An Bord Pleanála

Statement of Evidence

Particulate Emissions and Health

Proposed Ringaskiddy Waste-to-Energy Facility

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Vyvyan Howard is a medically qualified toxico-pathologist specialising in the problems associated with the action of toxic substances on the fetus 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 fetus.

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.

A large part of Professor Howard's current research is the investigation of the fate toxicology of nanoparticles. His research team is in receipt of two large EU grants; 'NanoInteract and 'NeuroNano'. He has co-edited a book entitled 'Particulate Matter: Properties and Effects upon Health' published in September 1999 [1].

Vyvyan Howard has sat on two EU expert groups considering the threats and benefits posed by nanotechnology and recently addressed the House of Lords Select Committee on Science and Technology investigating the use of nanotechnology in food.

1 Summary:

1.1 Incineration and Health:

Scientific knowledge regarding the effects of solid waste incineration facilities on the health of a population living nearby is constantly being updated.

Adverse health impacts arising from both inhalation of combustion products and from contaminated food from older incineration plants, generally those operating during the 1970's through to the 1990's, are reasonably well described in the epidemiological literature. The main health endpoints studied have tended to relate to

1. respiratory symptoms and illness
2. reproductive effects, especially congenital anomalies
3. cancer.

A practical issue, and one of significant policy importance, is that the majority of published epidemiological studies relate to these older plants. With the more recent European Union regulations [2] many older plants have closed, or been fitted with more stringent emission controls. While this is obviously 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. Proponents of new facilities tend to dismiss the older research as irrelevant. Opponents take a contrary view arguing, not unreasonably, 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 it's 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.

The comprehensive review by the Health Research Board [3], commissioned by Department of Environment and Local Government, was obviously aware of 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*".

The Health Research Board recognised the problems of isolating causation in real world epidemiology and commented that "*it is hard to separate the influences of other sources of pollutants, and other causes of cancer and, as a result, the evidence for a link between cancer and proximity to an incinerator is not conclusive*". They suggested that this could be addressed by "*further research, using reliable estimates of exposure, over long periods of time, is required to determine whether living near landfill sites or incinerators increases the risk of developing cancer. Studies of specific environmental agents and specific cancers may prove more definitive in the future*".

A more recent World Health Organisation ('WHO') report [4] similarly concludes by suggesting that "*Further insights on health effects of landfills and incinerators are likely to be gained only from studies that consider exposure pathways and biomarkers of exposure and effect, and compare waste–related exposures with those due to other sources of pollution*."

In that context this evidence reviews the possible health impacts associated with emissions from incinerators and a specifically the concerns associated with ultrafine particulates.

1.2 Air Pollution and Health:

The relationship between air pollution and mortality has been well known for many years. Two of the most notable pollution incidents confirming the effects of air pollution were firstly the tragic events of the Meuse Valley, Belgium, where in December 1930, in the small town of Engis 60 people died in the space of three days [5]. This disaster provided incontrovertible evidence that air pollution could kill and therefore it attracted considerable attention from the scientific community.

In a contemporary editorial in the British Medical Journal, Haldane [6] stated that "the possibility of a similar disaster happening in this country [the UK] is a matter of great public health interest". He thought that disaster had been avoided so far in London because the city emitted a lot of heat, which produced convection currents. He warned – though to no avail, against plans to build big electricity generating stations. The subsequent London pollution incident in December 1952 resulted in an increase in deaths that has been estimated to be of approximately 4,000 by Logan (1953) or 12,000 in a more recent retrospective study [7].

Despite these huge impacts, it has not been until the last decade did the scientific community focus in earnest on the potential health hazard of PM exposure [8].

1.3 Particulates and Health:

Epidemiological studies worldwide have consistently demonstrated links between ambient particulate matter exposure and adverse health outcomes, including increased rates of respiratory and cardiovascular illness, hospitalizations, and pre-mature mortality [9, 10]. Particles are usually defined by their size, e.g., PM10 and PM2.5, as the mass of particles with aerodynamic diameters less than 10 to 2.5 μm, respectively. Recently, however, interest has also focused on the fraction of ultrafine particles (UFP) with a diameter less than 0.1 μm, which are abundant in number but contribute little to the mass [11, 12]. The UFPs are only usually measured for research purposes and are effectively outside regulatory control. It is these emissions that are the main theme of this evidence.

Studies have shown that ultrafine particles are more toxic than larger particles [13-15]. Furthermore, individual particles have been shown to be capable of inducing inflammation and oxidative stress [15], suggesting that particle number concentrations, which are dominated by ultrafine particles, may be more indicative of some potential health impacts than particle mass concentrations. UFP are also important because of their high alveolar deposition fraction, large surface area, ability to induce inflammation, and potential to translocate into the blood circulation system. At a given mass, ultrafine particles (diameter < 0.1 μm) have 10^2 to 10^3 times more surface area than particles with diameters in the 0.1–2.5 μm range and approximately 10^5 times more surface area than coarse particles (2.5 μm < diameter < 10 μm) [16]. This surface area-to-mass effect may affect the relative toxicity of particles to respiratory systems, in combination with a higher deposition efficiency of ultra fines in the alveolar region (Hughes et al., 1998).

Estimates of the number of excess deaths on a global scale due to particle inhalation have been made, and they amount to about 2 million/year of which c.370,000 per year are within the EU. The health effects are not limited to lung injuries. They deaths also include

cardiovascular diseases and cancers [17]. It is interesting in the light of these impacts to consider that as recently as 1992 the Lancet editorial was claiming that "*environmental pollution is unlikely to result in gross excess mortality*" [18].

1.4 Ultrafine Particles and Incineration:

Although not such a high contributor to national PM inventories incinerators appear to be very important local sources of particulate contamination. Aboh [17] assessed the contribution of a modern incinerator in Sweden to local PM2.5 levels and concluded that between 17 % and 32% of the particulates arose from the incinerator. This contribution may seem to be large compared with the relatively small increased modelled by Indaver of 0.5 $\mu g/m^3$ compared with an assessed background level of c 7 $\mu g/m^{3.}$ Indaver appears to ignore, however, the very significant contribution made to particulate burdens by SOx and, especially, NOx emissions.

1.5 The Precautionary Principle:

There remains significant uncertainty about the level of health impacts associated with ultrafine particulates and other emissions from incinerators.

The WHO [4] emphasises that "*priority needs for research include development and application of biomonitoring, both in human observational studies and in toxicological research, the use of pharmacokinetic models to assess the influence of factors such as metabolism and timing of exposures, and the analysis of all relevant environmental matrices, in order to evaluate chemical exposure pathways and to assess the exposure for specific subsets of the population*".

I consider that the evidence of risk of harm to human health and the environment is sufficiently high that a precautionary approach should be taken towards the permitting of new incineration capacity at least until there is much better information from the biomarker studies recommended by the WHO [4] and the Health Research Board [3].

Whilst I believe that it is sufficiently compelling in itself the uncertainties associated with the health evidence are supported by strong policy arguments in areas beyond the scope of this evidence. The 2007 WHO report [4] 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*". I note that the Health Research Board review [3] 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.'*"

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:

'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.'

2 Properties of particulates

2.1 Particle Size

In 1979, the U.S. National Research Council said [19] that measuring particles by weight, without regard to particle size, has "*little utility for judging effects*". Particle size is therefore a vital consideration when it comes to air pollution and health. The respirable fraction of particles found in air are classified into size bands which are generally defined as:

Coarse + fine	PM_{10}	The mass of particles per cubic metre which pass through a size-selective inlet with a 50% efficiency cut-off at 10 μm aerodynamic diameter
Fine	$PM_{2.5}$	As for PM_{10} but with a 2.5 μm cut-off.
Ultrafine = UFP or 'nanoparticles'	$PM_{0.1}$	As for PM_{10} but with a 100 nm cut-off, i.e. up to 0.1 μm diameter

It is helpful to compare the size of the particles with common material like fine beach sand and human hair [20]:

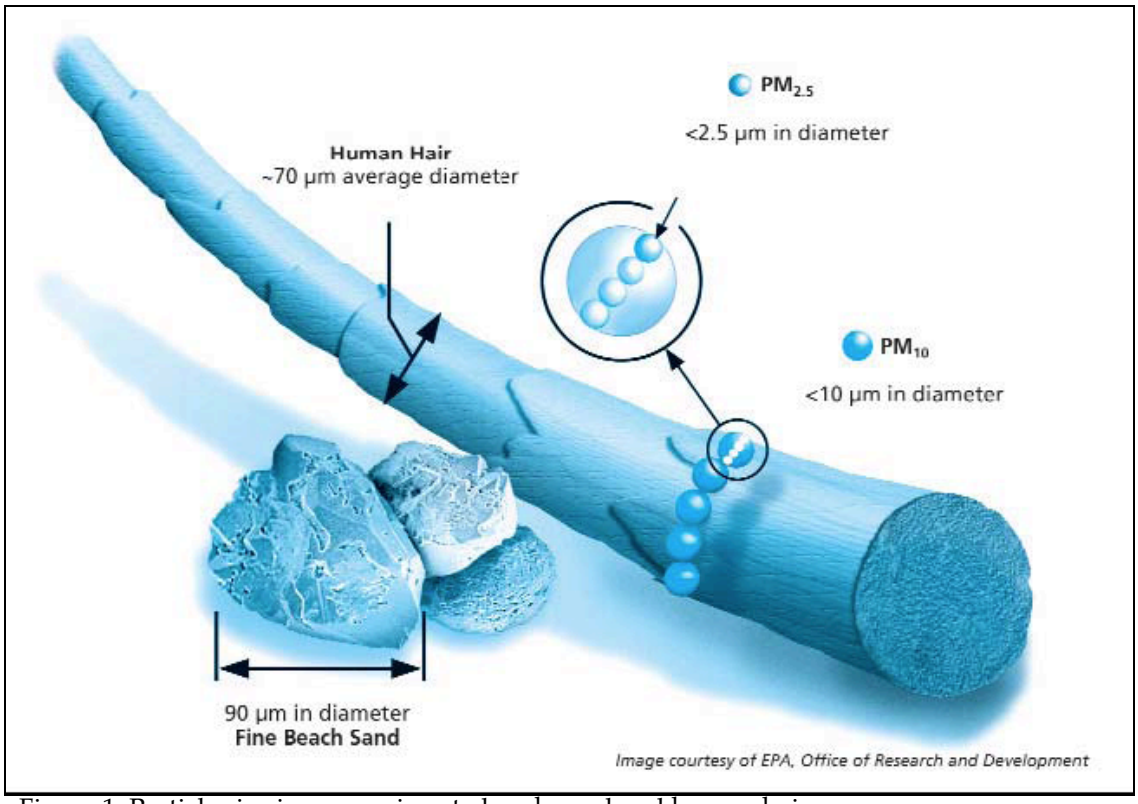

Figure 1: Particle size in comparison to beach sand and human hair

This relative size can also be illustrated by comparison to biological phenomena as per Brook et al. [21]:

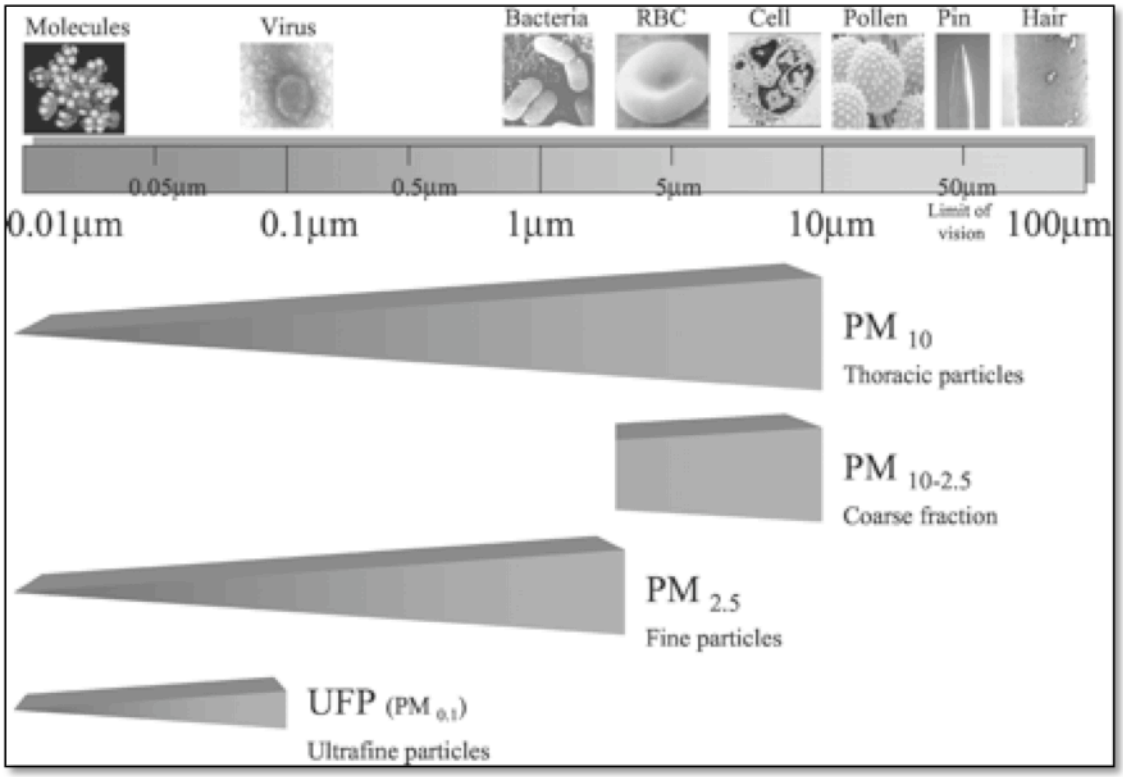

Figure 2: Particle size in comparison to common natural phenomena

The "coarse" particle mode is the difference between PM_{10} and $PM_{2.5}$. It is variable because it includes wind-blown dust and some contribution from building operations; as a 'rule of thumb' $PM_{2.5}$ is normally between 50% and 80% of PM_{10}. [22]

The figure below summarizes what is known about particle size distribution and how size distribution is connected to more common measures of particle number and mass. The percentage values were based on 1995–1998 data from Erfurt [23] and it can be seen that whilst c 97% of the particle mass is found in the components > $PM_{0.1}$ this constitutes only 12% of the particle numbers (note that this is based on total $PM_{2.5}$ levels being 100% of the mass).

