ehp.0800003. PMID 19165398.

[16] Mullerova D, Kopecky J, Matejkova D, Muller L, Rosmus J, Racek J, Sefrna F, Opatrna S, Kuda O, Matejovic M (December 2008). "Negative association between plasma levels of adiponectin and polychlorinated biphenyl 153 in obese women under non-energy-restrictive regime". *Int J Obes (Lond)* **32** (12): 1875–8. doi:10.1038/ijo.2008.169. PMID 18825156.

[17] Uemura H, Arisawa K, Hiyoshi M, Satoh H, Sumiyoshi Y, Morinaga K, Kodama K, Suzuki T, Nagai M, Suzuki T (September 2008). "Associations of environmental exposure to dioxins with prevalent diabetes among general inhabitants in Japan". *Environ. Res.* **108** (1): 63–8. doi:10.1016/j.envres.2008.06.002. PMID 18649880.

[18] Toxicological review of decabromodiphenyl ether (BDE-209)". U.S. Environmental Protection Agency. 20098-06-01. http://www.epa.gov/ncea/iris/toxreviews/0035-tr.pdf.

[19] Toxicological review of decabromodiphenyl ether (BDE-209)". U.S. Environmental Protection Agency. 20098-06-01. http://www.epa.gov/ncea/iris/toxreviews/0035-tr.pdf.

[20] Betts KS (May 2008). "New thinking on flame retardants". *Environ. Health Perspect.* **116** (5): A210–3. PMID 18470294. .

[21] Costa LG, Giordano G (November 2007). "Developmental neurotoxicity of polybrominated diphenyl ether (PBDE) flame retardants". *Neurotoxicology* **28** (6): 1047–67. doi:10.1016/j.neuro.2007.08.007. PMID 17904639.

[22] Eriksson P, Viberg H, Jakobsson E, Orn U, Fredriksson A (May 2002). "A brominated flame retardant, 2,2',4,4',5-pentabromodiphenyl ether: uptake, retention, and induction of neurobehavioral alterations in mice during a critical phase of neonatal brain development". *Toxicol. Sci.* **67** (1): 98–103. doi:10.1093/toxsci/67.1.98. PMID 11961221.

[23] Lema SC, Dickey JT, Schultz IR, Swanson P (December 2008). "Dietary exposure to 2,2',4,4'-tetrabromodiphenyl ether (PBDE-47) alters thyroid status and thyroid hormone-regulated gene transcription in the pituitary and brain". *Environ. Health Perspect.* **116** (12): 1694–9. doi:10.1289/ehp.11570. PMID 19079722.

[24] Kodavanti PR (2006). "Neurotoxicity of Persistent Organic Pollutants: Possible Mode(s) of Action and Further Considerations". *Dose Response* **3** (3): 273–305. doi:10.2203/dose-response.003.03.002. PMID 18648619.

[25] Dr Dick van Steemis - Incinerators – are WMDs? http://www.countrydoctor.co.uk/precis/precis%20-%20Incinerators%20-%20WMDs.htm

[26] The Parliamentary Office of Science and Technology Postnote, Air Quality in the UK, Nov 2002 http://www.parliament.uk/documents/post/pn188.pdf

[27] COMEAP (1998), Quantification of the effects of air pollution on health in the UK

[28] Environmental Risks, Ch 6 of Annual Report 2006 —French Institute for Public Health Surveillance: Example of municipal solid waste incinerators http://www.invs.sante.fr/publications/2007/annual_report_2006/chapitre_6_2006.pdf

[29] The Health Effects of Waste Incinerators, 4th Report of the British Society for Ecological Medicine, Second Edition, June 2008, Moderators: Dr Jeremy Thompson and Dr Honor Anthony

[30] Dockery DW, Pope Ca 3rd, Xu X et al. An association between air pollution and mortality in six US cities. N Eng J Med 1993; 329(24): 1753-9.

[31] Pope CA, Thun MJ, Namboodiri MM et al. Particulate air pollution as a predictor of mortality in a prospective study of US adults. Am J Respir Crit Care Med 1995; 151 (3 pt 1): 669-74.

[32] Goldberg MS, Burnett RT, Bailar JC et al. The association between daily mortality and ambient air particle pollution in Montreal, Quebec. 2. Cause-specific mortality. Environ Res 2001: 86(1): 26-36.

