The Impact on Health of Emissions to Air from Municipal Waste Incinerators

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Summary

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

The Agency's role is to provide expert advice on public health matters to Government, stakeholders and the public. The regulation of municipal waste incinerators is the responsibility of the Environment Agency.

Introduction

1. The use of incineration for waste disposal in the UK is increasing. Applications for permits to build and operate incinerators give rise to local concerns about possible effects on health of emissions. Responsibility for the environmental permitting of municipal waste incinerators lies with the Environment Agency. The Health Protection Agency (HPA) has a statutory responsibility to advise Government and Local Authorities on possible health impacts of air pollutants.

2. The operators of modern waste incinerators are required to monitor emissions to ensure that they comply, as a minimum, with the limits in the EU Waste Incineration Directive (2000/76/EC), which sets strict emission limits for pollutants. This Directive has been implemented in England and Wales by the Environmental Permitting (EP) (England and Wales) Regulations 2007 (note

that from April 2008 these replaced the Pollution Prevention and Control (PPC) (England and Wales) Regulations 2000).

3. Under the EP Regulations, the operator is required to apply for an environmental permit. Consideration of this application will include such issues as health effects and organisations such as the local Primary Care Trust (PCT); the HPA and Food Standards Agency (FSA) are usually consulted. The permit itself will set out strict operating requirements which must be complied with, this will include monitoring. Should a breach of the permit occur, action may be taken by the regulator.

4. Applications to build and operate incinerators invariably include an assessment of likely emissions to air. Modern incinerators emit only small amounts of chemicals to air (see para 16) in comparison with older incinerators and, although no absolute assurance of a zero effect on public health can be provided, the additional burden on the health of the local population is likely to be very small. Studies published in the scientific literature showing health effects in populations living around incinerators have, in general, been conducted around older incinerators with less stringent emission standards and cannot be directly extrapolated with any reliability to modern incinerators (see paras 6 and 26)

5. The incineration process can result in three potential sources of exposure, (1) emissions to the atmosphere, (2) via solid ash residues, and (3) via cooling water. Provided that solid ash residues and cooling water are handled and disposed of appropriately, atmospheric emissions remain the only significant route of exposure to people. This paper is thus concerned only with the health effects of emissions to air.

6. The comparative impacts on health of different methods of waste disposal have been considered in detail in a report prepared for the Department of Environment, Food and Rural Affairs (Defra 2004). This work was undertaken by a group of consultants led by the independent consultants Enviros and included experts in the air pollution field. The report was reviewed by The Royal Society and its comments were incorporated by the authors of the report. This report is the most extensive available in the field and concludes that well managed, modern incinerators are likely to have only a very small effect on health. Since the evidence base has not changed significantly since 2004 it would be an inefficient use of resources to repeat the work undertaken by Enviros (see above) for Defra when applications to build and operate individual incinerators are being considered. The HPA's view is that the study undertaken for Defra by Enviros can be relied on although, like all scientific findings, it may be subject to revision if new data were to emerge.

7. Concerns about possible effects on health of emissions to air tend to focus on a few well known pollutants: particles, polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzo-*p*-furans (commonly referred to as "dioxins") and other carcinogens such as the polycyclic aromatic hydrocarbons (PAH). Much is known about the effects on health of these

compounds. Detailed reports prepared by expert advisory committees are available: these include reports by the Department of Health's Committee on the Medical Effects of Air Pollutants (COMEAP) on particulate matter (COMEAP, 1995, 1998, 2001a, 2009); by Defra's Expert Panel on Air Quality Standards (EPAQS) on benzene, 1,3-butadiene (reports 1 and 2), particles (reports 1 and 2), PAH compounds, and metals and metalloids[1] (Department of the Environment, 1994a,b, 1995; Department of the Environment Transport and the Regions, 1999, 2001; Department for the Environment, Food and Rural Affairs, 2002, 2009) and the Committee on the Toxicity of Chemicals in Food, Consumer Products and the Environment's statement on dioxins and dioxin-like polychlorinated biphenyls (Committee on Toxicity, 2001).