	Contribution[a]	
Size (µm)	Number	Mass
Ultrafine particles		
$NC_{0.01-0.03}$ $NC_{0.03-0.05}$ $NC_{0.05-0.1}$	88%	3%
Fine particles		
$MC_{0.1-0.5}$ $MC_{0.5-1.0}$ $MC_{1.0-2.5}$	12%	97%
Total ultrafine and fine particles		
0.01–2.5	100%	100%
Coarse particles		
$PM_{10-2.5}$	—	20%
$TSP-PM_{10}$	—	30%

[a] Based on the data from Erfurt 1995 to 1998: contribution of ultrafine and fine particles to number and mass in the size range of 0.01–2.5 µm and contribution of coarse particles to mass of total aerosol size distribution.

Size Ranges and Contribution to Number and Mass Concentration [23]

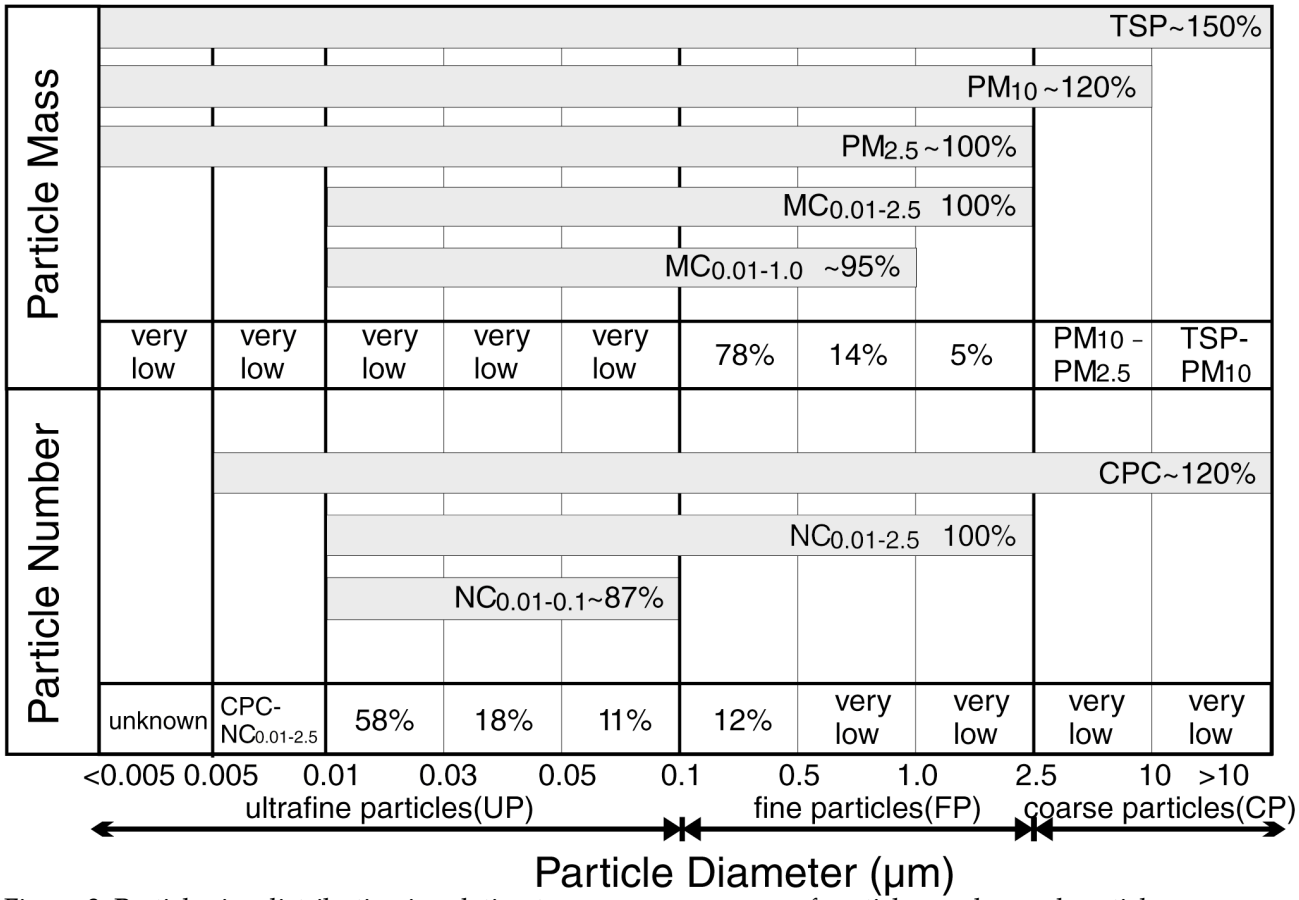

Figure 3: Particle size distribution in relation to common measures of particle number and particle mass

It is clear, therefore, that depending on their sizes, quite substantial differences in numbers or surfaces might constitute the same mass. Just one particle per cm^3 with a diameter of 2.5 μm is sufficient to result in a mass concentration of 10 $\mu g/m^3$ whilst more than two million particles of a diameter of 0.02 μm are needed to obtain the same mass concentration.

During the past 20 years, studies have largely been able to rule out sulphur dioxide and ozone pollution as the cause of the observed deaths although ozone is associated with increased mortality in daily time series studies (0.3–6.7% increase per 20 $\mu g/m^3$) and there is a weak association between SO2 and mortality (about 1% increase per 50 $\mu g/m^3$) which can be difficult to separate from particulate co-pollutants [24].

2.2 Ultrafine particles

Ultrafine particles (UFP) or nanoparticles[1], are very small pieces of matter defined as having dimensions less than 10^{-7} m. They constitute a small proportion of the mass of almost all types of particulate material. They also constitute the majority of the number of particles found in aerosols produced as a result of combustion processes. Their importance in the field of catalyst manufacturing, where their high surface area has a very great influence on reactivity, is widely known [25]. However, at present we know relatively little about their detailed structure, or their chemical and physical properties.

[1] Nanoparticles are smaller than 100nm, but in this evidence I take the terms to be interchangeable.

2.3 History and Regulation:

Regulation in Ireland of particulates as an air pollutant has been based on PM_{10} (particles of <10 μm) and, more recently on $PM_{2.5}$ – although not, so far as I am aware for setting emission standards from processes like incinerators.

In common with many leading researchers in this developing field of nano-toxicology such as Donaldson's [26] and Oberdörster's [27] groups, I have long considered ultrafine particles to be the main contributor to its adverse effects. Though UFP is only a small fraction of PM_{10} , Seaton et al. in 1995 [28] hypothesised biochemical processes whereby it might be the cause of acute cardiovascular effects. The 1999 Royal Society conference "Ultrafine particles in the atmosphere" and proceedings, published in 2000, consolidated the new thinking.

Urban air will often contain 100 billion (10^{11}) one-nanometre-diameter particles in each cubic meter of air, all of them invisible. By weight, these 100 billion particles will only amount to 0.00005 micrograms yet they may be responsible for much of the health damage created by fine-particle pollution. It is clear, therefore, that achievement of a regulatory standard does not ensure protection of health.

2.4 Lack of Standards and Monitoring for UFPs

Standards and monitoring are now being introduced for $PM_{2.5}$ particles – termed 'fine particles' and mostly 1,000 to 2,500nm in size – but there is nothing yet to cover the much smaller ones. The current standards are in terms of total mass, yet UFPs are generally around only one percent of the total mass but present the majority of the surface area that is reactive to human tissues. If the mass of a single inhaled 2.5 μm particle is divided into typical nanoparticles ~80nm, they would have 1000 times more surface area. For that reason alone, the mass-based PM standards are far from appropriate for UFPs.

Wichmann [23] reported some of the earliest epidemiology relating to UFPs and they showed a full distribution over particle sizes in urban air:

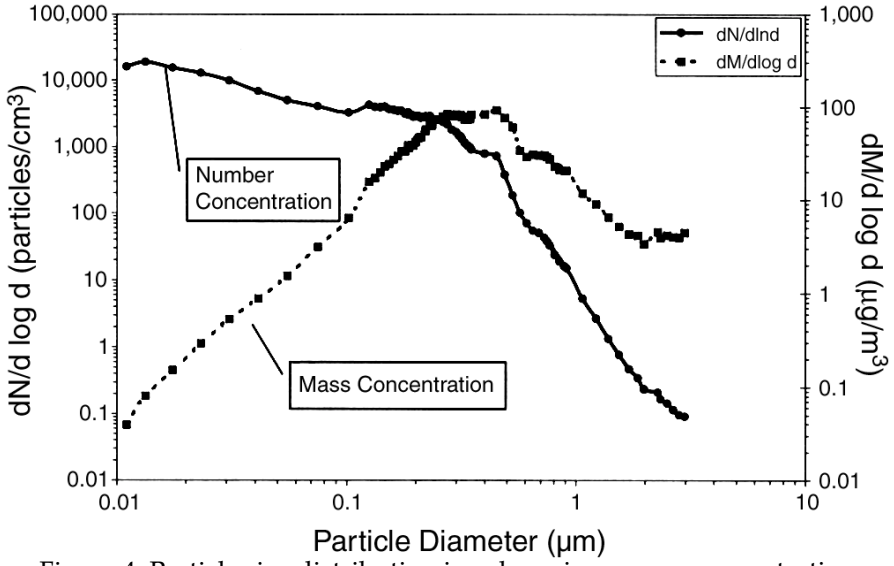

Figure 4: Particle size distribution in urban air mass vs. concentration

This does not show PM10 (cuts off at 3μm) but does indicate that most of the mass is in 0.2 to 0.5 μm particles, yet most of the particles ('number concentration') are under 0.2 μm (i.e. 200 nm).

2.5 Atomic Structure of Nanoparticles

It is only in the last twenty-five years, with the advent of high-resolution electron microscopy (HREM) at 0.1 nm (nanometre) levels, and the consequent ability to resolve inter-atomic spacings at this level, that any real attempt has been made to determine the atomic structure of ***individual*** particles. What has been learned is that these minute particles have an increasing proportion of surface atoms as the particle size decreases. Novel configurations of atoms have been demonstrated in nanoparticles, which cannot exist in the bulk material (Jefferson & Tilley, 1999). The imbalances between the number of atoms and number of electrons means the particles can be electrically charged and have raised chemical reactivity.

3 Damage to Health from Particulates

3.1 Fine Particles Linked to Human Deaths

US studies from the 90s first established that urban particulates in modern times were causing people to die. The 6-cities study of 1993 (Dockery et al.) was followed by the ACS study of half a million adult Americans in 151 metropolitan areas, which clearly established the relationship between fine-particle air pollution and human deaths, ruling out smoking as a cause of the observed deaths (Pope *et al.* 1995, Villeneuve *et al.* 2002, Pope *et al* 2002). 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.

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. This research was vehemently attacked from a number of quarters, particularly those industries potentially most affected by the findings, which labelled it 'junk science'. However, an independent scientific panel conducted a thorough 're-analysis' and confirmed that tiny soot particles can shorten lives (HEI 2000). This basic finding was supported by a European study that found 6% of all deaths correlate with urban concentrations of fine particles, mainly from traffic [29].

