Waste incineration—how big is the health risk?  
A quantitative method to allow comparison with other health risks

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ABSTRACT

Objective To assess the health risk from a medium-sized waste incinerator and develop a single comparable figure to quantify overall risk.

Method We used a prospective health risk assessment utilizing US Environmental Protection Agency Human Health Risk Assessment Protocol (HHRAP) for Hazardous Waste Combustion Facilities and UK coefficients for the impact of sulphur dioxide and particulates. Calculations were based on a resident population of 25,398 living within 5.5 km of the site.

Results Anxiety, employment, noise, occupational risks, road accidents, and reduced use of landfill were all considered to have a potential, but unquantifiable, effect on health. Stack emissions over 25 years in a population of 25,398 within 5.5 km of the stack would result in an additional 0.018 cancers, 0.46 deaths brought forward due to sulphur dioxide and 0.02 deaths due to fine particles. The overall risk of dying due to emissions in any one year was $2.49 \times 10^{-7}$ or 1 in 4 million.

Conclusion To facilitate better public understanding of the comparative risk of incinerator emissions, we propose a simple method of deriving a single annual risk figure allowing comparison with the risk of dying from other causes with which the public is more familiar.

Keywords air pollutants, dioxins, environmental exposure, environmental pollution, incineration, risk assessment, mortality, population, public health, Wales

Introduction

Incineration is associated with considerable public concern, which may have a significant harmful effect on the mental, physical and emotional health of local residents, regardless of whether emissions have any direct effect on health.¹² The National Waste Strategy requires increased incineration with energy recovery.³

It is now possible to explore beyond the conventional regulatory statement that ‘emissions are within allowed limits’ and calculate how many deaths or hospital admissions may be advanced or caused by the minutest increase in air pollution.⁴ This allows us to directly address the expressed concerns of local residents that emissions may harm their own health and that of their children, by describing how likely it is that harmful effects will actually occur, clearly communicating risk.⁵

The Departments of Health are encouraging health authorities to undertake health impact assessments (HIAs).⁶ This article describes a risk assessment undertaken as part of a HIA of a planning application for an incinerator designed to burn 52,500 tons of refuse-derived fuel annually to generate electricity, focusing on those health aspects which are of greatest public concern, in particular emissions of carcinogens and fine particles.

Methods

Resident’s concerns were obtained non-systematically through participation in an open public meeting, correspondence and discussion with local authority officers. The planning application, especially the HIA section, was reviewed for activities that may affect health. Data on emissions and predicted maximum ground level concentrations (MGLCs) and average concentration across a 10-km square grid centred on the site were obtained. Pollutant emission...
rates were determined using the limits set by the European Waste Incineration Directive (WID, 2000/76/EC).

Compounds of potential concern (COPC) were identified including dioxins and furans, polycyclic aromatic hydrocarbons (PAHs) and heavy metals. These substances were selected as representative of the potentially most harmful that could be emitted from the proposed facility and were suitable for assessment using the US Environmental Protection Agency (USEPA) Human Health Risk Assessment Protocol (HHRAP) for Hazardous Waste Combustion Facilities. Maximum and average concentrations and dry and wet deposition rates in both vapour and particulate phases were calculated across a 10-km square grid using Atmospheric Dispersion Modeling System version 3 (ADMS3). These data were then used to calculate media concentrations in soil, meat, dairy products and breast milk within USEPA HHRAP. Average daily intakes due to emissions were calculated, and from this cancer risks and number of additional cancers that would result were derived. The tolerable intake of dioxin used was that agreed provisionally by the World Health Organization (WHO) of 70 pg kg\(^{-1}\) body weight (bw) month\(^{-1}\), expressed as a tolerable daily intake of 2 pg kg\(^{-1}\) bw day\(^{-1}\), as preferred by the UK Committee on Toxicity.8

Acute effects associated with sulphur dioxide (SO\(_2\)) was assessed using the dose–response coefficients for deaths brought forward (d.b.f.) developed by Committee on the Medical Effects of Air Pollutants (COMEAP).4 Acute and chronic effects of fine particulate emissions were assessed using coefficients developed by the Institute of Occupational Medicine (IOM) for COMEAP.9 Environmental tobacco smoke was used as a comparison for exposure to ultra-fine particles in incinerator emissions.10,11 Comparative risks of an individual dying in any 1 year were adapted from Calman.12

The resident population (2000) within 5.5 km of the proposed site was obtained using a Geographical Information System (Arcview). Population structure was obtained from the Office for National Statistics. The crude cancer incidence (excluding skin) per 100 000 in Wales in 1994 (the latest year for which published data was available) was 522.0 in males, 497.6 in females and 509.8/100 000 overall.13 We used death rate from all causes in the UK in 1995, 1106.4 per 100 000 population, as used in the COMEAP report.4

These data were then used in the following main comparisons:

(i) Number of additional cancers resulting from dioxin and heavy metal emissions, against all cancers during the same period.