Particles

8. Questions are often asked about the possible effects on health of particles emitted by incinerators. The Committee on the Medical Effects of Air Pollutants (COMEAP) has published a series of statements and reports on the effects of air pollutants on health in the UK. It is accepted that exposure to current levels of common air pollutants damages health. The Air Quality Strategy for England, Scotland, Wales and Northern Ireland seeks to reduce concentrations of air pollutants. Where concentrations of air pollutants are raised, Air Quality Management Areas are defined and plans to reduce concentrations are developed by Local Authorities. Details of the Air Quality Strategy can be found on the Defra website: http://www.defra.gov.uk/environment/airquality/strategy/index.htm

9. Both long-term exposure and short-term increases in exposure to particles can damage health. This is widely accepted (World Health Organization, 2006). Long term exposure affects the risk of mortality, especially from cardiovascular disease and from lung cancer (COMEAP, 2009, COMEAP, 2006; Health Effects Institute, 2000). Short-term increases in concentrations cause cardio-respiratory effects including an increase in deaths from heart attacks and from respiratory disease, increased hospital admissions for treatment of these disorders and increases in related symptoms. No thresholds of effect can be identified for either the effects of long-term exposure or for the effects of short-term increases in concentrations. Thus, any increase in particle concentrations should be assumed to be associated with some effect on health. The critical step in assessment of effects on health is not simply making the correct assertion that some effect is possible but in estimating the size of that effect. This is discussed below.

10. Evidence of the effects of particles on health comes, in the main, from epidemiological studies. For the effects of long-term exposure attention has been focused on $PM_{2.5}$; for the effects of short-term increases in concentrations both $PM_{2.5}$ and PM_{10} have been extensively used as metrics of the ambient aerosol. PM_{10} is defined as the mass of particles of less than

[1] Arsenic, chromium, nickel and beryllium

(about) 10 microns in diameter per cubic metre of air. $PM_{2.5}$ is an analogous measure: in this case, the mass of particles of less than about 2.5 microns in diameter per cubic metre of air. The exact definitions are given in the recent Defra report on ambient particles (Defra, 2005). The exact mechanisms of effect of particles on health are incompletely understood but several plausible hypotheses are being pursued; the generation of free radicals in the respiratory system and more widely in the body, the induction of an inflammatory response in the lung, effects on clotting factors in the blood, effects on the rate of development of atherosclerotic plaques in coronary arteries and effects on the regulation of the heart beat are all being studied intensively. It is possible that metals found in association with particles play an important role. It is also possible that the ultrafine component of the ambient aerosol plays an important role. These, and other, possibilities are not yet proven.

11. The lack of a complete understanding of the mechanisms of effects of particles does not prevent prediction of the effects on health of increased concentrations of particles monitored as PM_{10} and/or $PM_{2.5}$. Meta-analytical techniques have been applied to the results of primary studies and summary coefficients linking PM_{10} and $PM_{2.5}$ with effects on health have been derived (COMEAP, 1998, 2009; World Health Organization, 2006). If these coefficients are applied to the small increases in concentrations of particles produced, locally, by incinerators, the estimated effects on health are likely to be small. This is because the coefficients themselves are small, the increase in concentration due to operation of the incinerator is likely to be small, and so is the size of the potentially exposed local population.

12. It is sometimes claimed that the “wrong particles” are considered when estimating the possible effects on health of emissions from incinerators. It should be understood that impact calculations of the effects on health of emissions from incinerators are done by using the coefficients derived from epidemiological studies. Because we do not know with certainty the active components of the ambient aerosol, coefficients linking effects on health with changes in mass concentrations (PM_{10} and/or $PM_{2.5}$) are used in the impact calculations. At present we have no clear epidemiological evidence to distinguish between the toxicity of samples of particles collected for PM_{10} or $PM_{2.5}$ measurements in different areas. National policy (Defra, 2007a,b) and the EC Directive on Ambient Air Quality and Cleaner Air for Europe (European Parliament and Council of the European Union, 2008) are based on the assumption that particles collected for PM_{10} and $PM_{2.5}$ measurements do not differ in their effects on health from place to place. In this context it is worth noting that PM_{10} and $PM_{2.5}$ samples from around the world can vary substantially in their chemical composition and size distribution but nonetheless exhibit similar concentration-response coefficients in time-series epidemiological studies. It is accepted that this view could change and that monitoring of chemical characteristics of the ambient aerosol (for example, its metallic components), the number of particles per unit of volume of air, the total surface area of particles per unit volume of air, or the capacity of particles to generate free radicals could prove more valuable than measurements of mass concentrations (PM_{10} and $PM_{2.5}$). But none of this is yet well