[33] Pope CA, Burnett RT, Thun MJ, et al. Lung cancer, cardiopulmonary mortality, and longterm exposure to fine particulate air pollution. JAMA 2002; 287(9): 1132-41.

[34] Maheswaran R, Haining RP, Brindley P et al. Outdoor air pollution and Stroke in Sheffield, United Kingdom, Small-Area Geographical Study. Stroke 2005; 36(2): 239-43.

[35] Jedrychowski W, Bendkowska I, Flak E et al. Estimated risk for altered fetal growth

resulting from exposure to fine particles during pregnancy: an epidemiologic prospective cohort study in Poland. Environ Health Perspect 2004; 112(14): 1398-1402

[36] Air Quality Guidelines for Europe, Section 7.3 p19, Second Edition, World Regional Publications, Regional European Series No 91, World Health Organisation, Regional Office for Europe, Copenhagen

[37] Perera FP, Rauh V, Whyatt RM et al. Molecular evidence of an interaction between prenatal environmental exposures and birth outcomes in a multiethnic population. Environ Health Perspect 2004; 112(5): 626-30

[38] Proceedings of the Third Colloquium on Particulate Air Pollution and Human Health 6-8 June 1999, Durham, North Carolina, Irvine, CA: Air Pollution Effects Laboratory, University of California,1999, 11/23

[39] Pope CA, Bumett RT, Thurston GD et al. Cardiovascular Mortality and Long-Term Exposure to Particulate Air Pollution: Epidemiological Evidence of General Pathophysiological Pathways of Disease. Circulation 2004; 109: 71-77

[40] Peters A, Dockery DW, Muller JE et al. Increased particulate air pollution and the triggering of myocardial infarction. Circulation 2001; 103 (23): 2810-5.

[41] Pope CA, Burnett RT, Thun MJ, et al. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA 2002; 287(9): 1132-41.

[42] Miller KA, Siscovick DS, SheppardL, et al. Long term exposure to air pollution and incidence of cardiovascular events in women. N Eng J Med 2007, 356:447-458

43 Baccerelli A, Martinelli I, Zanobetti A et al. Exposure to particulate air pollution and risk of deep venous thrombosis. Arch Int Med 2008; 168(9):920-7

[44] An Bord Pleanála, Statement of Evidence, Particulate Emissions and Health, Proposed Ringaskiddy Waste-to-Energy Facility, Professor C. Vyvyan Howard MB. ChB. PhD. FRCPath., June 2009

[45] Pope C.A. and Dockery D.W. Health effects of fine particulate air pollution: lines that connect. Journal of the Air and Waste Management Association 56 (2006) 709-742

[46] Brown L.M., Collings N., Harrison R.M., Maynard A.D. and Maynard R.L. Ultrafine particles in the atmosphere: introduction. Philosophical Transactions of the Royal Society of London A 358 (2000) 2563-2565.

[47] http://www.crwi.org/textfiles/partem.htm

[48] www.artac.info/static.php?op=MemorandumParisAppeal.txt&npds=1

[49] Memorandum submitted by Professor J C Dearden (Waste 69), Waste Strategy for England 2007 - Environment, Food and Rural Affairs Committee
http://www.publications.parliament.uk/pa/cm200910/cmselect/cmenvfru/230/230we57.htm

[50] Miller K.A., Siscovick D.S., Sheppard L., Shepherd K., Sullivan J.H., Anderson G.L. and Kaufman J.D. Long-term exposure to air pollution and incidence of cardiovascular events in women. New England Journal of Medicine 356 (2007) 447-458

[51] Pope C.A. Mortality effects of longer term exposures to fine particulate air pollution: review of recent epidemiological evidence. Inhalation Toxicology 19 (2007) 33-38.