(ii) Number of d.b.f. or additional due to SO\(_2\) and particulate emissions, against all deaths during the same period, including deaths due to existing levels of air pollution.

(iii) Total average annual risk of death due to all major emissions of concern, against risk of death from other causes.

The study did not require approval by the local ethics committee.

### Results

Residents were most concerned about the potential adverse health impact of dioxins, fine particles and carcinogens present in emissions. The potential health effects of the development outlined in the application, both positive and negative, were tabulated (Table 1).

### Cancer risks

Predicted emissions increased background concentrations (annual mean)\(^{20}\) over the 10-km grid by an average of 0.04%
for dioxins, 0.02% for PAHs, 0.13% for arsenic and 0.7% for cadmium. At the point of maximum impact (MGLC), increase over background was at least an order of magnitude greater at 1.5% for dioxins and 16% for cadmium.

Cancer risks are based on the effect of exposure durations of 25, 15 and 1 years for adult, children and infant, respectively. The highest estimated lifetime cancer risk for an adult, child and infant exposed to MGLC of the COPC in emissions were $5.93 \times 10^{-6}$, $7.62 \times 10^{-6}$ and $4.18 \times 10^{-5}$ (via dioxin in breast milk), respectively. The average lifetime risk for an adult, child and infant living within 5.5 km of the site were 1 in 3,955,760 ($2.53 \times 10^{-6}$), 1 in 3,078,433 ($3.25 \times 10^{-7}$) and 1 in 561,188 ($1.78 \times 10^{-6}$), respectively. The main contributor to this risk was cadmium (72%) followed by dioxins (17%), arsenic (10%) and PAH (1%).

The average risk applied to the resident population of 25,398 within 5.5 km (with the same age distribution as the UK, 2000) would result in 0.018 additional lifetime cancers in local residents due to emissions over the proposed 25-year life of the incinerator. Applying the incidence rate of all cancers in Wales in 1994 to the population within 5.5 km, the total number of new cancers from all causes within 5.5 km of the proposed development over 25 years would be 3,237. Therefore, over its operating life of 25 years, there is a 1.8% probability that, because of these emissions, one additional cancer would be caused, increasing the total from 3,237 to 3,238.

**Dioxin intake**

Because cancer is not the most sensitive end point for dioxin exposure, it is also necessary to compare additional intake due to emissions against the tolerable intake. Data in the application showed increased dioxin intake would be lowest in non-farming residents (adults and children) and highest in farm residents (adults and children). The estimated average daily additional intake of dioxin in farm adults and children would be 0.009 pg kg$^{-1}$ bw day$^{-1}$, representing <1% of both the WHO tolerable intake (2 pg kg$^{-1}$ bw day$^{-1}$) and of the UK current average intake in toddlers and adults (5.1 pg kg$^{-1}$ bw day$^{-1}$ and 1.8 pg kg$^{-1}$ bw day$^{-1}$, respectively). For other residents, and children at a school 800 m from the site, increases represent <1/100th of tolerable intake. The UK Committee on Toxicity states that the current UK intakes are unlikely to pose a risk to health, and the WHO recommends breastfeeding despite the presence of dietary dioxin in breast milk.17,22

**Air pollution risk**

On average, over the 10 km grid, emissions increased estimated existing background concentrations of particulates by 0.01% (24 h mean), nitrogen dioxide by 2.6% (1 h mean) and SO$_2$ by 0.5% (1 h mean). Emissions represented a small proportion of the Air Quality Standards at the point of maximum impact (MGLC) ranging from 9.6% for nitrogen dioxide (1 h mean) to 0.08% for particulates (annual mean).