established and international and national regulations are currently framed in terms of mass concentrations. It seems reasonable that these regulations and the approaches upon which they are based should be applied to considerations of the effects on health of particles emitted by incinerators. It may be asked why studies of the specific impacts on health of the small increases in local concentrations of particles produced by incinerators are not done routinely. The main reason for this is that the concentration increment produced by incinerators is likely to be too small to allow an impact on health to be identified in the local population.

13. It is sometimes claimed that PM_{10} measurements ignore particles most likely to be deposited in the lung, or, more specifically, in the gas exchange zone of the lungs. This is incorrect and stems from a misunderstanding of the term PM_{10}. Tapered element oscillating microbalance (TEOM) monitors are equipped with a sampling head that selects essentially all particles of less than 10 μm aerodynamic diameter. PM_{10} measurement is designed to collect effectively all those particles small enough to pass the upper airways (nose, mouth, pharynx, larynx) and thus of a size that allows a chance of deposition in the lung. $PM_{2.5}$ is intended to represent that fraction of the aerosol with a high probability of deposition in the gas exchange zone of the lung in vulnerable individuals. It will be obvious that PM_{10} includes $PM_{2.5}$ and that $PM_{2.5}$ cannot exceed PM_{10} in any given sample of air.

14. It is sometimes, further, claimed that PM_{10} or $PM_{2.5}$ do not include nanoparticles present in the air. This is also incorrect. Nanoparticles are efficiently collected by PM_{10} and $PM_{2.5}$ samplers but make only a small contribution to the results expressed as PM_{10} or $PM_{2.5}$. If particles of less than 100 nm diameter alone were collected from a known volume of air and weighed, the resulting concentration could be expressed as $PM_{0.1}$ (100 nm = 0.1 microns). In a sample of air collected in a UK urban area on a typical day we might expect results similar to those given below:

PM_{10}	20 μg/m^3
$PM_{2.5}$	13 μg/m^3
$PM_{0.1}$	1-2 μg/m^3

PM_{10} includes and exceeds $PM_{2.5}$ which in turn includes and exceeds $PM_{0.1.}$

15. It is quite correct to say that nanoparticles make a large contribution to the number of particles per unit volume of air. Particles of less than about 500 nm in diameter dominate the number concentration of ambient particles. It might be correctly suggested that if a specified source, for example an incinerator, produced mainly nanoparticles, changes in local mass concentrations (PM_{10} and to a lesser extent $PM_{2.5}$) would not reflect the increase in numbers of particles in the air. We do not, however, know how to interpret measurement of number concentrations of particles in health terms. Work in this area is developing. It may be that, although the evidence is as yet weak in comparison with that relating to mass concentrations, particle numbers will link with some effects on health better than mass concentrations. No generally accepted coefficients that allow the use of number

concentrations in impact calculations have yet been defined. As stated above, regulations are currently framed in terms of mass concentrations and it is unreasonable to expect local health professionals to interpret number concentrations in quantitative health terms when national experts have not yet judged that the evidence is sufficient to do so. COMEAP will be looking at whether quantification of the effects of particle number concentrations is possible as part of its work on the quantification of the health effects of air pollution. No Air Quality Standards are defined in terms of number concentrations of particles.

16. The contribution made by waste incineration to national emissions of particles is low. Data provided by Defra (National Emissions Inventory www.naei.org.uk) show that 2006 national emissions of PM_{10} from waste incineration are 0.03% of the total compared with 27% and 25% for traffic and industry respectively[2]. This low proportion is also found at a local level – the Environment Agency have informed HPA of one incinerator modelling study that found a modelled ground level increment in PM_{10} of 0.0005 $\mu g/m^3$ as an annual average. The increment in $PM_{2.5}$ could not exceed this, and would be likely to be lower. In addition, Defra is expanding its general $PM_{2.5}$ monitoring and will scrutinise this to see if any individual sources make a noticeable addition to measured concentrations.