[52] Curtis L., Rea W., Smith-Willis P., Fenyves E. and Pan Y. Adverse health effects of outdoor air pollutants. Environment International 32 (2006) 815-830

[53] Pope C.A., Burnett R.T., Thun M.J., Calle E.E., Krewski D., Ito K. and Thurston G.D. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. Journal of the American Medical Association 287 (2002) 1132-1141

[54] Woodruff T.J., Darrow L.A. and Parker J.D. Air pollution and postneonatal infant mortality in the United States, 1999-2002. Environmental Health Perspectives 116 (2008) 110-115

[55] Pino P., Walter T., Oyarzun M., Villegas R. and Romieu I. Fine particulate matter and wheezing illness in the first year of life. Epidemiology 15 (2004) 702-708

[56] Commision of the European Communities, COM(2005) 446 final COMMUNICATION FROM THE COMMISSION TO THE COUNCIL AND THE EUROPEAN PARLIAMENT
Thematic Strategy on air pollution {SEC(2005) 1132}. 2005

[57] European Environment Agency, EEA Technical report No 12/2011, *Air quality in Europe — 2011 report*, ISBN: 978-92-9213-232-3, ISSN Annual report series: 1977-284X, ISSN EEA Technical report series: 1725-2237, doi:10.2800/8321

[58] WHO, 2006, *Air quality guidelines — global update 2005*, World Health Organization Regional Office for Europe, Copenhagen, Denmark

[59] WHO, 2008, 'Air quality and health — Fact sheet no 313 — Updated August 2008'
http://www.who.int/ mediacentre/factsheets/fs313/en/

[60] Polichetti, G., et al., *Effects of particulate matter (PM10, PM2.5 and PM1) on the cardiovascular system*. Toxicology. In Press, Corrected Proof. Cited by Professor CV Howard in 2009

[61] Pope, A.C., 3rd and D.W. Dockery, *Health Effects of Fine Particulate Air Pollution: Lines that Connect.* Journal of the Air & Waste Management Association, 2006. 56: p. 709-742.

[62] http://ec.europa.eu/environment/archives/cafe/activities/pdf/case_study2.pdf

[63] Cormier, S.A., et al., *Origin and health impacts of emissions of toxic by-products and fine particles from combustion and thermal treatment of hazardous wastes and materials.* Environ Health Perspect, 2006. **114**(6): p. 810-7

[64] Particulate Matter: Properties and Effects upon Health, BIOS Scientific Publishers Ltd, Oxford p 63-84.

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

[66] EC (1998) Proposal for a Council Directive on the incineration of waste. Brussels 07.10.1998 COM (1999) 558final. 98/0289 (SYN)

[67] Stolzel M, Breitner S Cyrys J et al. Daily mortality and particulate matter in different size classes in Erfurt, Germany. J Expo Sci Environ Epidemiol.Research, 2007; 17(5): 459-67

[68] Air Quality Guidelines for Europe, Section 7.3 p19, Second Edition, World Regional Publications, Regional European Series No 91, World Health Organisation, Regional Office for Europe, Copenhagen

[69] Annesi-Maesano I, Forastiere F, Kunzli N et al. Partciculate matter, Science and EU Policy. Eur Resp J 2007; 29: 428-431

[70] Schwartz J, Laden F, Zanobetti A. The concentration-response relation between PM2.5 and daily deaths. Environ Health Perspect 2002; 110(10): 1025-9.

[71] Samet JM, Dominici F, Curriero FC et al. Fine particulate air pollution and mortality in 20 US cities 1987-1994. N Eng J Med 2000; 343(24): 1742-9

[72] Elliot P, Shaddick G, Kleinschmidt I etal, Cancer incidence near municipal solid waste incinerators in Great Britain. Brit J Cancer 1996; 73(5): 702-10

[73] Knox E.G. (2000). Childhood cancers, birthplaces, incinerators and landfill sites. International Journal of Epidemiology 29:391-397 http://ije.oxfordjournals.org/cgi/reprint/29/3/391.pdf

[74] Biggeri A, Barbone F, Lagazio C, et al. Air pollution and lung cancer in Trieste, Italy: Spatial analysis of risk as a function of distance from sources. Environ Health Perspect 1996; 104 (7): 750-4.

[75] Viel JF, Arveux P, Baverel J, et al. Soft tissue sarcoma and non Hodgkin's lymphoma clusters around municipal solid waste incinerators with high dioxin emission levels. Am J Epidemiology 2000; 152(1): 13-19 -

[76] Ohta S, Kuriyama S, Nakao et al. Levels of PCDDs, PCDFs and non-ortho coplanar PCBs in soil collected from high cancer-causing area close to batch-type municipal solid waste incinerator in Japan. Organohalogen Compounds 1997; 32: 155-60.