The review of air pollution under the European Commission (Clean Air for Europe: CAFÉ) assisted by the WHO led to the Commission declaring in the *Thematic Strategy on Air Quality* that "*serious air pollution impacts persist*" [30].

The Commission also said "*currently in the EU there is a loss in statistical life expectancy of over 8 months due to $PM_{2.5}$ 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).

3.2 Effects of Particle Types and Mixtures

The effect of mixtures of particles of differing chemical composition entering the blood stream via the lungs in large numbers on a daily basis is beginning to be understood. There is no doubt that some particulate aerosols are indeed hazardous. However the degree of hazard associated with specific types of particle and the precise mechanisms by which exposure leads to pathology are as yet poorly understood and currently the subject of increasingly intense research.

Boekelheide [31] reported that pregnant rat dams were exposed to mixtures of phthalates (suppressors of testosterone synthesis within the fetal testis) and androgen receptor antagonists (acting at the end organs of this signalling pathway). The exposures were orchestrated so that any agent alone had very limited effects while the collective exposure robustly induced hypospadias and epididymal agenesis in the developing males. Overall, the chemicals clearly acted with dose additivity, not response additivity. These effects were induced by chemicals acting by different molecular mechanisms within different organ

systems with different absorption, distribution, metabolism, excretion patterns, and differently shaped dose response curves. By all of our familiar criteria, these chemicals are not toxicologically similar and do not share a mode of action as defined by the USEPA; and yet they can act together to inhibit this developmentally sensitive signalling pathway.

3.3 Threshold Levels

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.

3.4 Respiration of particulates:

The average human lung contains about 2,300 km of airways and 480 million alveoli [32, 33]. On a daily basis, humans inhale around 10,000 litres of ambient air, which comes in close contact with a lung surface area of between 75 and 140 m^2. From this, 350 litres of oxygen diffuses across the alveolar capillary basement membrane into the 10,000 litres of blood flowing through the lungs daily [34]. The respiratory tract, therefore, comes into close contact with a large volume of ambient air and its components on a daily basis – the potential for uptake of contamination contained within that air is obvious.

Whilst US researchers switched to correlating $PM_{2.5}$ with health indicators authorities in Europe have tended to remained entrenched with the concept of $PM_{10.}$ There is, however, no longer and serious doubt that the size of the particles is the most important issue from a public health viewpoint and 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 [35]. The figure below shows whilst that 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, $PM_{2.5}$, and ultrafine PM, $PM_{0.1,}$ enter the nonciliated alveolar regions and deposit deep within the lungs.

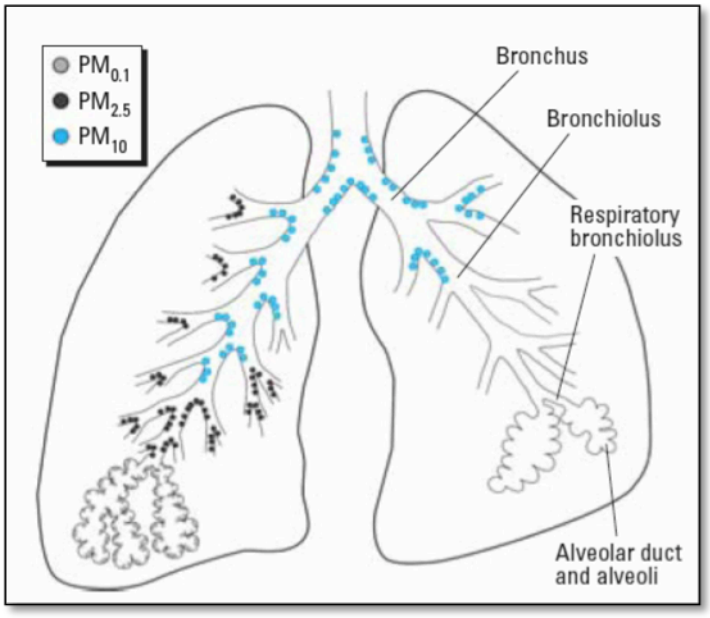

Figure 5: PM in the lungs (from [35])

Not all particles are retained. Larger particles deposit in the airways or mouth and throat, whereas smaller particles deposit in the alveolar region. A higher proportion of particles <1 μm that than those of $PM_{1.0}$ can be exhaled, thereby reducing deep lung deposition:

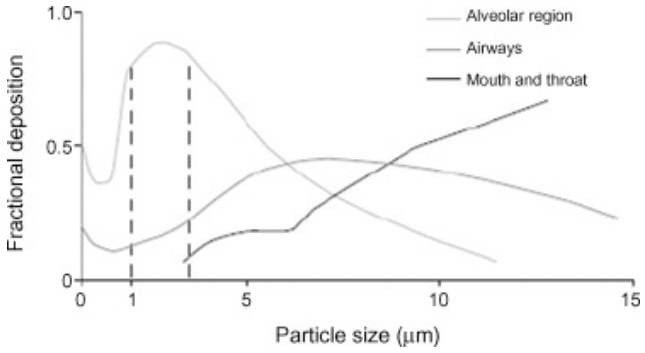

Figure 6: The effect of particle size on the deposition of aerosol particles in the human respiratory tract following a slow inhalation and a 5 s breath hold (from [33])

3.5 Fate of particulates deposited in the lung

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. 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; Donaldson et al. 1999), 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. Gumbleton [36], and more recently, Yang [33] have reviewed nanoparticle mobility and removal mechanisms including endocytosis. UFPs can cross biological membranes, in common with many viruses, and their mobility within the body is thought to be high.

3.6 The mechanism of toxic action

I have summarised and discussed a number of mechanisms by which UFPs can induce cell damage in my 2009 nanoparticle review for the WHO. Unfortunately this is not yet in the public domain and cannot yet be supplied to this inquiry. I will, however, briefly review some of the key developments here.

In recent years it has been established that Ultrafine particles:

- have a high specific surface area, which can catalyse reactions and adsorb high amounts of toxic substances (like PAH), providing a carrier deep into the lung during inhalation [28];

- have a higher deposition probability particularly in small airways and the alveolar region of the lungs than fine particles [11];

- respond differently in men and women - Women receive a greater dose than men in the head and tracheobronchial regions, for example [37];

- are less well phagocytized by alveolar macrophages than larger particles and inhibit their phagocytic ability [38];

- are taken up by other cells of the respiratory epithelium, such as epithelial cells, dendritic cells [39, 40];

- may form complexes with proteins and biomolecules which may result in functional changes of the latter [41];

- have greater access to interstitial spaces than larger particles [42, 43]);

- have access to the blood circulation [43-45];

- induce more oxidative stress than fine particles [15, 46] ;

- cause more pro-inflammatory responses than larger particles [47] ;

- 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 [48] ;

- adversely affect cardiac functions and vascular homeostasis [49];

- affect the immune system [27].

For all of these hypotheses there exists a growing body of studies on a mechanistic level providing plausibility or evidence, however, on different levels of causality. From many of these studies it became also clear that the hypotheses listed above may only be applicable to susceptible organisms and individuals predisposed either by disease, genetics or age while the healthy organism does not show any such sensitive reactions.

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.

The 1995 hypothesis of Seaton *et al.* [28] suggested that the particles retained in the deep lung cause inflammation which, in turn, releases natural chemicals into the blood stream causing coagulation of the blood. This was to explain epidemiological findings of increased cardiovascular disease in populations exposed to higher than average PM_{10} exposure [50]. There may be a low exposure threshold, above which these effects will occur, but it appears the classical toxicological dose-response curve is not appropriate. The main end point under investigation is arterial damage, which is consistent with the 1965 findings of Aurerbach that smokers, who voluntarily inhale particulate aerosols, almost all sustain arterial damage themselves.

In vivo studies performed on laboratory animals have looked at the ability of UFPs to produce inflammation in lungs after exposure to UFP aerosols [26, 47, 51, 52] . The degree to which UFPs appear to be able to produce inflammation is related to the smallness of the particles, the 'age' of the aerosol and the level of previous exposure. It has been hypothesised [28] that the chronic inhalation of particles can set up a low grade inflammatory process that can damage the lining of the blood vessels, leading to arterial disease.

Most health studies are now using $PM_{2.5}$, though as runs of data in Europe tend to be of PM_{10}, uncertain corrections are often made. There are few data runs for ultrafine particles ($PM_{0.1}$), despite the finding [53] that they were on an increasing trend (while PM_{10} was decreasing) and probably more hazardous.

3.7 UFPs penetrating into the human body

There is considerable evidence to show that inhaled UFPs can gain access to the blood stream and are then distributed to other organs in the body [54].They can even cross the placental barrier.

One needs also to compare the particle sizes with biology, as in figure two above from Brook et al. [21]. UFPs are much smaller than bacteria, against which cells can defend themselves, and of similar size or smaller than viruses, which can relatively easily penetrate between cells.

The 'passageways' for nanoparticles into and then subsequently around the body are the 'caveolar' openings in the natural membranes which separate body compartments. These openings are between 40 and 100 nm in size and are thought to be involved in the transport of 'macromolecules' such as proteins, including on occasion viruses. They also happen to be about the right size for transporting UFPs. Most of the research on that, to date, has been performed by the pharmaceutical industry, which is interested in finding

ways of improving drug delivery to target organs. This is particularly so for the brain, which is protected by the 'blood brain barrier' which can be very restrictive. This has been reviewed by Gumbleton [36].

Although there are clear advantages to the intentional and controlled targeting of 'difficult' organs, such as the brain, with nanoparticles to increase drug delivery, the obverse of this particular coin needs to be considered. When environmental UFPs (such as from traffic pollution or incineration) gain unintentional entry to the body, it appears that there is a pre-existing mechanism which can deliver them to vital organs [36]. The body is then 'wide open' to any toxic effects that they can exert. The probable reason that we have not built up any defences is that any such environmental toxic UFPs were not part of the prehistoric environment in which we evolved and therefore there was no requirement to develop defensive mechanisms.

Peters et al. [55] having established the vulnerability of remote organs – and particularly the brain - wrote "*The results indicating that particles may contribute to the overall oxidative stress burden of the brain is particularly troublesome, as these long-term health effects may accumulate over decades*". They stressed the need for increased efforts to quantify the relative risks for long-term particle exposure on the onset of Parkinson's and Alzheimer's disease adding "*both Parkinson's and Alzheimer's disease are only diagnosed once manifest clinical signs and symptoms are evident and impact the diseased persons by long years of disabilities and diminished quality of life*". The exposure of the brain to UFPs is a matter of great concern - if our limited capacity to deal with misfolded protein is exceeded then the likely sequelae would be an increase in the incidence of protein misfolding disease in the general population and a tendency to an earlier average age onset.

3.8 Quantifying the Established Health Impacts

A range of impacts have been reported by different researchers for different outcomes. Kunzli [56], for example, reported elevations of 10 $\mu g/m^3$ and 20 $\mu g/m^3$ in $PM_{2.5}$ were associated with 5.9% and 12.1% increases in the development of atherosclerosis in "*healthy*" people who had no previous signs of acute coronary syndromes, but had small elevation of low-density lipoprotein.

Miller et al. reported an increased relative risk of 1.76 for death from cardiovascular disease for every increase of 10 μg per cubic meter in the mean concentration of $PM_{2.5}$ [57].

By comparison, a study by the American Cancer Society showed that each increase of 10 μg per cubic meter in the mean $PM_{2.5}$ concentration was associated with an increased relative risk of 1.12 for death from cardiovascular disease, 1.18 for death from ischemic heart disease (the largest proportion of deaths), and 1.13 for death from arrhythmia, heart failure, or cardiac arrest [58].

Commenting on these data in an editorial of the New England Journal of Medicine Dockery [59] wrote:

"A multifaceted approach that encompasses both public health and medical interventions is needed to reduce the burden of cardiovascular disease attributable to air pollution. Comprehensive management of the harmful effects of fine particles must start with intensive efforts to reduce this destructive form of air pollution. Fine particulate air pollution results not only from the combustion of carbonaceous fuels in our vehicles, power plants, and factories but also from secondary particles produced by oxidation of gaseous pollutants emitted by these same sources".

I note that these secondary particles have not been considered in the application at all and have not been incorporated in the (very limited) assessment of risks. It is clear however that even without the consideration of secondary particulates it is not reasonable to describe the particulate emissions from the proposed incinerators as having no impacts.

3.9 Children as vulnerable and sensitive sub-population:

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* [60] reviewed factors affecting children's susceptibility, effects on pregnancy outcomes, infant and childhood mortality, lung function development, asthma and allergies, neurobehavioural 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 [61], 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. It's now recognised that the inhibition of children's lung development can be very serious, potentially meaning long term harm to their respiratory health. Evidently air pollutants, most probably including particulates, cause harm to children differently to adults.