17. Questions are often asked about the effects of air pollutants, including those emitted by waste incineration, on children's health. The World Health Organization (WHO) in its 2005 report on Air Pollution and Children's Health and Development, concluded that there was an association between air pollution and infant mortality that appeared to be mainly due to particulate air pollution. COMEAP, in a 2008 statement on Air Pollution and Children's Health, endorsed WHO's general conclusions although the COMEAP statement does not comment on which pollutant is likely to be responsible. Annexes to the statement indicate that, of the studies published since the WHO report, some find effects of particulate air pollution and some do not. Metrics of particulate air pollution used in these studies included PM_{10} and total suspended particulates, as well as $PM_{2.5}$. The size of the effects reported in these studies relates to large changes in $PM_{2.5}$, larger than would be expected to be caused by the operation of an incinerator. Given the small effects of incinerators on local concentrations of particles, it is highly unlikely that there will be a detectable effect of any particular incinerator on local infant mortality.

18. When carrying out studies which investigate health effects around point sources of pollution such as incinerators, or when mapping health effects around such sources, it is important to control for other factors which can influence the health outcomes under investigation before drawing any conclusions. So when investigating the effect of a source of $PM_{2.5}$ emissions on infant mortality rates, it would be important to control for other sources of $PM_{2.5}$ emissions, and for factors which are known to influence infant mortality

[2] National Atmospheric Emissions Inventory PM_{10} http://www.naei.org.uk/emissions/emissions_2006/summary_tables.php?action=unece&page_name=PM1006.html'

rates, for example, socio-economic factors or ethnicity. Maps showing death rates or levels of morbidity are useful in raising hypotheses, but they do not supply evidence of cause and effect.

Carcinogens

19. Chemicals which cause cancer are described as carcinogens. For risk assessment purposes, carcinogens are divided into two groups depending on their mechanism of action:

(a) Genotoxic carcinogens: these induce cancer by a mechanism that involves the compound itself, or a metabolite, reacting directly with the genetic material of cells (DNA), producing a mutation. This process is called mutagenicity. It is theoretically possible that one "hit" on DNA may produce a mutation that can eventually develop into a tumour. The assumption is thus made for genotoxic carcinogens that they do not have a threshold and that any exposure is associated with an increase in risk, albeit this may be very small. Most of the known human chemical carcinogens are in this group, e.g. aflatoxins, benzene, 1,3-butadiene, 2-naphthylamine, polycyclic aromatic hydrocarbon (PAH) compounds.

(b) Non-genotoxic carcinogens: these induce cancer by mechanisms that are not based on mutagenicity. These chemicals give negative results in the well recognised tests for mutagenicity. Unlike the genotoxic carcinogens, which are characterised by a common mechanism, there are a number of different mechanisms involved. Examples include sustained cell proliferation in a sensitive tissue (resulting in expression of a spontaneous mutation) due to cytotoxic effects, hormonal stimulation or immunosuppression. These effects have a threshold based on the precursor toxicological effect such as cytotoxicity, i.e. there is a level of exposure below which they do not have an effect. Examples of such compounds are oestrogens and 2,3,7,8-tetrachlorodibenzo-para-dioxin (TCDD or "dioxin").

20. In the air pollution field, genotoxic carcinogens are the major focus of interest. In the following discussion, the term "carcinogens" is used to represent genotoxic carcinogens.

21. The carcinogenic effects of PAH compounds can be identified by means of studies in experimental animals only at very much higher concentrations than occur in ambient air. These high exposures are necessary because practical limitations regarding the number of animals used in these tests mean that they cannot reliably detect increases in tumour incidence below a few percent. However, for public health purposes, the principal concern is about effects that occur at a much lower incidence in the human population, but are undetectable in animal studies. The calculation of cancer risk at low environmental exposures from mathematical modelling of

the results from the high dose animal data presents great difficulty. The expert advisory committee, the Committee on the Carcinogenicity of Chemicals in Food, Consumer Products and the Environment (COC) has consistently expressed concern at the use of such modelling to extrapolate to levels of exposure that are orders of magnitude lower than the observed range. This was most recently stated in the 2004 guidelines. (The reasons are based on the fact that the various models available do not take into account the biological complexity of the carcinogenesis process, the extrapolations are based on a few data points over a very narrow and high dose range, and very wide variations in risk estimates are produced depending on the models used. Their use gives an impression of precision that cannot be justified). The COC does not recommend their use for routine risk assessment.