[77] Comba P, Ascoll V, Belli S et al Risk of soft tissue sarcomas and residence in the neighbourhood of an incinerator of industrial wastes. Occup Environ Med 2003; 60(9): 680-3

[78] Zambon P, Ricci P, Bovo E et al. Sarcoma risk and dioxin emissions from incinerators and industrial plants: a population-based case-control study (Italy). Environ Health 2007; 6:

[79] Gustavsson P. Mortality among workers at a municipal waste incinerator. Am J Ind Med 1989; 15(3): 245-53

[80] Gustavsson P, Evanoff B, Hogstedt C. Increased risk of esophageal cancer among workers exposed to combustion products. Archives Environ Med 1993; 48(4): 243-5.

[81] Ohta S, Kuriyama S, Nakao et al. Levels of PCDDs, PCDFs and non-ortho coplanar PCBs in soil collected from high cancer-causing area close to batch-type municipal solid waste incinerator in Japan. Organohalogen Compounds 1997; 32: 155-60

[82] Ogulei, D., et al., Source apportionment of Baltimore aerosol from combined size distribution and chemical composition data. Atmospheric Environment, 2006. 40(Supplement 2): p. 396-410.

[83] Ogulei, D., et al., Receptor modeling for multiple time resolved species: The Baltimore supersite. Atmospheric Environment, 2005. 39(20): p. 3751-3762.

[84]Ehmann WD, Markesbery WR, Alauddin M et al. Brain trace elements in Alzheimer's disease. Neurotoxicology 1986; 7 (1): 195-206

[85] Thompson CM, Markesbery WR, Ehmann WD et al. Regional trace-element studies in Alzheimer's disease. Neurotoxicology 1988; 9(1): 1-7

[86] Wenstrup D, Ehmann WD, Markesbery WR. Trace element imbalances in isolated subcellular fractions of Alzheimer's disease brains. Brain Res 1990; 533(1): 125-31

[87] Schettler T. Toxic threats to neurological development of children. Environ Health Perspect 2001; 109 (Suppl 6): 813-6.

[88] Grandjean P, Weihe P, White RF et al. Cognitive deficit in 7-year old children with prenatalexposure to methyl mercury. Neurotoxicol Teratol 1997; 19(6): 417-28

[89] Palmer RF, Blanchard S, Stein Z et al. Environmental mercury release, special education rates,and autism disorder: an ecological study of Texas. Health Place, 2006;12(2): 203-9.

[90] Palmer RF, Blanchard S, Wood R. Proximity to point sources of environmental mercury release as a predictor of autism prevalence. Health Place, 2008; doi:10.1016/j.healthplace.2008.02.001.

[91] Peters JM, Thomas D, Falk H et al. Contribution of metals to respiratory cancer. Environ Health Perspect 1986;70: 71-83.

[92] Thun MJ, Schnorr TM, Smith AB, et al. Mortality among a cohort of US cadmium production workers – an update. J Natl Cancer Inst 1985; 74(2): 325-33.

[93] Blot WJ, Fraumeni JF Jnr. Arsenical air pollution and lung cancer. Lancet 1975; 2 (7926):142-4

[94] Severs R, Whitehead L, Lane R. Air quality correlates of chronic disease mortality: Harris County, Texas 1969-71. Tex Rep Biol Med 1978; 36: 169-84.

[95] David OJ, Hoffman SP, Sverd J, et al. Lead and hyperactivity: Behavioural response to chelation. Am J Psych 1976; 133(10): 1155-8 .

[96] Masters RD. Biology and politics: linking nature with nurture. Ann Rev Polit Sci 2001; 4: 345-65.

[97] Leviton A, Bellinger D, Allred EN et al. Pre and postnatal low-level lead exposure and children's dysfunction in school. Environ Res 1993: 60(1): 30-43.

[98] Eppright TD, Sanfacon JA, Horwitz FA. Attention deficit hyperactivity disorder, infantile autism and elevated blood lead: a possible relationship. Mol Med 1996: 93(3): 136-8.