The expert science view, summarised by Joel Schwartz [62] 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. The review by Heinrich and Slama [63] 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. [64] found bronchitis in early childhood correlates with $PM_{2.5}$ and PAH levels (UFPs may be a carrier for PAH – see above). While these findings may not all be conclusive, there can be no doubt that children and even the fetus are particularly vulnerable to particulate air pollutants – while this has largely been overlooked in setting current standards and controls.

A review of health effects of poor air quality on children's health [65] emphasised the hazards associated with the siting of major particle-emitting plants and roads in the vicinity of schools or communities containing children.

3.10 Prenatal Exposure:

A 2007 Editorial [66] in the Journal "Reproductive Toxicology" summed up the increasing concerns associated with prenatal exposure admirably:

"There is a major paradigm shift taking place in science that while simple is profound. It states that the root of many diseases, including reproductive diseases and dysfunctions, will not be found by examination of disease onset or etiology hours, days, weeks, or even years prior to disease onset. The new paradigm suggests that susceptibility to disease is set in utero or neonatally as a result of the influences of nutrition and exposures to environmental stressors/toxicants. In utero nutrition and/or in utero or neonatal exposures to environmental toxicants alters susceptibility to disease later

in life as a result of their ability to affect the programming of tissue function that occurs during development. This concept, that is still a hypothesis undergoing scientific testing and scrutiny, is called the developmental basis of health and disease".

There is a growing recognition of the importance of the prenatal period as a ''window of exposure'' for the development of childhood, and possibly adulthood, disease [67]. Henderson et al. [68] have investigated the effects of mothers' exposure to household chemicals during pregnancy, but they acknowledged the difficulty in determining whether the reported health effects could be attributed to pre- or postnatal exposure, or even both. They observed that chemical use in the home before and after birth was highly correlated, making it difficult to separate potential effects of exposure during these periods.

Jedrychowski et al. [69] reported that prenatal exposure to $PM_{2.5}$ particulate matter had a moderate but significant impact on severity of respiratory illness in postnatal early life. The biological mechanisms whereby prenatal $PM_{2.5}$ exposure might cause adverse health outcomes in children are yet unclear. $PM_{2.5}$ is a proxy measure of a whole complex of toxic agents present in the environment – including PAHs – that could adversely affect growth and maturation of lung in early childhood.

Fine particles are usually a product of combustion processes that generate other toxic agents which may interact at the molecular level with DNA as described by Perera et al. [70]. Prenatal exposure to immunotoxic fine particles may impair the immune function of the fetus and subsequently may be responsible for an increased susceptibility of newborns and young infants to respiratory infections.

The synergism of recently proposed role of sulphur dioxide metabolites as inhibitors of enzymes and antioxidants and the adverse effects of nitrogen oxide metabolites in the early embryonic development may lead to symmetric intrauterine growth restriction and premature delivery or low birthweight. The research is directed to point out the toxics from coal combustion products as neglected causes of oxidative stress on human embryogenesis, prematurity, and low birthweight. [71]

3.11 Future Research:

Cormier et al [35] have reviewed the evidence for potential health impacts of particulate emissions from combustion processes. They posed a series of questions that require addressing:

- How are combustion-generated fine PM and ultrafine PM formed?
- How do their chemical properties differ from larger PM?
- What is the nature of association of chemicals with these particles?
- How is the chemical and biological reactivity of these chemicals changed by association with the particles?
- What is the role of PM-associated persistent free radicals in the environmental impacts of fine and ultrafine PM?
- What is the role of PM on cell/organ functioning at initial sites of exposure?
- What is the bioavailability of these particles to other tissues?
- How are these particles translocated to these secondary sites, and do their chemical properties change en route?
- How does acute/chronic exposure lead to adverse organ pathophysiology? Is developmental timing of exposure important?
- What effect does exposure have on predisposing to disease states or on disease progression?

- Most important, what are the specific cellular and molecular mechanisms associated with airborne exposures?

Medical science has been rather slow to fully recognize and explore the serious problems that particulate emissions cause. In spite of the thousands of papers that have been published over the past decade on the issue of UFPs it will inevitably be many years before the answers to all the questions posed are available. Meanwhile it is sensible that particulate emissions, especially those produced in conjunction with toxic chemicals, are reduced so far as possible and that new sources are avoided.

4 Particulate Releases from Incinerators

Modern incinerators are a major source of fine particulate emissions. In 2007, for example, Widory et al. [72] found:

"The main sources of atmospheric particle pollution in Paris are vehicles, central heating and waste incinerators".

It is important to bear in mind that the contribution is not just direct PM emissions, which are now relatively low in terms of total mass and emission concentrations (though not in terms of numbers). Particulate emissions and impacts also include secondary inorganic compounds which can account for a major fraction of PM_{10}, and especially of the $PM_{2.5}$ mass [73]. Almeida [74] found lower but still significant contributions from these secondary particles.

As NO_x emissions from modern incinerators are still rather high (I understand that they normally operate close to the 200 mg/m^3 emission limit) then because of the increased size of modern plants compared with those operated in the early 1990's total levels are of the same order as historically – and the NO_x emissions can form nitrates with metals in the incinerator plume and thus increase the toxicity and availability of the emissions as described by Moffet [75]:

"The frequent observation of these metal-rich particles in an urban area with a high population density also has important implications for health effects. The largest fraction of the Pb-containing particles is less than 2.5 μm, meaning that these particles may be efficiently inhaled. Also, there may be important health ramifications if salts such as $Pb(NO_3)_2$ are formed because lead nitrate is soluble, and therefore more mobile within the human body".

Indaver appear to have completely omitted any consideration of secondary particulates and their impacts from their assessment.

Table 9.2 of the application shows that the proposed Ringaskiddy incinerators would produce 125,486 Nm^3/hr from the grate incinerator and 116,995 Nm^3/hr from the Fluidised bed incinerator i.e a total emission of 242,481 Nm^3/hr. The permitted particulate emission standard, subject to statistical limits, would be 10 mg/m^3 and for oxides of nitrogen 200 mg/m^3. Daily emissions could therefore total 5,819,544 m^3 containing 58.2 kg of particulates and 1,164 kg of NO_x .

These are large emissions in any terms – without any consideration of secondary particulates the authorised incinerator emissions would have the potential to daily fill a space 11km x 11km by 50 m deep to the WHO annual guideline of 10 $\mu g/m^3$ for $PM_{2.5}$.

Secondary particles should, of course, be considered in any case. The formation mechanism of nitrates as secondary particles is illustrated below [76]:

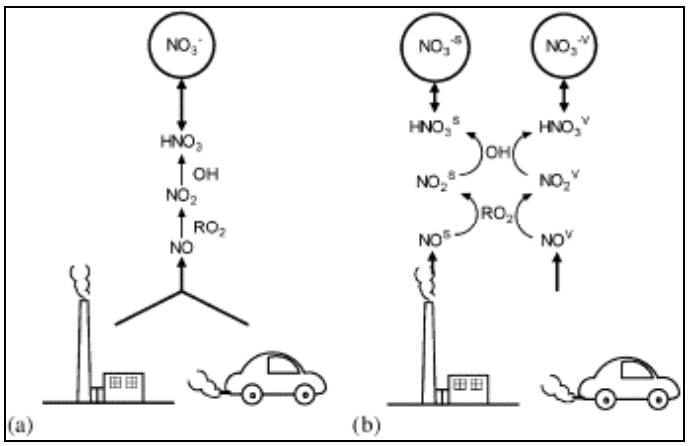

Figure 7: Illustration of source apportionment for secondary PM2.5 nitrate from two sources. (a) Formation of secondary PM2.5 nitrate in traditional air quality model using lumped NO emissions. (b) Formation of secondary PM2.5 nitrate from NO emitted from two sources tracked separately in the source-oriented air quality model used by Ying (from [76]). RO_2 represents a peroxy-type radical, and OH represents hydroxyl radical.

Furthermore emissions from an incinerator installed with a selective non-catalytic reduction (SNCR) NO_x control system as proposed here may actually increase direct emissions of ammonium nitrate which is an important component of $PM_{2.5}$

The efficiency of the filter is therefore not the most significant aspect of the total particulate emission and control of NOx (and to a lesser extent SOx is actually more significant in terms of the contribution to ground level concentrations although neither appear to have been modelled in this application.

4.1 Filter Efficiency:

The proposed incinerator would use a bag filter as the main primary particulate abatement technology. For a given fibrous filter, there is a particle size, usually between 0.05 and 0.5 μm that has the minimum collection efficiency [77] ; that is, all particles, larger or smaller than this size, are collected with greater efficiency. For a given size particle, there is also a velocity for minimum collection efficiency. It is important to establish where this minimum efficiency lies, what the particle density of the emissions at that point are and what the speciation of contaminants (both metals and products of incomplete combustion) carried by those particulates is.

Waste incinerators with the most modern bag filter technology for clean-up of flue gases still emit an aerosol of ultrafine particles, unlimited by legislation [78-81].

Collection efficiencies for particles < 2.5 μm are between 5 and 30% before the filters become coated with lime and activated carbon.

Particle size	Collection efficiency
PM10's	between 95% and 98%
PM 2.5's	between 65% and 70%
PM below 2.5	between 5% and 30%

Efficiency of baghouse filters for particles of differing sizes as claimed by operators. (Onyx 1999)

Though there have been improvements since 1999, the bag filter technology generally used on municipal waste incinerators is not efficient at filtering very fine particles. For particles of less than 1 μm down to about 0.2 μm the abatement efficiency is low. Although very high capture rates, based on gravimetric indices, are generally claimed, the majority by number of ultrafine particles will pass through and current standards do not take into consideration the sizes of the particles emitted by an incinerator. Thus modern plants with their very high gas fluxes are guaranteed to produce an ultrafine particulate aerosol.

Aboh [17] concluded that depending on the number of variables considered, waste incineration and local sources contributed between 17 and 32 percent of $PM_{2.5}$. Whilst the quantitative contribution from the different sources may be treated as indicative since the number of observations were small compared to the number of variables relative strength of the identified sources was seen to change when the variables included in the analysis were varied in number and character, although the same sources remained:

	Waste incineration and local sources	Oil incineration	Biomass burning	Long distance transport (LDT)	Traffic emissions
19 variables	32	33	18	16	1
14 variables	28	29	9	23	12
8 variables	17	21	7	41	14
6 variables	24	11	8	51	6

Ogulei [82] 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.

Ultrafine particle concentrations have been shown to be raised in the plume of a hospital incinerator[3] 350 metres downwind of the plant [84].

4.2 Bimodal Size distribution

It has been known for many years that Aerosol emissions from combustion processes including waste incineration tend to show a bimodal mass distribution with a peak of coarse particles and another of ultrafines *[85, 86].*

Friedlander [87] wrote:

The coarse mode consists of particles with diameters in the range between 1μm and about 100μm. In pulverized coal combustion they are formed from the nonburnable mineral inclusions within the fuel particles (Flagan and Friedlander, 1978). In addition to the large fly ash particles there often exists a

[3] The ratio of SO_2/NOx is greater than from vehicle emissions suggesting a fuel of higher sulphur content and discounting a gas fired boiler as an alternative source.

mode of small submicron sized particles which pose a health risk because they are inhalable and may be enriched in toxic metal compounds.

Friedlander pointed out, as we return to below, that the submicron particles are usually less efficiently captured by filter devices and hardly fall under gravity so remain longer in the air .