22. In some cases, carcinogenic effects have been demonstrated in epidemiological studies in humans. Such studies have almost always involved occupational exposure where workplace levels in the past may have been much higher than those in ambient air. It is difficult to demonstrate the effects of exposure to ambient concentrations of carcinogens (the concentrations are so low that vast numbers of people would need to be studied to produce clear results) but such effects are assumed to be possible, on the grounds that there is no threshold for the effects of many of these compounds. If good quality epidemiological studies are available it is possible to derive models of the relationship between exposure and effect that allow prediction, with some confidence, of likely cancer incidence at ambient concentrations. It should be noted, however, that the actual accuracy of such predictions cannot be assessed and such extrapolations still involve some considerable uncertainty and should be used with caution.

23. The Expert Panel on Air Quality Standards (EPAQS) has recommended air quality standards for benzene, 1,3-butadiene and PAH compounds using a different approach from that used by the World Health Organization (WHO), which is based on quantitative risk assessment. This is because of the concerns of the COC regarding the use of mathematical models to estimate cancer risk. Indeed, the COC endorsed the approach used by EPAQS. This involved the application of Uncertainty Factors to the results of studies of the effects on man of exposure to high concentrations of the carcinogens specified above. Standards derived in this way do not offer a complete guarantee of safety (this is impossible with non-threshold compounds) but do define concentrations at which the risks to health are likely to be very small and unlikely to be detectable. If it is found that incinerators emit the carcinogens considered by EPAQS, it is reasonable to compare the augmented local concentration (i.e. the local background concentration plus the increment contributed by the incinerator) with the EPAQS standard. If this is not exceeded it may be reasonably assumed that the additional risk imposed by the emissions is minimal. If, on the other hand, the emissions cause the local concentrations to exceed the EPAQS standard(s), the appropriate regulator would need to decide whether the additional risk posed by the incinerator was a cause for concern and what further reductions may be necessary.

Dioxins

24. It is recognised that there are particular concerns about emissions of dioxins from incinerators. The HPA and DH are advised on the health effects of such compounds by the independent expert advisory committee, the Committee on the Toxicity of Chemicals in Food, Consumer Products and the Environment (COT). The COT has recommended a tolerable daily intake (TDI) for dioxins, which is the amount which can be ingested daily over a lifetime without appreciable health risk. This TDI is based on a detailed consideration of the extensive toxicity data on the most well studied dioxin, TCDD, but may be used to assess the toxicity of mixtures of dioxins and dioxin-like PCBs by use of Toxic Equivalency Factors, which allow concentrations of the less toxic compounds to be expressed as an overall equivalent concentration of TCDD. These toxicity-weighted concentrations are then summed to give a single concentration expressed as a Toxic Equivalent (TEQ). The system of Toxic Equivalency Factors (TEFs) used in the UK and a number of other countries is that set by the World Health Organization (WHO)[3], and the resulting overall concentrations are referred to as WHO-TEQs (van den Berg, 2006). Thus, the COT has recommended a tolerable daily intake for dioxins of 2 picograms WHO-TEQ/kg body weight/day based on the most sensitive effect of TCDD in laboratory animals, namely, adverse effects on the developing fetus resulting from exposure *in utero*. As this was the most sensitive effect it will protect against the risks of other adverse effects including carcinogenicity. The advice of the other sister committees, COC and the Committee on Mutagenicity of Chemicals in Food, Consumer Products and the Environment (COM), informed the conclusion, namely that dioxins do not directly damage genetic material and that evidence on biological mechanisms suggested that a threshold based risk assessment was appropriate. The full statement is available (COT, 2001).