[99] Bellinger D, Leviton A, Waternaux C, et al. Longitudinal analyses of prenatal and postnatal lead exposure and early cognitive development. N Eng J Med 1987; 316 (17): 1037-43.

[100] Tong S, Baghurst P, McMichael A et al. Lifetime exposure to environmental lead and children's intelligence at 11 – 13 years: the Port Pirie Cohort Study. BMJ 1996; 312 (7046): 1569-75.

[101] Needleman HL, Riess JA, Tobin MJ, et al. Bone lead levels and delinquent behaviour. JAMA 1996; 275 (5); 363-9

[102] Mispelstraat: Living under the smoke of a waste incinerator. Report on the health impact of the MIWA waste incinerator in Sint Niklaas, Belgium. www.milieugezondheid.

[103] Gottscalk LA, Rebello T, Buchsbaum MS et al. Abnormalities in hair trace elements as indicators of aberrant behaviour. Comp Pyschiatry 1991; 32 (3): 229-37.

[104] Schauss AG. Comparative hair-mineral analysis results of 21 elements in a random selected behaviourally "normal" 19-59 year old population and violent adult criminal offenders. Int J Biosoc Res 1981; 1: 21-41. -

[105] Papers cited by BSEM:

[105a)] Bowdler NC, Beasley DS, Fritze EC et al. Behavioural effects of aluminium ingestion on animal and human subjects. Pharmacol Biochem Behav 1979: 10(4): 502-12.

[105b)] Trapp GA, Miner GH, Zimmerman RL et al. Aluminium levels in the brain in Alzheimer's disease. Biol Pyschiatry 1978; 13(6): 709-18.

[105c)] Multhaup G. Amyloid precursor protein, copper and Alzheimer's disease. Biomed Pharmocother 1997: 51(3): 105-11.

[105d)] Zapatero MD, Garcia de Jalon A, Pascual F, et al. Serum aluminium levels in Alzheimer's disease and other senile dementias. Biol Trace Elem Res 1995; 47 (1-3): 235-40.

[105e)] Martyn CN, Barker DJ, Osmond C et al. Geographical relationship between Alzheimer's disease and aluminium in drinking water. Lancet 1989; 1(8763): 59-62.
[105f)] Crapper DR, Krishnan SS, Dalton AJ et al. Brain aluminium distribution in Alzheimer's disease and experimental neurofibrillary degeneration. Science 1973: 180(85): 511-3.
[105g)] Neri LC, Hewitt D. Alzheimer's disease and drinking water. Lancet 1991; 338 (8763): 390.

[106] Zayed J, Ducic S, Campanella G, et al. Environmental factors in the etiology of Parkinson's disease. Can J Neurol Sci 1990: 17(3): 286-91.

[107] Centers for Disease Control. Blood and hair mercury levels in young children and women of childbearing age. United States 1999 Morbidity and Mortality Report, 2001; 50: 140-43.

[108] Anderson LM, Diwan BA, Fear NT, Roman E. *Critical windows of exposure for children's health: cancer in human epidemiological studies and neoplasms in experimental animal models*. Environ Health Perspect 2000; 108 suppl 3: 573-94

[109] Porterfield SP. *Vulnerability of the developing brain to thyroid abnormalities and environmental insults to the thyroid system.* Environ Health Perspect 1994; 102 Supp 2: 125-30.

[110] Sonnenschein C, Soto AM. An Updated review of environmental estrogen and androgen mimics and antagonists. J Steroid Biochem Mol Biol 1998; 65 (1-6): 143-50.

[111] Markey CM, Coombs MA, Sonnenschein C, Soto AM. *Mammalian development in a changing environment: exposure to endocrine disruptors reveals the developmental plasticity of steroid-hormone target organs*. Evol Dev 2003; 5(1): 67-75.

[112] Weisgals-Kuperas N, Patandin S, Berbers GA, et al. Immunological effects of background exposure to polychlorinated biphenyls and dioxins in Dutch preschool children. Environ Health Perspect 2000; 108(12): 1203-

[113] Mispelstraat: Living under the smoke of a waste incinerator. Report on the health impact of the MIWA waste incinerator in Sint Niklaas, Belgium. www.milieugezondheid.