Ruokojarvi [88] found that half the particle mass in incinerator emissions was under 1.6*μm*, the remainder in a broad distribution up to 14.5*μm.*

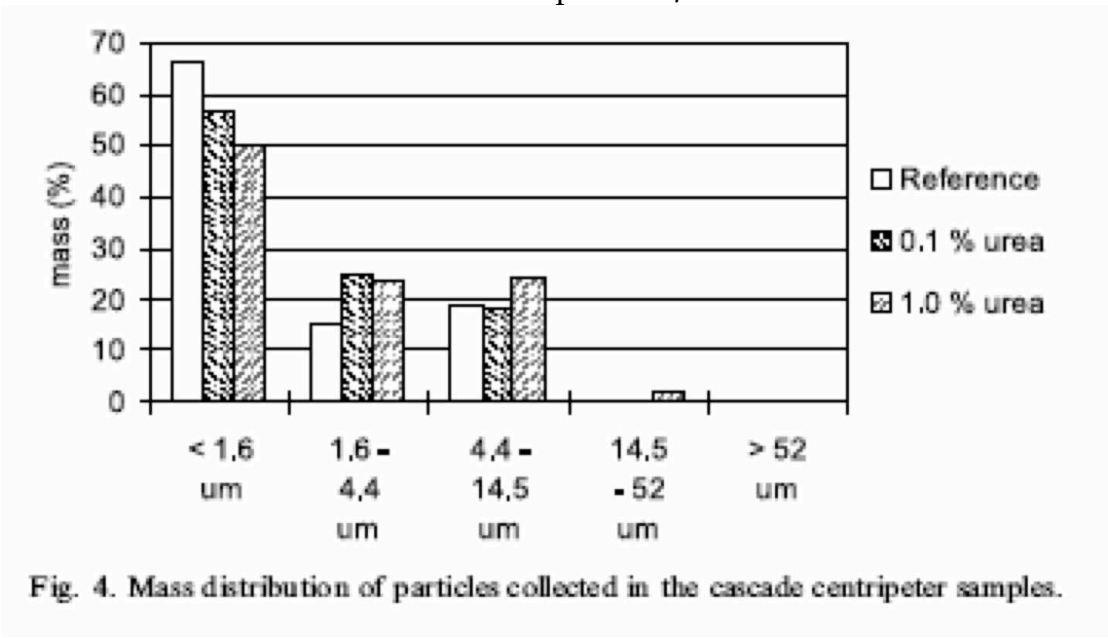

Fig. 4. Mass distribution of particles collected in the cascade centripeter samples.

This figure shows that half the mass is below 1.6μm, somewhat less than in the urban air of Wichmann [23] but it doesn't show the UFPs. Little information has been provided on particles under 1 μm size as the industry is uncomfortable over the issue. Some other data is given below.

4.3 Surface Area of incinerator particles:

The US EPA [89] characterisation of incinerator particulate emissions in the Table below showed that particles <0.7 μm have half the total surface area. Insofar as surface area in contact with lung's surface (epithelium cells) is relevant to exposure/dose effects, the smallest particles carry high weighting, unlike where the total mass (PM index) is considered.

Particle Diameter (μm)[a]	Particle Radius (μm)	Surface Area/ Volume	Fraction of Total Weight	Proportion Available Surface Area	Fraction of Total Surface Area
>15.0	7.50	0.400	0.128	0.0512	0.0149
12.5	6.25	0.480	0.105	0.0504	0.0146
8.1	4.05	0.741	0.104	0.0771	0.0224
5.5	2.75	1.091	0.073	0.0796	0.0231
3.6	1.80	1.667	0.103	0.1717	0.0499
2.0	1.00	3.000	0.105	0.3150	0.0915
1.1	0.55	5.455	0.082	0.4473	0.1290
0.7	0.40	7.500	0.076	0.5700	0.1656
<0.7	0.40	7.500	0.224	1.6800	0.4880

Total surface area: 3.4423 μm^2

Notes: a. Geometric mean diameter in a distribution. Distribution from EPA (1980).

Research has shown that even normally harmless bulk materials tend to become toxic when divided into ultrafine particles. Generally, the smaller the particles, the more reactive and toxic their effect [51, 52]. This is no surprise, because catalysts to enhance industrial chemical reactions are commonly made this way. Making surfaces that are irregular on the scale of just a few hundred atoms creates an enormous area of reactive surface. It is on this surface that catalytic reactions, such as the formation of halogenated organic molecules, can occur. Indeed, because of surface roughness, ash particles can have surface areas 20-30 times the surface area of equivalent spheres [90]. Some of the most reactive nanoparticles to have been studied to date are metals and spinel metal oxides [25]. The upper size limit for such enhanced toxicity of UFPs is not well defined but is generally given between 65 and 200 nm.

4.4 Speciation – inorganic components

Although the particles emitted from large-scale industrial combustion sources are all predominantly in the fine-particle range, their chemical compositions varies substantially depending largely upon fuel types and boiler or furnace operating conditions. This can be illustrated using the fractional abundances of the elements and chemical compounds in the particulate emissions[91].

Typical chemical abundances in source emissions

Source	Dominant particle size	Chemical abundance (mass fractions)			
		>10%	1–10%	0.1–1%	<0.1%
Coal-fired boiler	Fine	Si	SO_4^{2-}, OC, EC, S, Ca, Fe, Al	NH_4^+, P, K, Ti, V Ni, Zn, Sr, Ba, Pb	Cl, Cr, Mn, Ga, As, Se, Br, Rb, Zr
Incinerator	Fine	NH_4^+, Cl, SO_4^{2-}, OC	NO_3^-, Na, EC, Si, S, Ca, Fe, Br, Pb	K, Al, Ti, Zn, Hg	V, Mn, Cu, Ag, Sn
Residual oil boiler	Fine	S, SO_4^{2-}	Ni, OC, EC, V	NH_4^+, Na, Zn, Fe, Si	K, OC, Cl, Ti, Cr, Co, Ga, Se
Wood waste boiler	Fine	K	Na, Fe, Mn	Zn, Br, Cl, Rb	Cr, Cu, Co, Ni, Se, Cd, Ar, Cr, Pb

Key: OC = organic carbon, EC = elemental carbon.

This indicates incinerators are special for Pb, Hg and Br emissions (none of which come in particulates from vehicle emissions).

4.5 Particle Speciation:

Metal emissions from incineration of solid wastes are impacted by compositions of feedstocks and the chemical form of the metals depends on the operating conditions of the incinerator (Wey et al. [92]). A number of studies have identified the 'signature' of incinerators from the metal species. Harrison et al. reported on Birmingham air sampling in 1997 [93], finding zinc and copper to indicate an incineration source. They saw this as the large municipal refuse incinerator within the city (Tyseley), which at the time of sampling was not subject to the tighter Waste Incineration Directive limits.

In the city of Seoul, Mishra et al. [94] found via principal components analysis suggest incineration and the iron and steel industry as possibly significant sources of Pb in particulate matter. Doucet and Carignan [95] examined lead isotopes in French lichens and flyash from different municipal solid waste combustors in the Rhine valley and in other areas of France, concluding that "*these plants* (ie the incinerators) *might be an important source of industrial Pb in the atmosphere*".

Pancras reported [96] "*Large but brief 1.5-h excursions in Zn, Cd, and Pb were found to correlate with winds from the direction of an incinerator in Florida at 17km distance*".

4.6 Speciation – volatile and organic components

Out of over 11 million known chemicals, about 100,000 are being produced on industrial scale and about 1,000-2,000 new chemical entities are being introduced each year [97]. Any of these industrial chemicals may be disposed of by incineration and there is a near infinite number of possible combustion and incomplete combustion products that may be emitted either as particulate matter or by adsorbtion onto or reaction on the surface of particulates. Even if these emissions were monitored, and the vast majority are not, then little or nothing is known about the possible health impacts of the bulk of these emissions.

Volatile chemicals condense on particle surfaces as the incinerator exhaust gases cool. Their concentration on smaller particles is higher, being related to surface area rather than particle mass. This has been subject to particular studies for dioxin and dioxin-like chemicals, but is likely to be similar for many others e.g. [98]. It also holds for volatile chemicals that incinerator UFPs pick up from urban air, specifically the PAHs from vehicle emissions. These cannot penetrate into the body as gases, but if attached firmly to UFPs can be carried through the lung epithelium.

4.7 Range of chemicals coating the particles

There are thousands of chemicals emitted by incinerators. Jay and Stieglitz [99] identified 227 individual organic compounds[4] corresponding to ca. 42% of the total organic carbon

[4] Including: acetic acid, acetone, acetonitrile, aliphatic alcohol, aliphatic amide, aliphatic carbonyl, anthraquinone, benzaldehyde, benzene, benzoic acid, benzoic acid methyl ester, benzoic acid phenyl ester, benzonitrile, benzophenone, benzothiazole, benzyl alcohol, benzyl alcohol, benzylbutylphthalate, bibenzyl, bromochlorobenzene, bromochlorophenol, 2-bromo-4-chlorophenol, bromodichlorophenol, 4-bromo-2,5-dichlorophenol, butanoic acid ethyl ester, 2-butoxyethanol, butyl acetate, C10H20 HC, C10H22 HC (1), C10H22 HC (2), C11H15O2N aromatic, C12H26 HC, C12H26O alcohol, C13H28 HC, C15 acid phthalic ester, C4 alkylbenzene, C5 alkylbenzene, C6H10O2 aliphatic carbonyl, C6H12O, C8H14O cyclohexanone, derivative, C8H5BrCl3 aromatic, MW, 284, C8H5O2N, C9H18O3 aliphatic, C9H8O aromatic, caffeine,

(TOC) in flue gas from an incineration facility of MSW. The identifications exceeded ~50 ng/m^3, 500x higher than the dioxin emission limit set in the Waste Incineration Directive. About 3% of the TOC consisted of halogenated compounds, almost all of which were volatile compounds, while all of the identified semi- and nonvolatile halogenated compounds were aromatic compounds. Besides, 7% of the TOC was aromatic hydrocarbons and 3% of the TOC was phenols [100]. Highly carcinogenic compounds such as dibenzopyrene isomers have been identified and determined in Swedish incinerator emissions by other researchers [101] and it is likely that due to the very heterogeneous nature of the waste emissions will constantly vary with consequences for the speciation of ultrafine particulate emissions.

Similarly Leach [102] found a wide range of VOCs in ground level monitoring around the Marchwood incinerator pre and post shutdowns in November 1996. Although that incinerator has since been replaced the results are indicative of the range of post combustion VOCs that are likely to be found in more modern facilities.

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chlorobenzene, chlorobenzoic acid, 4-chlorobenzoic acid, chloroform, 2-chloro-6-methylphenol, 4-(chloromethyl)toluene, 2-chlorophenol, 4-chlorophenol, cholesterol., cyclohexane, cyclopentasiloxanedecamet, hyl, cyclotetrasiloxaneoctamethy, l, decane, decanecarboxylic acid, dibenzothiophene, dibutylphthalate, 1,2-dichlorobenzene, 1,3-dichlorobenzene, 1,4-dichlorobenzene, 2,4-dichloro-6-cresol, dichloromethane, 2,6-dichloro-4-nitrophenol, 2,4-dichlorophenol, dichloromethylphenol, 1,3-diethylbenzene, diisooctylphthalate, 2,2'-dimethylbiphenyl, 2,3'-dimethylbiphenyl, 2,4'-dimethylbiphenyl, 3,3'-dimethylbiphenyl, 3,4'-dimethylbiphenyl, 1,2-dimethylcyclohexane, 1,2-dimethylcyclopentane, 1,3-dimethylcyclopentane, dimethyldioxane, dimethyloctane, 2,2-dimethyl-3-pentanol, dimethylphthalate, 2,6-di-t-butyl-*p*benzoquinone, 2,4-di-t-butylphenol, docosane, dodecane, dodecanecarboxylic acid, eicosane, ethanol-1-(2-butoxyethoxy), ethyl acetate, 4-ethylacetophenone, ethyl benzaldehyde, ethylbenzene, ethylbenzoic acid, 2-ethylbiphenyl, ethylcyclohexane, ethylcyclopentane, ethyldimethylbenzene, ethylhexanoic acid, 1-ethyl-2-methylbenzene, 1-ethyl-4-methylbenzene, ethylmethylcyclohexane, 2-ethylnaphthalene-1,2,3,4-, tetrahydro, 1-ethyl-3,5-xylene, 2-ethyl-1,4-xylene, fluorene, fluorenone, fluoroanthene, formic acid, 2-furanecarboxaldehyde, heneicosane, heptadecane, heptadecanecarboxylic acid, heptane, 20, heptanecarboxylic acid, 2-heptanone, hexachlorobenzene, hexachlorobiphenyl, hexadecane, hexadecane amide, hexadecanoic acid, hexadecanoic acid, hexadecyl ester, 9-hexadecene carboxylic, acid, hexanecarboxylic acid, 2-hexanone, hydroxybenzonitrile, hydroxychloroacetophenone, 2-hydroxy-3,5-, dichlorobenzaldehyde, hydroxymethoxybenzaldehy, de, 2-(hydroxymethyl) benzoic, acid, iodomethane, 1(3H)-isobenzofuranone-5-, methyl, isopropylbenzene, methyl acetophenone, 2-methylbenzaldehyde, 4-methylbenzaldehyde, methylbenzoic acid, 4-methylbenzyl alcohol, 2-methylbiphenyl, methylcyclohexane, methyldecane, 3-methyleneheptane, 5-methyl-2-furane, carboxaldehyde, methylhexadecanoic acid, 2-methylhexane, 3-methylhexane, methyl hexanol, 2-methylisopropylbenzene, 2-methyloctane, 2-methylpentane, methylphenanthrene, nonedecane, 4-methylphenol, 1-methyl-2-, phenylmethylbenzene, 2-methyl-2-propanol, 1-methyl-(1-, propenyl)benzene, 2-methylpropyl acetate, 1-methyl-2-propylbenzene, 1-methyl-3-propylbenzene, methylpropylcyclohexane, 12-, methyltetradecanecarboxyli, c acid, naphthalene, N-bearing aromatic, MW, 405, nitrogen compd, MW 269, 2-nitrostyrene, nonane, octadecadienal, octadecadienecarboxylic, acid, octadecane, octadecanecarboxylic acid, octane, octanoic acid, paraldehyde, pentachlorobenzene, pentachlorobiphenyl, pentachlorobiphenyl, pentachlorophenol, pentadecacarboxylic acid, pentane, pentanecarboxylic acid, phenanthrene, phenol, phthalic ester, phthalic ester, propylbenzene, propylcyclohexane, pyrene, Si organic compd, sulphonic acid m.w. 192, sulphonic acid m.w. 224, 2-t-butyl-4-methoxyphenol, tetrachlorobenzene, 1,2,3,5-tetrachlorobenzene, tetrachlorobenzofuran, tetrachloroethylene, 2,3,4,6-tetrachlorophenol, tetradecanecarboxylic acid, tetradecanoic acid isopropyl, ester, toluene, 1,2,3-trichlorobenzene, 1,2,4-trichlorobenzene, 1,2,4-trimethylbenzene, 1,2,5-trichlorobenzene, trichloroethene, trichlorofluoromethane, 3,4,6-trichloro-1-methylphenol, 2,3,4-trichlorophenol, 2,3,5-trichlorophenol, 2,4,6-trichlorophenol, 3,4,5-trichlorophenol, tridecanoic acid, 1,3,5-trimethylbenzene, trimethylcyclohexane, undecane, xylene