25. The majority (more than 90%) of non-occupational human exposure to dioxins occurs via the diet, with animal-based foodstuffs like meat, fish, eggs, and dairy products being particularly important. Limited exposure may also occur via inhalation of air or ingestion of soil depending on circumstances. Regarding emissions from municipal waste incinerators, the current limit for dioxins and furans is 0.1 nanogram per cubic metre of emitted gases. A nanogram is one thousand millionth of a gram. Inhalation is a minor route of exposure and, given that Defra has calculated that incineration of municipal solid waste accounts for less than 1% of UK emissions of dioxins[4], the contribution of incinerator emissions to direct respiratory exposure of dioxins is a negligible component of the average human intake. However, dioxins may make a larger contribution to human exposure via the food chain, particularly fatty foods. Dioxins from emissions could also be deposited on soil and crops and accumulate in the food chain via animals that graze on the pastures,

[3] Note: The Waste Incineration Directive (2000/76/EC) sets Air Emission Limit Values for dioxins using a slightly different system of TEQs i.e. international- or I-TEQs, which vary slightly from WHO-TEQs.

[4] Review of Environmental and Health Effects of Waste Management: Municipal Solid Waste and Similar Wastes**.** Extended Summary. Enviros, University of Birmingham and Defra. May 2004.

though dioxins are not generally taken up by plants. Thus the impact of emissions on locally produced foods such as milk and eggs is considered in deciding whether to grant a permit. These calculations show that, even for people consuming a significant proportion of locally produced foodstuffs, the contribution of incinerator emissions to their intake of dioxins is small and well below the tolerable daily intake (TDI) for dioxins recommended by the relevant expert advisory committee, Committee on Toxicity of Chemicals in Food, Consumer (see http://cot.food.gov.uk/cotstatements/cotstatementsyrs/cotstatements2001/dioxinsstate).

Epidemiological studies: municipal waste incinerators and cancer

26. The COC has issued two statements on the cancer epidemiology of municipal waste incinerators. The initial statement followed a review of a large study by the Small Area Health Statistics Unit which examined cancer incidence between the mid 1970s and the mid 1980s in 14 million people living within 7.5 km of 72 municipal solid waste incinerators in Great Britain[5] (Elliott *et al*, 1996; COC, 2000). Prior to this there had been very few studies of cancer mortality around municipal waste incinerators and none in the UK. The incinerators studied by Elliott *et al* (1996) were the older generation operating prior to introduction of strict emission controls and were more polluting than modern incinerators. After considering this study, the COC concluded that: "*any potential risk of cancer due to residency (for periods in excess of 10 years) near to municipal solid waste incinerators was exceedingly low, and probably not measurable by the most modern techniques"* (COC, 2000).

27. In 2008, the Committee reviewed seven new studies on cancer incidence near municipal solid waste incinerators which had been published since 2000 (Comba et al, 2003; Floret et al, 2003; Knox E, 2000; Viel et al, 2000; 2008a and 2008b; Zambon et al, 2007). All had studied the older generation of incinerator and three studies were of an incinerator for which emissions of dioxins were reported to have exceeded even the older emission standard. There were problems interpreting most of these studies due to factors such as failure to control for socio-economic confounding or inclusion of emission sources other than municipal waste incinerators. The COC concluded that *"Although the studies indicate some evidence of a positive association between two of the less common cancers i.e. non-Hodgkin's lymphoma and soft tissue sarcoma and residence near to incinerators in the past, the results cannot be extrapolated to current incinerators, which emit lower amounts of pollutants. …Moreover, they are inconsistent with the results of the larger study…carried out by the Small Area Health Statistics Unit."* It concluded that there was no need to change its previous advice but that the situation should be kept under review (COC, 2009).

[5] These included all known municipal incinerators which opened before 1976. Incinerators starting from 1976 were excluded, to ensure an appropriate lag period for development of any cancer associated with the emissions.

Conclusions

28. Modern, well managed incinerators make only a small contribution to local concentrations of air pollutants. It is possible that such small additions could have an impact on health but such effects, if they exist, are likely to be very small and not detectable. The Agency, not least through its role in advising Primary Care Trusts and Local Health Boards, will continue to work with regulators to ensure that incinerators do not contribute significantly to ill-health.

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Glossary

Aflatoxins
Naturally occurring toxins produced by the fungus *Aspergillus sp.*

Aerodynamic diameter
The actual diameter of a spherical particle of unit density with the same terminal velocity as the particle under consideration. The term aerodynamic diameter allows particles of differing densities and shapes to be compared in terms of their likelihood of depositing in the lung.