[114] ten Tusscher GW, Stam GA, Koppe JG. Open chemical combusting resulting in a localised increased incidence of orofacial clefts. Chemosphere 2000; 40(9-11): 1263-70.

[115] Van Lorebeke N. Health effects of a household waste incinerator near Wilrijk, Belgium. *In* Health Impacts of Waste Management Policies, Hippocrates Foundation, Kos, Greece, 2000.

[116] Cordier S, Chevrier C, Robert-Gnansia E et al. Risk of congenital anomalies in the vicinity of municipal solid waste incinerators. Occup Environ Med 2004: 61(1): 8-15.

[117] Dummer TJ, Dickinson HO, Parker L. Adverse pregnancy outcomes around incinerators and crematoriums in Cumbria, North-west England, 1956-93. J Epidemiol Community Health 2003: 57(6): 456-61

[118] Newland M.C. and Rasmussen E.B. Behavior in adulthood and during aging is affected by contaminant exposure in utero. *Current Directions in Psychological Science* 12 (2003) 212-217.

[119] Landrigan PJ, Garg A. Chronic effects of toxic environmental exposures in children's health. J Toxicol Clinical Toxicol 2002; 40(4): 449-56

[120] Calabrese E.J. Age and Susceptibility to Toxic Substances. New York, John Wiley & Sons 1986.

[121] Toxins in breastmilk
http://www.babycentre.co.uk/baby/breastfeeding/problemsandsolutions/toxinsinbreastmilk/?_requestid=7932199

[122] Rogan WJ, Bagniewska A, Damstra T. Pollutants in breast milk. N Engl J Med 1980; 302(26): 1450-3 –

[123]BBC News – Health: Breast milk studied for toxins http://news.bbc.co.uk/1/hi/health/391514.stm

[124] Perera FP, Tang D, Tu YH et al. Biomarkers in maternal and newborn blood indicate heightened fetal susceptibility to procarcinogenic DNA damage. Environ Health Perspect 2004; 112(10): 1133-6

[125] Jedrychowski W, Bendkowska I, Flak E et al. Estimated risk for altered fetal growth resulting from exposure to fine particles during pregnancy: an epidemiologic prospective cohort study in Poland. Environ Health Perspect 2004; 112(14): 1398-1402

[126] Perera FP, Rauh V, Whyatt RM et al. Molecular evidence of an interaction between prenatal environmental exposures and birth outcomes in a multiethnic population. Environ Health Perspect 2004; 112(5): 626-30

[127] Burden of disease attributable to selected environmental factors and injury among children and adolescents in Europe (no authors listed). Child Care Health Dev 2004; 30(6): 731-732

[128] Knox EG. Childhood cancers and atmospheric carcinogens. J Epidemiol Community Health 2005; 59(2): 101-5

[129] Needleman HL, Leviton A, Bellinger D. Lead-associated intellectual deficit. N Eng J Med 1982; 306(6): 367.

[130] World Health Organisation, *Effects of air pollution on children's health and development – a review of the evidence*. 2005, Special Programme on Health and Environment, European Centre For Environment and Health: Bonn.

[131] World Health Organisation Europe. *Children's Environment and Health Action Plan for Europe*. in *Fourth Ministerial Conference on Environment and Health*. 2005. Budapest, Hungary.

[132] Schwartz, J., *Air pollution and children's health.* Pediatrics, 2004. **113**(4 Suppl): p. 1037-43.

[133] Heinrich, J. and R. Slama, *Fine particles, a major threat to children.* International Journal of Hygiene and Environmental Health, 2007. **210**(5): p. 617-622

[134] Hertz-Picciotto, I., et al., *Early childhood lower respiratory illness and air pollution.* Environ Health Perspect, 2007. **115**(10): p. 1510-8.

[135] Newland M.C. and Rasmussen E.B. Behavior in adulthood and during aging is affected by contaminant exposure in utero. *Current Directions in Psychological Science* 12 (2003) 212-217

[136] Kunzli, N., et al., *Breathless in Los Angeles: The Exhausting Search for Clean Air.* Am J Public Health, 2003. **93**(9): p. 1494-1499.

[137] Ashford N, Miller C. Chemical Exposures: Low Levels and High Stakes. John Wiley & Sons 1998.