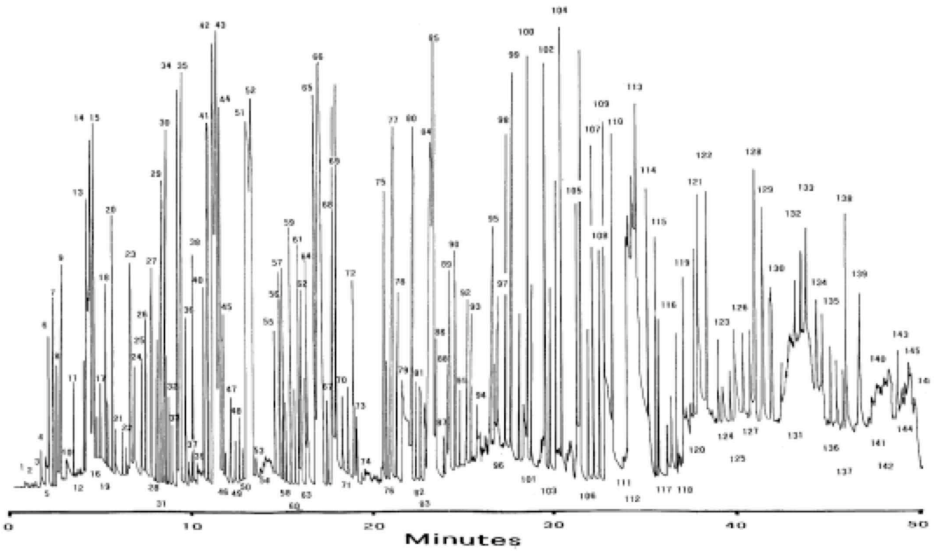

Fig. 4. Representative cGC-FID chromatogram of VOC identified at Sample Station 4, located 100 m south of Marchwood municipal incinerator (September 1996). Peak identifications are given in Table 2.

The toxicity of chemically-coated particles can be enhanced over expectations for single chemicals, because of synergies (coalitive effect, cosynergism and potentiation).

4.8 Dioxins and PCBs on Small Particles:

Fängmark et al. [13] concluded from analyzing incinerator flyash that chlorinated organics tend to be concentrated on the smaller particles. A similar result by Ruokojärvi et al. [9] found the < 1.6-μm fraction was disproportionately loaded. The distribution of PCDD/F with particle size in atmospheric dust collected at four Japanese sites was examined by Kurokawa et al. [11]. The maximum size collected was 30 μm in aerodynamic diameter, and the smallest 0.1 μm. Particles less than 1.1 μm contributed 50% of the total PCDD/F, with an almost equivalent I-TEQ proportion. The distribution of homologues changed with size, with the fraction of less chlorinated congeners in the homologue groups increasing with increasing particle size.

Chang [5] sampled air around a 1995 incinerator in Taiwan that had been fitted with activated carbon filtration to reduce the dioxin emissions to the EU standard of 0.1 ng/m3 and still found PCDD/F concentrations downwind of the MWI to be the highest and upwind to be the lowest among all sampling sites, concluding the MWI is noticeably contributing to dioxin levels in the ambient atmosphere.

Similarly Chao [103] sampled sites 1.1 and 2.1 km downwind from a municipal incinerator in central Taiwan and showed that PCDD/Fs were associated with the full size range of atmospheric particles.

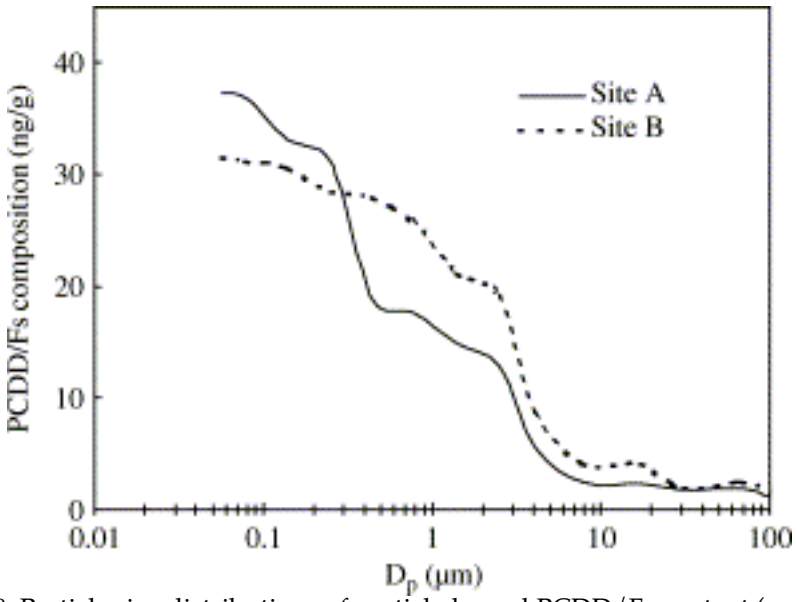

Fig8: Particle size distributions of particle-bound PCDD/Fs content (ng g^{-1})

More than 80% of the PCDD/Fs and toxic equivalents (TEQs) were found to be associated with fine particles of aerodynamic diameter 2.0 μm. Generally a smaller particle had a higher PCDD/Fs content and the dioxin concentration can be seen to increase to the very finest particles. The particle size distributions of PCDD/Fs and TEQs were shifted to larger particles with increasing time and distance.

Professor Sakai [104] analysed the mass balance of total and dioxin-like (co-planar) PCBs across a municipal waste incinerator and found that whereas the input of Co-PCBs into the MSW incineration facilities was 0.13–0.29 μg-TEQ per ton waste, the total output of Co-PCBs (the sum of Co-PCBs released from emission gas, fly ash, and bottom ash) was 4.9μg-TEQ per ton waste. Whilst over 90% of the total PCBs were destroyed in the incineration process the toxicity of the output was found to be higher than that of the input. This emphasizes the importance of assessing PCB emissions as well as those of dioxins and as the indications are that PCB synthesis was taking place post-combustion it is likely that the contaminants on the smallest particles would include PCBs as well as dioxins.

4.9 Halogenated Dioxins

It should be noted that whilst currently 17 dioxins and furans are measured there are actually many more – and this has been recognised for more than 20 years. In 1987, for example, Schechter [105] wrote:

"We are faced with the problem that animal data, upon which risk assessment and standard setting is based, is very incomplete. Also, as noted by Buser, in addition to the 200 plus chlorinated dibenzodioxins and dibenzofurans which may exist, there may be 5,000 chlorinated, brominated or bromochlorodioxins and dibenzofurans which may exist from incineration sources and which may be of potential concern".

Since 1987 it has been demonstrated beyond doubt that brominated and mixed halogenated dioxins are produced by incinerators and that their toxicity is similar to - and sometime greater – than the chlorinated dioxins. In spite of this these dioxins are still not incorporated into incinerator risk assessments.

4.10 Combined Particle Size Distribution and Speciation:

Unfortunately few researchers have combined data on particle size distribution and speciation. Greenberg [106] tested emissions from the Nicosia incinerator and found 70-90% of the Zn, Cu, Cd and Pb to reside in the smallest particles (< 0.8*μm)*. However, that facility had only an electrostatic precipitator at the time, so the results are not directly transferrable to a more modern plant with a bag filter. Nonetheless it is clear that the majority of the metals exposure should be anticipated to arise from the ultrafine fraction of the emissions.

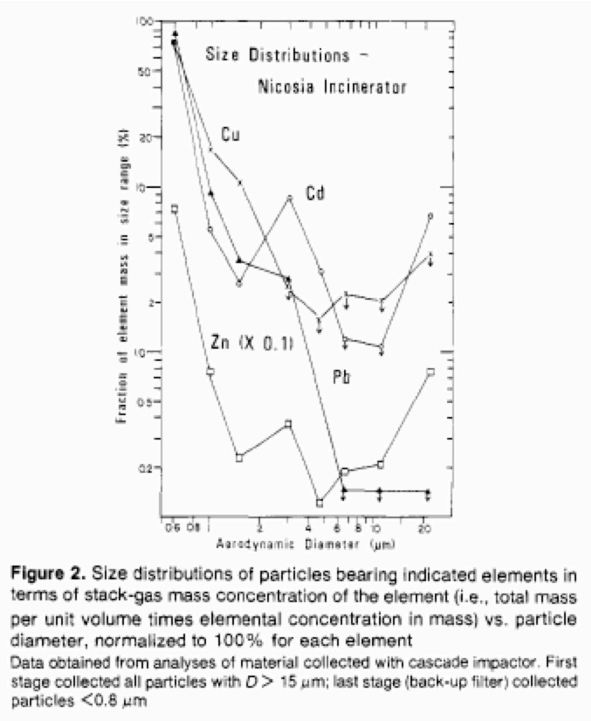

Figure 2. Size distributions of particles bearing indicated elements in terms of stack-gas mass concentration of the element (i.e., total mass per unit volume times elemental concentration in mass) vs. particle diameter, normalized to 100% for each element
Data obtained from analyses of material collected with cascade impactor. First stage collected all particles with $D > 15$ μm; last stage (back-up filter) collected particles <0.8 μm

4.11 Future Risks – Disposal of Nanotechnology wastes:

Nanomaterials are already reportedly used in over 800 products and the sales of which were valued at $147 billion in 2007 and are expected to soar over the coming years with a predicted value of $3.1 trillion by 2015 [107]. Inevitably the quantities of waste containing nanoparticles will increase rapidly but little thought has yet been given to the consequences of this. When products are incinerated, the thermal properties of nanoparticles determine their fate. There is evidence that at least some nanoparticles will pass through incinerators and be dispersed into the environment..

Franco [108] writes: " *whereas the onset temperature reaction for C60 is very low (315 °C), carbon nanotubes display very low reactivity under combustion conditions (onset temperature = 820 °C) and hence may not breakdown in an incinerator [109]. In theory, this means that they could end up in the gaseous effluent and released into the atmosphere*".

This is a significant concern given the inability to filter ultra-fine particles even with modern bag filters [78-81]. Any nanoparticles released from an incinerator increase the risk described above and incineration may increasingly play a role as a very effective delivery mechanism directly into the alveoli for a wide range of products of waste nanotechnology products.

4.12 Risk Assessment:

The risk assessment in relation to particulates that has been undertaken by the Indaver is rather simplistic. The principle assumption, and the basis for the conclusion, it that if air quality standards are not exceeded by the combination of existing ambient concentrations and the marginal increase from the incinerator then no harm is assumed to occur.

This approach is, of course, fundamentally flawed for those emissions, like particulates for which no safe level can be demonstrated.

Kunzli [110] wrote "*In many countries, policy makers currently face the problem that air quality criteria regulations are intended to "protect health", including the health of the most vulnerable people; to date, research has failed to obtain any evidence for a no-effect threshold. Thus, similar to carcinogens, the natural "threshold" might be zero exposure. Therefore, non-zero target values of clean air acts, inherently assume that some health impact of air pollution may be accepted. Impact assessors must choose a level below which they explicitly want to ignore the impact on air pollution*".