Air Quality Standard (AQS)
The concentration of a pollutant (expressed, generally, as mass per unit volume) and qualified by an averaging time, regarded as acceptable by an Expert Group or other standard setting body. Air Quality Standards do not provide an absolute guarantee of safety for health.

Ambient aerosol
An aerosol is a suspension of fine particles or liquid droplets in a gas. Ambient refers to the surroundings. In the air pollution context, this refers to the suspension of fine particles in the general outdoor air.

Atherosclerotic plaques
The discrete lesions of the arterial wall in atherosclerosis i.e., disease of the blood vessels involving the accumulation of fatty material in the inner layer of the arterial wall resulting in narrowing of the artery. These fatty deposits are known as plaques.

1,3-butadiene
An industrial chemical used in the production of synthetic rubber. It is also produced by the combustion of petrol and diesel. It is efficiently removed by catalytic convertors.

Carcinogens
Agents that cause cancer. Chemical carcinogens are chemicals that may produce cancer.

Cell proliferation
An increase in the number of cells as a result of cell growth and cell division.

Clotting factors
Substances (proteins) in blood that act in a complex series of reactions to stop bleeding by forming a clot.

Coefficients
A constant multiplication factor. For example, a health effect might increase by 0.5% for every unit increase in the concentration of a pollutant. This can be derived as the slope from a graph relating health effects and pollutant concentrations.

Coronary arteries
The network of blood vessels that supply heart muscle with oxygen-rich blood.

Cytotoxic
Toxic to cells.

Dioxins
This refers to a large group of chemicals with similar chemical structure (chlorinated dibenzo-p-dioxins and chlorinated dibenzo-p-furans). They vary greatly in toxicity, some being very toxic, others showing a similar pattern of toxicity but of lower potency. They are not produced commercially but are formed in small amounts in most forms of combustion (fires etc.). The most studied compound in this series is the highly toxic TCDD (2,3,7,8-tetrachlorodibenzo-p-dioxin).

Dioxin-like PCBs
Polychlorinated Biphenyls (PCBs) are another group of substances, some of which have similar biological activity to dioxins. These are referred to as Dioxin-like PCBs. There are many other PCBs that do not have dioxin-like properties.

Epidemiological studies
Studies of the distribution and the aetiology (causes) of disease in humans.

Free radicals
Highly reactive chemical structures (due to the presence of a chemical species that has lost an electron and thus contains an unpaired electron in the outer shell of the molecule). They are unstable and can react in biological systems with nearby substances such as lipids, proteins or DNA producing damage.

Furans
Chemicals related to furan. Furan contains carbon, hydrogen and oxygen with the carbon atoms and an oxygen atom forming a 5 sided ring.

Gas exchange zone
The part of the lung in which oxygen diffuses from the air to the blood and carbon dioxide diffuses from the blood to the air. The alveoli, alveolar ducts and respiratory bronchioles make up the gas exchange zone.

Immunosuppression
Suppression of the immune system.

Incidence
New occurrence of a disease over a specified time period.

In-utero
In the uterus (womb).

Larynx
Dilated region of the airway above the upper end of the trachea or windpipe. The vocal cords lie within the larynx.

Mass concentration of particles
The mass of particles per unit volume of air. Usually expressed as $\mu g/m^3$ (micrograms per cubic metre).

Metabolite
Chemicals that enter the body can be changed by processes in the body into different chemicals. These are described as metabolites of the original chemical.

Metalloid
An element that is not clearly a metal or non-metal but has some intermediate properties in terms of malleability, ductility, conductivity and lustre. The following elements are generally considered to be metalloids: boron; silicon; germanium; arsenic; antimony; tellurium; polonium.

Meta-analysis
In the context of epidemiology, a statistical analysis of the results from independent studies which aims to produce a single estimate of an effect.

Metric
A measure for something. PM_{10} is a measure (or metric) of the concentration of particles in the air.

Microgram (µg)
One microgram is 1×10^{-6}g. There are 1,000,000 (1 million) micrograms in a gram.

Micron (μm)
This is a unit of length that equals one thousandth of a millimetre.

Mortality
Deaths.

Mortality rate
The number of deaths in a population.

Morbidity
Ill health.