The average predicted concentration of SO$_2$ from emissions over the 10-km grid was 0.11 $\mu$g m$^{-3}$ (24 h mean). COMEAP calculated the dose–response coefficient$^4$ for SO$_2$ as a 0.6% increase in all cause mortality (d.b.f.) per 10 $\mu$g m$^{-3}$ (24 h mean) increase in concentration. In main residential areas within 5.5 km, the average predicted concentration of SO$_2$ due to emissions was 0.11 $\mu$g m$^{-3}$ (24 h mean). Applying the death rate from all causes in the UK in 1995 to the local population, the total number of deaths from all causes within 5.5 km of the proposed development over 25 years would be 7,025. Therefore, there would be $0.006 \times (0.11/10) \times 7,025 = 0.46$ d.b.f. Therefore, over its operating life of 25 years there is a 4.6% probability that, because of SO$_2$ emissions, one of the 7,025 deaths from all causes would be brought forward, resulting in the loss of several months of life. In comparison, existing levels of air pollution from all other sources may bring forward around 211 (3%) of these deaths (assuming average UK levels).

The average predicted concentration of PM$_{10}$ (Particulate matter with a diameter of 10 microns or smaller) particles from emissions over the 10-km grid was 0.001 $\mu$g m$^{-3}$ (annual mean). Using a coefficient$^9$ of 0.3% increase in all cause mortality per 1 $\mu$g m$^{-3}$ PM$_{2.5}$ (Particulate matter with a diameter of 2.5 microns or smaller), and assuming all stack emissions are PM$_{2.5}$ the increase in mortality rate because of emissions would be $0.001 \times 0.3\% = 0.0003\%$. Therefore, fine particulate emissions would result in 0.000003 $\times 7,025 = 0.02$ additional deaths over 25 years. Of 7,025 all cause deaths within 5.5 km over 25 years, exposure to existing background fine particle levels in the area would contribute to around 240 (3.4%) of these deaths (assuming PM$_{2.5}$ = background PM$_{10}$ $\times 0.6 = 19 \times 0.6 = 11.4 \mu$g m$^{-3}$). Therefore, over its operating life of 25 years, there is a 2% probability that, because of fine particle emissions, one death would occur in addition to the 236 deaths related to existing levels of fine particle pollution in the area from all other sources, particularly vehicle emissions, increasing the total from 7,025 to 7,026.

**Overall risk**

Rather than illustrate risk using stand-alone comparisons, such as comparing particle exposure with environmental tobacco smoke,10,11,24 impacts on cancer and mortality were combined to provide a crude estimate of the overall increase in risk of death due to emissions, assuming at least a year of life lost per death. Deaths over 25 years were due to carcinogens (assuming 0.018 cancers = 0.018 additional deaths), SO$_2$ (0.46 d.b.f., which assuming 1 d.b.f. = 3 months of additional life lost$^{25}$ = 0.12 deaths, in which 1 year of life is lost) and...
particulates (0.02 additional deaths); other emissions had a much smaller effect on mortality. Therefore, based on these estimates, there would be 0.018 + 0.12 + 0.02 = 0.158 additional deaths over 25 years in 25 389 persons in the communities within 5.5 km of the development. Therefore, the average individual risk of dying in any 1 year because of the extra local pollution would be 1 in \[\frac{0.158}{25,398 \times 25}\] = 2.49 \times 10^{-7} = 1 in 4 018 670. This risk can be compared with the risk of dying from other more familiar causes (Table 2).

For example, risk of dying from incinerator emissions is over 500 times lower than the risk of dying in a road accident.

Discussion

Main findings of this study

We were able to illustrate the magnitude of the impact of incinerator emissions on health by manipulating mortality and other data, making broad assumptions to enable simple comparisons to allow the public to judge whether emissions represent a large or small risk. It was not possible to calculate the risks avoided by not disposing of this waste in other ways or determine the direction of the overall health effects of the proposed development.6

What is known already

Several studies suggest that the risk posed by emissions from modern incinerators to populations living near them is very low. Elliott et al. found an increased risk of liver cancer of around 1 in 160 000 within 1 km of 72 incinerators operating before 1976, which the UK Committee on Carcinogenicity found reassuring.26–28 Knox studied childhood cancers around the same 72 incinerators, and examined a suggestion by Elliott et al. that older (pre-1955) incinerators were more toxic than more recent (1955–76) plants.29 He stated that his data tended ‘to confirm the suggestion’ and ‘seemed to exonerate the more modern plants’, as incinerators operating after 1955 had no detectable impact on cancers in children. The Environment Agency estimated that of the 24 000 d.b.f. annually in the UK by all sources of air pollution, fewer than three of these were because of pollutants from the 10 incinerators operating in 2000.30 Cancer risks due to dioxin emissions from a Spanish incinerator were of the same order of magnitude as found here.31 No cellular evidence of increased exposure to genotoxins was found in children living near a Belgian incinerator.32 Several groups have conducted an overview of studies. Perhaps the most authoritative is that of the US National Research Council, part of the National Academies, which, while acknowledging the need for further study, stated ‘when operated properly by well trained employees, modern waste incinerators pose little risk to public health’.33 A report by the Institute for European Environmental Policy came to a similar conclusion.34 In contrast, Greenpeace concluded ‘incinerators are potentially very damaging to health’.35