Chao [103] comments that even though a large number of atmospheric dispersion models exist and are readily available for use, the risk assessor is generally faced with little or no data on the atmospheric particle size distribution of PCDD/Fs. Lohman and Seigneur [111] conclude that "*it is essential to obtain accurate characterizations of the particle size distribution of particulate PCDD/F because the dry deposition flux is very sensitive to the particle size distribution*". Without such data accurate risk assessment is not possible and yet there is no evidence that it has been collected or used in relation to this application.

4.13 Conclusions on UFPs from Incinerators:

Not only do a high proportion of the UFPs escape the filters, but they are chemically reactive and carry a wide range of products of incomplete combustion and adsorbed metals with them. The subsequent direct uptake of these respirable particles and the ready transfer from the lungs into the blood stream may be part of the reason that traditional toxicology is at a loss to explain the level of impacts for such apparently low exposures.

Aerosols in the ultra-fine size range have much higher mobility in the air and can more effectively deposit in the respiratory system.

Ultrafine particles have been found to be chemically highly reactive, even when originating from a relatively unreactive bulk material [25]. The massive surface area associated with a small mass of nanometre-sized particles can act as a catalytic surface for the secondary formation of organic compounds such as the *de novo* synthesis of dioxins.

The relative toxicity of ultrafine particles arising from different processes remains un-researched. The levels of heavy and transition metal inputs in municipal solid waste are very much higher than with conventional fuels. Such increases must inevitably be associated with an increase in toxicity and consequently the likelihood of adverse health effects among the local receptors.

In my opinion, there is also a need to determine the relative toxicity of the particulate aerosols in the gases emitted by different waste disposal routes, to facilitate rational decisions as to the best disposal method, particularly with respect to public health. This should be addressed urgently but, in the meantime with the significant prospects of serious harm to health, high weight must be given to the precautionary principle.

5 The Precautionary Principle

The Twenty-fourth Report of the Royal Commission on Environmental Pollution, *Chemicals in Products: Safeguarding the Environment and Human Health*, [112]pointed out that the historical record is replete with unexpected toxicological impacts arising following the use of anthropogenic chemicals.

The Royal Commission emphasized that whilst we have learnt a great deal from some of the early episodes we may still be caught unawares, as witnessed with the emergence of a large number of different endocrine disrupting chemicals during the 1980s and 1990s.

"*It was not foreseen that low concentrations of chemicals used as antifouling agents (tributyltin), surfactants (nonyl phenol), flame retardants (polybrominated diphenylethers) and plasticisers (phthalates) would bind to hormone receptors or disrupt hormone metabolism in birds, reptiles, fish and invertebrates and influence sperm counts and the development of testicular malignancy in humans* [113, 114]."

These examples refer to chemicals whose reactivity it was felt was reasonably well understood. This is not the case with the UFPs with their wide range of chemical loading that are released in large quantities from modern incinerators. Apart from the fact that we know they are likely to be harmful at concentrations well below current air quality standards little is known of about the likely extent of environmental effects or their likelihood of causing unintended harm. Furthermore as nanotechnology expands there are even greater future risks from relying on technologies which, in at least some cases, are more likely to disperse them into the atmosphere than to destroy them as described above.

Having reviewed the science and the hazards of ultrafine particles I agree with Kunzli [110] who wrote "*In the light of all the uncertainties and limitations, researchers should not lose sight of the general patterns and perspectives. Given the current level of evidence of the association between air pollution and health, the precautionary principle may provide excellent guide to rigorously implement clean air strategies*".

The precautionary principle is part of the framework for sustainable development and I consider that the principle should be regarded more seriously when considering incineration processes, where there is significant scientific uncertainty and serious risks of harm.

The precautionary principle in its modern formulation is a means to safeguard public health. The European Commission advised the inclusion of public health in 2000 (European Commission Communication on Precautionary Principle, 2 February 2000), saying that the precautionary principle should be applied where "*there are reasonable grounds for concern that potential hazards may affect the environment or human, animal or plant health, and when at the same time the lack of scientific information precludes a detailed scientific evaluation*".

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.

I would therefore recommend that this application should not be approved in the light of the likely risks to public health and the Environment detailed in this evidence.

6 EndNotes:

1. Maynard, R. and C. Howard, Eds, *Particulate Matter: Properties and Effects upon Health*. 1999, Oxford: BIOS Scientific Publishers.
2. European Commission, *DIRECTIVE 2000/76/EC OF THE EUROPEAN PARLIAMENT AND OF THE COUNCIL of 4 December 2000 on the incineration of waste.* Official Journal of the European Communities, 2000. **L 332**.
3. Health Research Board, et al., *Health and Environmental Effects of Landfilling and Incineration of Waste – A Literature Review*. 2003, Dublin: Health Research Board. viii, 284 p.
4. 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.
5. Nemery, B., P.H.M. Hoet, and A. Nemmar, *The Meuse Valley fog of 1930: an air pollution disaster.* The Lancet, 2001. **357**(9257): p. 704-708.
6. Haldane, J.S., *Atmospheric Pollution and Fogs.* Br Med J, 1931. **1**(3660): p. 366-367.
7. Bell, M.L. and D.L. Davis, *Reassessment of the lethal London fog of 1952: novel indicators of acute and chronic consequences of acute exposure to air pollution.* Environ Health Perspect, 2001. **109 Suppl 3**: p. 389-94.
8. Polichetti, G., et al., *Effects of particulate matter (PM10, PM2.5 and PM1) on the cardiovascular system.* Toxicology. **In Press, Corrected Proof**.
9. 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.
10. Nawrot, T.S., et al., *Stronger associations between daily mortality and fine particulate air pollution in summer than in winter: evidence from a heavily polluted region in western Europe.* J Epidemiol Community Health, 2007. **61**(2): p. 146-149.
11. Donaldson, K., X.Y. Li, and W. MacNee, *Ultrafine (nanometre) particle mediated lung injury.* Journal of Aerosol Science, 1998. **29**(5-6): p. 553-560.
12. Penttinen, P., et al., *Number concentration and size of particles in urban air: effects on spirometric lung function in adult asthmatic subjects.* Environ Health Perspect, 2001. **109**(4): p. 319-23.
13. Wahlin, P., et al., *Pronounced decrease of ambient particle number emissions from diesel traffic in Denmark after reduction of the sulphur content in diesel fuel.* Atmospheric Environment, 2001. **35**(21): p. 3549-3552.
14. Donaldson, K., et al., *Combustion-derived nanoparticles: A review of their toxicology following inhalation exposure.* Particle and Fibre Toxicology, 2005. **2**(1): p. 10.
15. Li, N., et al., *Ultrafine particulate pollutants induce oxidative stress and mitochondrial damage.* Environ Health Perspect, 2003. **111**(4): p. 455-60.
16. Harrison, R., et al., *Measurement of number, mass and size distribution of particles in the atmosphere.* Philos Trans R Soc A, 2000. **358**: p. 2567–2579.
17. Aboh, I.J.K., et al., *EDXRF characterisation of elemental contents in PM2.5 in a medium-sized Swedish city dominated by a modern waste incineration plant.* X-Ray Spectrometry, 2007. **36**(2): p. 104-110.
18. Bencko, V., *Use of human hair as a biomarker in the assessment of exposure to pollutants in occupational and environmental settings.* Toxicology, 1995. **101**(1-2): p. 29-39.
19. National Research Council, N.A.o.S., *"Airborne Particles" quoted by RACHEL'S ENVIRONMENT & HEALTH WEEKLY #440 4th May1995 The Holy Grail of Scientific Certainty*. 1979, Baltimore, Md: University Park Press.
20. Bai, N., et al., *The pharmacology of particulate matter air pollution-induced cardiovascular dysfunction.* Pharmacology & Therapeutics, 2007. **113**(1): p. 16-29.
21. Brook, R., et al., *Air pollution and cardiovascular disease: a statement for healthcare professionals from the Expert Panel on Population and Prevention Science of the American Heart Association.* Circulation, 2004. **109**: p. 2655 - 2671.

22. Ballester, F., et al., *Reducing ambient levels of fine particulates could substantially improve health: a mortality impact assessment for 26 European cities.* J Epidemiol Community Health, 2008. **62**(2): p. 98-105.
23. Wichmann, H.-E., et al., *Daily Mortality and Fine and Ultrafine Particles in Erfut, Germany. Part I: Role of Particle Number and Particle Mass. Research Report 98*. 2000, Health Effects Institute: Cambridge MA.
24. Sawyer, R.F., *Science based policy for addressing energy and environmental problems.* Proceedings of the Combustion Institute, 2009. **32**(1): p. 45-56.
25. Jefferson, D.A. and E.E.M. Tilley, *The structural and physical chemistry of nanoparticles*, in *Particulate matter: properties and effects upon health*, R.L. Maynard and C.V. Howard, Editors. 1999, BIOS Scientific Publishers Ltd: Oxford. p. 63-84.
26. Donaldson, J., V. Stone, and W. MacNee, *The toxicology of ultrafine particles*, in *Particulate matter: properties and effects upon health*, R.L. Maynard and C.V. Howard, Editors. 1999, BIOS Scientific Publishers Ltd: Oxford. p. 115-129.
27. Oberdorster, G., E. Oberdorster, and J. Oberdorster, *Nanotoxicology: an emerging discipline evolving from studies of ultrafine particles.* Environ Health Perspect, 2005. **113**: p. 823 - 839.
28. Seaton, A., et al., *Particulate air pollution and acute health effects.* The Lancet, 1995. **345**(8943): p. 176-178.
29. Kunzli, N., et al., *Public-health impact of outdoor and traffic-related air pollution: a European assessment.* The Lancet, 2000. **356**(9232): p. 795-801.
30. 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.
31. Boekelheide, K., *Mixed Messages.* Toxicol. Sci., 2007. **99**(1): p. 1-2.
32. Ochs, M., et al., *The Number of Alveoli in the Human Lung.* Am. J. Respir. Crit. Care Med., 2004. **169**(1): p. 120-124.
33. Yang, W., J.I. Peters, and R.O. Williams Iii, *Inhaled nanoparticles--A current review.* International Journal of Pharmaceutics, 2008. **356**(1-2): p. 239-247.
34. Salvi, S., *Health effects of ambient air pollution in children.* Paediatric Respiratory Reviews, 2007. **8**(4): p. 275-280.
35. 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.
36. Gumbleton, M., *Caveolae as potential macromolecule trafficking compartments within alveolar epithelium.* Advanced Drug Delivery Reviews, 2001. **49**(3): p. 281-300.
37. Kim, C.S. and P.A. Jaques, *Respiratory dose of inhaled ultrafine particles in healthy adults.* Philosophical Transactions of the Royal Society of London. Series A: Mathematical, Physical and Engineering Sciences, 2000. **358**(1775): p. 2693-2705.
38. Lundborg, M., et al., *Human Alveolar Macrophage Phagocytic Function is Impaired by Aggregates of Ultrafine Carbon Particles.* Environmental Research, 2001. **86**(3): p. 244-253.
39. Muller, A.K., et al., *DNA damage in lung after oral exposure to diesel exhaust particles in Big BlueÆ rats.* Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis, 2004. **550**(1-2): p. 123-132.
40. Bermudez, E., et al., *Pulmonary Responses of Mice, Rats, and Hamsters to Subchronic Inhalation of Ultrafine Titanium Dioxide Particles.* Toxicol. Sci., 2004. **77**(2): p. 347-357.
41. Borm, P.J. and W. Kreyling, *Toxicological hazards of inhaled nanoparticles--potential implications for drug delivery.* J Nanosci Nanotechnol, 2004. **4**(5): p. 521-31.
42. Stearns, R.C., J.D. Paulauskis, and J.J. Godleski, *Endocytosis of Ultrafine Particles by A549 Cells.* Am. J. Respir. Cell Mol. Biol., 2001. **24**(2): p. 108-115.
43. Oberdorster, G., et al., *Extrapulmonary translocation of ultrafine carbon particles following whole-body inhalation exposure of rats.* J Toxicol Environ Health A, 2002. **65**(20): p. 1531-43.