Mutation
A permanent change in the amount or structure of the genetic material (DNA) in a cell or organism which can result in a change in its characteristics. A mutation in the germ cells of sexually reproducing organisms may be transmitted to the offspring, whereas a mutation that occurs in somatic cells may be only transferred to descendent daughter cells.

Nanogram (ng)
One nanogram is 1×10^{-9} gram. There are 1,000,000,000 ng in one gram.

Nanoparticles
These are usually considered to be particles of less than 100 nanometres diameter. One nanometre is a millionth of a mm. To put into some context this is about a ten thousandth of the width of a human hair.

2-naphthylamine
A chemical used in the past in the manufacture of dyes. It is made up from 2 benzene rings with a nitrogen and hydrogen side chain.

Non-Hodgkin lymphoma
A type of malignant cancer of the lymphatic system or lymphoid tissue. Most lymphoma are of this type (as opposed to being Hodgkin lymphoma).

Number concentration of particles
The number of particles found in a specified volume of air, usually 1 cubic metre.

Pharynx
The throat and back of the nose.

Point sources
Sources of pollution from a fixed point in space e.g. an industrial site. The term is used in contrast to mobile sources of pollution e.g. cars.

Polycyclic aromatic hydrocarbons (PAHs)
These are a group of structurally related organic compounds that contain 2 or more fused rings. They are formed as a result of combustion/pyrolysis.

PM_{10}, $PM_{2.5}$
The concentration (expressed in $\mu g/m^3$) of particles generally less than 10μm and 2.5μm respectively[6]. The terms PM_{10} and $PM_{2.5}$ are sometimes used to describe particles of diameter of less than 10 and 2.5 μm respectively but this is not strictly correct: the terms refer to the concentrations of particles and not to the particles themselves.

Picogram (pg)
A picogram is 1×10^{-12} gram. There are 1,000,000,000,000 pg in one gram.

[6] Strictly, particles that pass a sampler entry with 50% efficiency at 10 micrometres or 2.5 micrometres respectively.

Spontaneous mutation
A mutation that occurs as a result of natural processes in cells, as opposed to those that arise because of interaction with an outside agent or mutagen.

Soft tissue sarcomas
These are a rare type of cancer that develop from cells in the soft, supporting tissues of the body such as muscle, fat and blood vessels. They may occur in limbs, chest, abdomen or pelvis and less commonly in head and neck.

TCDD
The most studied dioxin, and the one that is used as a reference compound when considering the toxicity of mixtures of dioxins, is often referred to simply as TCDD. This is an abbreviation of its full chemical name, 2,3,7,8-tetrachlorodibenzo-p-dioxin. It is considered the most toxic dioxin.

TEOM
Tapered Element Oscillating Micro-balance. An instrument used to measure the mass concentration of particles in the air. Particles are collected on a vibrating rod: the mass deposited affects the frequency of vibration of the rod and this, being recorded, allows the mass of particles in the air to be calculated.

Tolerable Daily Intake (TDI)
An estimate of the amount of contaminant, expressed on a body weight basis (e.g., mg/kg body weight) that can be ingested daily over a lifetime without appreciable health risk.

Total suspended particulates
A measure of particles derived by collecting particles of approximately 100 µm or less in a sampler. This includes particles that are too large to enter the lung. The measurement method has generally been superseded by measurement of PM_{10}.

Toxic Equivalency Factor (TEF)
A measure of the relative toxicological potency of a chemical compared to a well characterised reference compound. TEFs can be used to sum the toxicological potency of a mixture of chemicals which are all members of the same chemical class, having common structural, toxicological and biochemical properties e.g. dioxins. In the case of dioxins the reference compound is TCDD.

Toxic Equivalent (TEQ)
This is a method of comparing the total relative toxicological potency within a mixture using TEFs (see above). It is calculated as the sum of the products of the concentration of each chemical multiplied by the TEF.

Ultrafine component
The component of particles less than about 100 nm in diameter.

Uncertainty factors
Value used in extrapolation from experimental animals to man (assuming that man may be more sensitive) or from selected individuals to the general population; for example, a value applied to the No Observed Adverse Effect Level (NOAEL) to derive a TDI. The value depends on the size and type of population to be protected and the quality of the toxicological information available.