[138] Hattis D, Russ A, Goble R, et al. Human interindividual variability in susceptibility to airborne particles. Risk Anal 2001; 21(4): 585-99.

[139] Pritchard C, Baldwin D, Mayers A. Changing patterns of adult neurological deaths (45-74 years) in the major western world countries (1979-1997). Public Health 2004; 118(4): 268-83.

[140] Taylor B, Miller E, Farrington CP et al. Autism and measles, mumps and rubella vaccine: no epidemiological evidence for a causal association. Lancet 1999; 353(9169): 2026-9

[141] National Research Council (2000): Waste Incineration and Public Health ISBN: 0-309-06371-X,Washington DC, National Academy Press.

[142] Mittal AK, Van Grieken R, Ravindra.. Health risk assessment of urban suspended particulate matter with special reference to polycyclic aromatic hydrocarbons: a review. Rev Environ Health 2001; 16 (Pt 3): 169-89.

[143] Final report to the North American Commission for Environmental Cooperation (Flushing, N.Y.: Centre for the Biology of Natural systems, Queens College, CUNY, 2000).

[144] Raloff FJ. Even Nunavut gets plenty of dioxin. Science News 2000; 158 : 230.

[145] Energy Technology Support Unit (ETSU), 1996, Economic Evaluation of the draft incinerator Directive, European Commission

[146] Final report for DEFRA by Enviros Consulting Ltd in association with ENTEC: Valuation of the External Costs and Benefits to Health and Environment of Waste Management Options. Dec 2004

[147] Eshet T, Ayalon O, Schecter M. A Critical Review of Economic Evaluation Studies of Externalities from Incineration and landfilling. Waste management Res, 2005; 23:487-504

[148] Miranda ML, Hale B. Waste not, want not, the private and social cost of energy production. Energy Policy, 1997; 25:587-600 -

[149] Wulf-Schnabel J, Lohse J. Economic evaluation of dust abatement techniques in the European Cement Industry. A report produced for the European Commission, May 1999.

[150] Pianin E. Study finds Net Gain from Pollution rules. Washington Post, Sept 27th, 2003.

[151] Thompson J. and Anthony H. The health effects of waste incinerators. *Journal of Nutritional and Environmental medicine* 15 (2005) 115-156

[152] Roberts R.J. and Chen M. *Waste incineration—how big is the health risk? A quantitative method to allow comparison with other health risks.* Journal of Public Health 28 (2006) 261-266.

[153] Castorina R. and Woodruff T.J. Assessment of potential risk levels associated with U.S. Environmental Protection Agency reference values. *Environmental Health Perspectives* 111 (2003) 1318-1325.

[154] COT (Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment) report: Variability and uncertainty in toxicology of chemicals in food, consumer products and the environment, March 2007.

[155] Michaelson J. Rethinking regulatory reform: toxics, politics, and ethics. *Yale Law Journal* 105 (1996) 1891-1925.

[156] Ricci P.F., Cox L.A. and MacDonald T.R. Precautionary principles: a jurisdiction-free framework for decision-making under risk. *Human and Experimental Toxicology* 23 (2004) 579-600.

[157] Mukerjee D. Health risk of endocrine-disrupting ortho-substituted PCBs emitted from incinerators. *Environmental Engineering Science* 15 (1998) 157-168

[158] Calabrese E.J. Toxicological awakenings: the rebirth of hormesis as a central pillar of toxicology. *Toxicology and Applied Pharmacology* 204 (2005) 1-8.

[159] Tuomisto J.T., Pekkanen J., Kiviranta H., Tukiainen E., Vartiainen T. and Tuomisto J. Soft tissue sarcoma and dioxins—a case control study. *International Journal of Cancer* 108 (2004) 893-900.

[160] Tuomisto J., Pekkanen J., Kiviranta H., Tukiainen E., Vartiainen T., Viluksela M. and Tuomisto J.T. Dioxin cancer risk—example of hormesis? *Dose-Response* 3 (2005) 332-341.

[161] Lippmann M. The search for non-linear exposure-response relationships at ambient levels in environmental epidemiology. *Nonlinearity in biology, Toxicology, and Medicine* 3 (2005) 125-144.