44. Nemmar, A., et al., *Passage of Inhaled Particles Into the Blood Circulation in Humans.* Circulation, 2002. **105**(4): p. 411-414.
45. Kreyling, W.G., et al., *Translocation of ultrafine insoluble iridium particles from lung epithelium to extrapulmonary organs is size dependent but very low.* J Toxicol Environ Health A, 2002. **65**(20): p. 1513-30.
46. Stone, V., et al., *Increased calcium influx in a monocytic cell line on exposure to ultrafine carbon black.* Eur Respir J, 2000. **15**: p. 297 - 303.
47. Donaldson, K., et al., *Ultrafine particles.* Occup Environ Med, 2001. **58**(3): p. 211-216.
48. Geiser, M., et al., *Ultrafine particles cross cellular membranes by nonphagocytic mechanisms in lungs and in cultured cells.* Environ Health Perspect, 2005. **113**(11): p. 1555-60.
49. Stone, P.H. and J.J. Godleski, *First steps toward understanding the pathophysiologic link between air pollution and cardiac mortality.* American Heart Journal, 1999. **138**(5): p. 804-807.
50. Dockery, D.W., et al., *An association between air pollution and mortality in six U.S. cities.* N Engl J Med, 1993. **329**(24): p. 1753-9.
51. Donaldson, K., et al., *Ultrafine particles: mechanisms of lung injury.* Philosophical Transactions of the Royal Society of London. Series A: Mathematical, Physical and Engineering Sciences, 2000. **358**(1775): p. 2741-2749.
52. Oberdorster, G., *Toxicology of ultrafine particles: In vivo studies.* Philosophical Transactions of the Royal Society A: Mathematical, Physical and Engineering Sciences, 2000. **358**(1775): p. 2719-2740.
53. Wichmann, H.E. and A. Peters, *Epidemiological evidence of the effects of ultrafine particle exposure.* Philosophical Transactions of the Royal Society A: Mathematical, Physical and Engineering Sciences, 2000. **358**(1775): p. 2751-2769.
54. Howard, C.V., *Nano-particles and Toxicity - Annex (P15-20) in No Small Matter II: The Case for a Global Moratorium Size Matters! April 2003*, in *Occasional Paper Series 7:1* 2003, ETC Group
55. Peters, A., et al., *Translocation and potential neurological effects of fine and ultrafine particles a critical update.* Part Fibre Toxicol, 2006. **3**: p. 13.
56. Kunzli, N., et al., *Ambient air pollution and atherosclerosis in Los Angeles.* Environ Health Perspect, 2005. **113**(2): p. 201-6.
57. Miller, K.A., et al., *Long-Term Exposure to Air Pollution and Incidence of Cardiovascular Events in Women.* N Engl J Med, 2007. **356**(5): p. 447-458.
58. Pope, C.A., III, et al., *Cardiovascular Mortality and Long-Term Exposure to Particulate Air Pollution: Epidemiological Evidence of General Pathophysiological Pathways of Disease.* Circulation, 2004. **109**(1): p. 71-77.
59. Dockery, D.W. and P.H. Stone, *Cardiovascular Risks from Fine Particulate Air Pollution.* N Engl J Med, 2007. **356**(5): p. 511-513.
60. 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.
61. World Health Organisation Europe. *Children's Environment and Health Action Plan for Europe*. in *Fourth Ministerial Conference on Environment and Health*. 2005. Budapest, Hungary.
62. Schwartz, J., *Air pollution and children's health.* Pediatrics, 2004. **113**(4 Suppl): p. 1037-43.
63. 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.
64. Hertz-Picciotto, I., et al., *Early childhood lower respiratory illness and air pollution.* Environ Health Perspect, 2007. **115**(10): p. 1510-8.
65. Kunzli, N., et al., *Breathless in Los Angeles: The Exhausting Search for Clean Air.* Am J Public Health, 2003. **93**(9): p. 1494-1499.

66. Heindel, J.J., *Role of exposure to environmental chemicals in the developmental basis of disease and dysfunction.* Reproductive Toxicology, 2007. **23**(3): p. 257-259.
67. Yeatts, K., et al., *A brief targeted review of susceptibility factors, environmental exposures, asthma incidence, and recommendations for future asthma incidence research.* Environ Health Perspect, 2006. **114**(4): p. 634-40.
68. Henderson, J., et al., *Household chemicals, persistent wheezing and lung function: effect modification by atopy?* Eur Respir J, 2008. **31**(3): p. 547-554.
69. Jedrychowski, W., et al., *Early wheezing phenotypes and severity of respiratory illness in very early childhood: Study on intrauterine exposure to fine particle matter.* Environment International, 2009. **35**(6): p. 877-884.
70. Perera, F.P., et al., *Molecular evidence of an interaction between prenatal environmental exposures and birth outcomes in a multiethnic population.* Environ Health Perspect, 2004. **112**(5): p. 626-30.
71. Mohorovic, L., *First two months of pregnancy--critical time for preterm delivery and low birthweight caused by adverse effects of coal combustion toxics.* Early Human Development, 2004. **80**(2): p. 115-123.
72. Widory, D., *Nitrogen isotopes: Tracers of origin and processes affecting PM10 in the atmosphere of Paris.* Atmospheric Environment, 2007. **41**(11): p. 2382-2390.
73. Querol, X., et al., *Speciation and origin of PM10 and PM2.5 in selected European cities.* Atmospheric Environment, 2004. **38**(38): p. 6547-6555.
74. Almeida, S.M., et al., *Approaching PM2.5 and PM2.5†-†10 source apportionment by mass balance analysis, principal component analysis and particle size distribution.* Science of The Total Environment, 2006. **368**(2-3): p. 663-674.
75. Moffet, R., et al., *Characterization of Aerosols Containing Zn, Pb, and Cl from an Industrial Region of Mexico City.* Environmental Science & Technology, 2008. **42**(19): p. 7091–7097.
76. Ying, Q. and M.J. Kleeman, *Source contributions to the regional distribution of secondary particulate matter in California.* Atmospheric Environment, 2006. **40**(4): p. 736-752.
77. Hinds, W.C., *Filtration*, in *Aerosol Technology Properties, Behavior, and Measurement of Airborne Particles*. 1999, Wiley: New York. p. 182-205.
78. Lighty, J.S., J.M. Veranth, and A.F. Sarofim, *Combustion aerosols: factors governing their size and composition and implications to human health.* J Air Waste Manag Assoc, 2000. **50**(9): p. 1565-618; discussion 1619-22.
79. Buonanno, G., G. Ficco, and L. Stabile, *Size distribution and number concentration of particles at the stack of a municipal waste incinerator.* Waste Management, 2009. **29**(2): p. 749-755.
80. Maguhn, J., et al., *On-line analysis of the size distribution of fine and ultrafine aerosol particles in flue and stack gas of a municipal waste incineration plant: effects of dynamic process control measures and emission reduction devices.* Environ Sci Technol, 2003. **37**(20): p. 4761-70.
81. Yuan, C.-S., et al., *Partition and size distribution of heavy metals in the flue gas from municipal solid waste incinerators in Taiwan.* Chemosphere, 2005. **59**(1): p. 135-145.
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. Shi, J.P., et al., *Sources and concentration of nanoparticles (<10 nm diameter) in the urban atmosphere.* Atmospheric Environment, 2001. **35**(7): p. 1193-1202.
85. Bennett, R.L. and K.T. Knapp, *Characterization of particulate emissions from municipal wastewater sludge incinerators.* Environ Sci Technol, 1982. **16**(12): p. 831-836.
86. Markowski, G.R., et al., *A submicron aerosol mode in flue gas from a pulverized coal utility boiler.* Environ Sci Technol, 1980. **14**(11): p. 1400-1402.

87. Friedlander, S.K., W. Koch, and H.H. Main, *Scavenging of a coagulating fine aerosol by a coarse particle mode.* Journal of Aerosol Science, 1991. **22**(1): p. 1-8.
88. Ruokojarvi, P., et al., *Effect of urea on fly ash PCDD/F concentrations in different particle sizes.* Chemosphere, 2001. **43**(4-7): p. 757-762.
89. United States Environmenal Protection Agency, *Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds Part I: Estimating Exposure to Dioxin-Like Compounds Volume 4: Site-Specific Assessment Procedures p 3-73 Draft Final EPA/600/P-00/001Ad March 2000*. 2000.
90. Rose, W.I., C.M. Riley, and S. Dartevelle, *Sizes and Shapes of 10-Ma Distal Fall Pyroclasts in the Ogallala Group, Nebraska.* The Journal of Geology, 2003. **111**(1): p. 115-124.
91. Morawska, L. and J. Zhang, *Combustion sources of particles. 1. Health relevance and source signatures.* Chemosphere, 2002. **49**(9): p. 1045-1058.
92. Wey, M.Y., J.L. Su, and J.C. Chen, *Influence of operating conditions on the formation of heavy metal compounds during incineration.* Journal of the Air & Waste Management Association, 1999. **49**(4): p. 444-453.
93. Harrison, R.M., et al., *Comparative receptor modelling study of airborne particulate pollutants in Birmingham (United Kingdom), Coimbra (Portugal) and Lahore (Pakistan).* Atmospheric Environment, 1997. **31**(20): p. 3309-3321.
94. Mishra, V.K., et al., *Wintertime sources and distribution of airborne lead in Korea.* Atmospheric Environment, 2004. **38**(17): p. 2653-2664.
95. Doucet, F.J. and J. Carignan, *Atmospheric Pb isotopic composition and trace metal concentration as revealed by epiphytic lichens: : an investigation related to two altitudinal sections in Eastern France.* Atmospheric Environment, 2001. **35**(21): p. 3681-3690.
96. Pancras, J.P., et al., *Identification of sources and estimation of emission profiles from highly time-resolved pollutant measurements in Tampa, FL.* Atmospheric Environment, 2006. **40**(Supplement 2): p. 467-481.
97. Younes, M., *Specific issues in health risk assessment of endocrine disrupting chemicals and international activities.* Chemosphere, 1999. **39**(8): p. 1253-1257.
98. Kurokawa, Y., et al., *Distribution of polychlorinated dibenzo-p-dioxins and dibenzofurans in various sizes of airborne particles.* Chemosphere, 1998. **37**(9-12): p. 2161-2171.
99. Jay, K. and L. Stieglitz, *Identification and quantification of volatile organic components in emissions of waste incineration plants.* Chemosphere, 1995. **30**(7): p. 1249-1260.
100. Kato, M., K. Urano, and T. Tasaki, *Development of Semi- and Nonvolatile Organic Halogen as a New Hazardous Index of Flue Gas.* Environ Sci Technol, 2000. **34**(19): p. 4071-4075.
101. Bergvall, C. and R. Westerholm, *Identification and Determination of Highly Carcinogenic Dibenzopyrene Isomers in Air Particulate Samples from a Street Canyon, a Rooftop, and a Subway Station in Stockholm.* Environ Sci Technol, 2007. **41**(3): p. 731-737.
102. Leach, J., A. Blanch, and A.C. Bianchi, *Volatile organic compounds in an urban airborne environment adjacent to a municipal incinerator, waste collection centre and sewage treatment plant.* Atmospheric Environment, 1999. **33**(26): p. 4309-4325.
103. Chao, M.R., et al., *Size distribution of particle-bound polychlorinated dibenzo-p-dioxins and dibenzofurans in the ambient air of a municipal incinerator.* Atmospheric Environment, 2003. **37**(35): p. 4945-4954.
104. Sakai, S.-i., et al., *Substance flow analysis of coplanar PCBs released from waste incineration processes.* Journal of Material Cycles and Waste Management, 1999. **1**(1): p. 62-74.
105. Schecter, A., *Risk assessment and standard setting: Rapporteur's summary and discussion.* Chemosphere, 1987. **16**(8-9): p. 2205-2210.
106. Greenberg, R.R., et al., *Composition of particles emitted from the Nicosia municipal incinerator.* Environ Sci Technol, 1978. **12**(12): p. 1329-1332.
107. Singh, N., et al., *NanoGenotoxicology: The DNA damaging potential of engineered nanomaterials.* Biomaterials, 2009. **30**(23-24): p. 3891-3914.

108. Franco, A., et al., *Limits and prospects of the ‘‘incremental approach’’ and the European legislation on the management of risks related to nanomaterials.* Regulatory Toxicology and Pharmacology, 2007. **48**: p. 171–183.
109. Cataldo, F., *A study on the thermal stability to 1000 C of various carbon allotropes and carbonaceous matter both under nitrogen and in air.* Fullerenes Nanotubes and Carbon Nanostructures, 2002. **10**(4): p. 293–311.
110. Kunzli, N., *The public health relevance of air pollution abatement.* Eur Respir J, 2002. **20**(1): p. 198-209.
111. Lohman, K. and C. Seigneur, *Atmospheric fate and transport of dioxins: local impacts.* Chemosphere, 2001. **45**(2): p. 161-171.
112. Royal Commission on Environmental Pollution (RCEP), *24th report on Chemicals in Products - Safeguarding the Environment and Human Health Cm 5827 June 2003* 2003.
113. Sharpe, R.M., *Male Reproductive Health Disorders and the Potential Role of Exposure to Environmental Chemicals*. 2009, Commissioned by CHEM Trust.
114. ENDS, *Chemical threat to men’s sexual health.* Environmental Data Services (ENDS), 2009. **412**: p. 6.