What this study adds

Although this plant was not a Hazardous Waste Combustion Facility, the USEPA Protocol (USEPA HHRAP) was selected as the most appropriate methodology available. The USEPA protocol evaluates both direct and indirect risks to human health. Direct exposure results from the direct inhalation of vapours and dusts, whereas indirect exposure results from contact of human receptors with soil, plants, meat or water bodies on which emissions may have been deposited. This protocol has been used in the US to support planning permits associated with proposed waste combustion facilities by performing site-specific risk assessments that can be protective of human health and the environment. The protocol comprises facility characterization, air dispersion and deposition modeling, exposure assessment and risk and hazard characterization. It involves over 250 parameters describing physical, chemical and biological processes, toxicity and exposure scenarios. Many of these parameters are the USEPA default values to ensure conservatism in the risk calculation, and those parameters related to exposure time and frequency, toxicity and consumption rates for meat and vegetables have been modified to reflect site-specific conditions. Nevertheless, it is more likely that some levels of uncertainties would exist. At the time of analysis, there were no UK-specific guidelines and related models for undertaking human health risk assessment for organic compounds and heavy metals. There were many UK models, none of which included the HIA of organic compounds and metals. The USEPA HHRAP was the only integrated risk calculation package available that takes into account
of air dispersion and deposition and allows hazard index and cancer risks to be evaluated.

We used death rate from all causes in the UK in 1995, not more recent data, because this figure was used in the COMEAP report and its use facilitated comparison with data from that report. Our decision not to include nitrogen dioxide in our calculations was based on COMEAP’s view that robust evidence of effect on mortality was lacking. It is accepted that, subject to several conditions, COMEAP coefficients can be used to calculate the impact of point sources of pollution. Our study focused on quantifiable mortality, although air pollution also has an impact on hospital admissions and respiratory symptoms.

Limitations to this study
Several caveats must apply. First, our understanding of the health impact of environmental pollution is constantly improving but has limitations. Our study is based on the air quality impact assessment using a well-established ADMS model. The WID limits are upper limits on the emissions from the facility and as such they are the worst-case values and actual emissions are expected to be lower. The predicted MGLC for primary air pollutants, dioxins, PAHs and metals under the WID limits would not exceed the relevant statutory limits and guidelines imposed for the protection of human health and the environment.

The methods and assumptions used are useful for the purposes of illustration but are not epidemiological projections. They are an attempt to overcome criticism of work by other authors by combining the acute and chronic effects of air pollution, dioxins and other carcinogens on mortality. The data are derived from different types of study, so summation is problematic, but approximated comparisons have been acceptable elsewhere to estimate the health impact of air pollution, including conversion of d.b.f. to years of life lost. Whether an individual risk results from voluntary activity or is involuntary affects its public acceptability. However, most of the comparison risks presented are involuntary.

Conclusion
Government strategy requires increased incineration of waste. However, this policy is unlikely to gain wide public acceptance unless the government finds ways to clearly communicate the risks involved to address public anxiety. If decision makers, and local residents, are to understand the public health implications of incineration, it is essential for them to appreciate the magnitude of the risks involved. When considering the toxicity of emissions from industrial processes, it is obvious that harmful effects are possible, but a simple method to explain the size of the overall risk has been elusive. The only way we can properly understand how big the risk of harm actually is in comparison with other exposures or risks with which we are more familiar. This risk assessment places data in context by providing a single risk figure for comparison with more familiar risks or exposures. The quantification methods used could be applied to other point sources of industrial emissions.

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Statement of contribution
M.C. carried out the original calculations of cancer risk using the USEPA HHRAP. R.R. calculated non-cancer risks, calculated comparable risks and conceived the idea of aggregating emission risks into a single figure.

Conflict of interest
M.C. had been previously employed by Environmental Resources Management Ltd, SECOR Ltd and Westlakes Scientific Consulting Ltd (we do not believe this creates a conflict, but include it for information).

References