[162] Kaiser J. Panel cautiously confirms low-dose effects. *Science* 290 (2000) 6952-6953.

[163] Irigaray P., Newby J.A., Lacomme S. and Belpomme D. Overweight/obesity and cancer genesis: more than a biological link. *Biomedicine & Pharmacotherapy* 61 (2007) 665-678.

[164] World Health Organisation, *Population health and waste management: scientific data and policy options Report of a WHO workshop Rome, Italy, 29-30 March 2007*. 2007.

[165] Elliott 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.

[166] Committee on the Carcinogenicity of Chemicals in Food, Consumer Products and the Environment (2000). *Cancer Incidence near municipal solid waste incinerators in Great Britain*. http://www.iacoc.org.uk/statements/Municipalsolidwasteincineratorscoc00s1march2000.htm

[167] Comba P, Ascoli V, Belli S, Benedetti M, Gatti L, Ricci P, Tieghi A. (2003). Risk of soft tissue sarcomas and residence in the neighbourhood of an incinerator of industrial wastes. Occup Environ Med. 60(9):680-683.

[168] Floret N, Mauny F, Challier B, Arveux P, Cahn JY, Viel JF. (2003). Dioxin emissions from a solid waste incinerator and risk of non-Hodgkin lymphoma. Epidemiology. 14(4):392-398.

[169] Knox E. (2000) Childhood cancers, birthplaces, incinerators and landfill sites. Int J Epidemiol. 29(3):391-397.

[170] Viel JF, Arveux P, Baverel J, Cahn JY. (2000) Soft-tissue sarcoma and non-Hodgkin's lymphoma clusters around a municipal solid waste incinerator with high dioxin emission levels. Am J Epidemiol. 152(1):13-19.

Viel JF, Daniau C, Goria S, Fabre P, de Crouy-Chanel P, Sauleau EA, Empereur-Bissonnet P (2008a). Risk for non Hodgkin's lymphoma in the vicinity of French municipal solid waste incinerators. Environ Health.7:51.

Viel JF, Clément MC, Hägi M, Grandjean S, Challier B, Danzon A.(2008b) Dioxin emissions from a municipal solid waste incinerator and risk of invasive breast cancer: a populationbased case-control study with GIS-derived exposure. Int J Health Geogr. 7:4.

[171] Zambon P, Ricci P, Bovo E, Casula A, Gattolin M, Fiore AR, Chiosi F, Guzzinati S (2007). Sarcoma risk and dioxin emissions from incinerators and industrial plants: a population-based case-control study (Italy). Environ Health.16; 6:19.

[172] Health Research Board, et al., *Health and Environmental Effects of Landfilling and Incineration of Waste – A Literature Review*. 2003, Dublin: Health Research Board. viii, 284p.
[173] http://www.environment-agency.gov.uk/research/library/data/34419.aspx

[174] Health Protection Agency, The Impact on Health of Emissions to Air from Municipal Waste Incinerators, September 2009, http://www.hpa.org.uk/web/HPAwebFile/HPAweb_C/1251473372218

[175] Scotgen (Dumfries) Ltd, Dargavel Energy from Waste Plant, Site Status Report http://www.gainscotland.org.uk/Dum_Scotgen_SiteStatusRptV2_Sep10.pdf

[176] Sunday Herald, 12 September 2010
http://www.robedwards.com/2010/09/exposed-scotlands-99-dirty-polluters.html

[177] Assessment of the Health Risk of Dioxins: Re-evaluation of the Tolerable Daily Intake (TD), WHO Consultation, May 25-29 1998, Geneva, Switzerland

[178] Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment, Statement on the Tolerable Daily Intake for Dioxins and Dioxin-like Polychlorinated Biphenyls, COT/2001/07 (October 2001)

[179] Our Common Future, Report of the World Commission on Environment and Development, World Commission on Environment and Development, 1987.
Published as Annex to General Assembly document A/42/427, Development and International Co-operation: Environment August 2, 1987

[180] EUR-LEX - Access to European Union law
Communication from the Commission on the precautionary principle
http://eur-lex.europa.eu/smartapi/cgi/sga_doc?smartapi!celexplus!prod!DocNumber&lg=en&type_doc=COMfinal&an_doc=2000&nu_doc=1