Cambridge Friends of the Earth's Written Submission Opposing the Application by Medworth CHP Limited for an Order Granting Development Consent for the Medworth Energy from Waste Combined Heat and Power Facility in Wisbech Cambridgeshire

Reference MVV- Medworth EN010110

Cambridge Friends of the Earth is concerned about this application and wishes to raise the following objections:

HEALTH EFFECTS - EMISSIONS

Cambridge Friends of the Earth's main concerns are, perhaps unsurprisingly, air pollution and carbon dioxide emissions.

With regards to air pollution, perhapsthe most damming evidence against waste incineration comes from a UK report entitled "The Health Effects of Waste Incinerators" by the British Society for Ecological Medicine was conducted in 2005 and then updated in 2008 (See Attachment1).

The Preface to this report states:
"Since the publication of this report, important new data has been published strengthening the evidence that fine particulate pollution plays an important role in both cardiovascular and cerebrovascular mortality (see section 3.1) and demonstrating that the danger is greater than previously realised. More data has also been released on the dangers to health of ultrafine particulates and about the risks of other pollutants released from incinerators (see section 3.4). With each publication the hazards of incineration are becoming more obvious and more difficult to ignore."

A further, telling, quote from this report states:
" ……Large studies have shown higher rates of adult and childhood cancer and also birth defects around municipal waste incinerators: the results are consistent with the associations being causal. A number of smaller epidemiological studies support this interpretation and suggest that the range of illnesses produced by incinerators may be much wider"

Other reports concerning the risks from waste incinerator emissions include:

A report from the Energy Justice Network (See Attachment2) states that, in comparison to Coal Fired Power Plants, in order to release the same amount of energy Incineration releases:

6 times more Lead (See attached supplementary document: 'Cambridge FoE Additional Submission - Cadmium EN010110')

3 times more Nitrogen Oxide.

A gas that primarily contributes to eye, nose, throat and lung irritation and respiratory problems like shortness of breath that can trigger asthma.

2 times more Carbon Monoxide

A contributor to the formation of ground-level ozone pollution with the potential to aggravate asthma.

70% more Sulphur Dioxide.

A cause of acid rain which is also damaging to lungs, with even short exposures to ambient levels having the potential to cause bronchial constriction and increased asthma symptoms.

Furthermore, many studies have shown a link between waste incinerators and increased incidences of cancer in areasdownwind and/or in proximity to such sites; an example of such a study can be found in Attachment 3, **'Study of the incidence of cancers close to municipal solid waste incinerators' by the French Institute of Public Health and Survellance.**

We believe that this risk alone would be enough to turn down the application

Following on from this, Cambridge Friends of the Earth is particularly concerned about the production and dispersal of dioxins and related compounds from the proposed incinerator.
The combustion of any heterogenous waste stream risks the production of extremely toxic and carcinogenic dioxin compounds. Each substance in an incinerator's feedstock has an optimum temperature at which it should be combusted to prevent the possibility of dioxins being produced; chlorinated organic compounds in particular require particularly high temperatures during combustion to preclude the production of dioxins. Cambridge Friends of the Earth believes it is highly unlikely that high enough temperatures will be achieved in the proposed incinerator to prevent the production of dioxins from, amongst other compounds, chlorinated organic compounds and therefore the production of dioxins by the incinerator is highly likely.

HEALTH EFFECTS – VEHICLE MOVEMENTS

Considering the large volumes of waste that the proposed incinerator is designed to handle, we feel that the consequent increase in vehicle (mostly HGV) movements are a major health concern that should count against this application.

Emissions from the additional (diesel powered) HGV movements must be put together with the emissions from the Incinerator itself.

Issues surrounding diesel pollution are outlined in Attachment 4.

HEALTH EFFECTS - FLY ASH

Waste Incinerators produce bottom and fly ash which amount to a least 25% by volume of the original waste – a percentage of this is now highly toxic and requires transportation to specialist toxic landfill sites.

The British Society of Ecological Medicine states:

"This fly ash is light, readily windborne and mostly of low particle size. It represents a considerable and poorly understood health hazard."

See Page 6 of Attachment 1

CARBON DIOXIDE EMISSIONS

Evidence from UK Without Incineration and Isonomia show that for every tonne of waste burned more than a tonne of CO2 is released into the atmosphere (See Attachment 5)

This means that incineration has a higher carbon intensity than conventional use of fossil fuels and should in no way be viewed as 'clean' or 'green' energy.

Friends of the Earth estimate 33% more greenhouse gases are released during incineration of waste than during use of traditional gas-fired power stations https://www.foeeurope.org/incineration .

The Energy Justice Networks suggests the figures are more like 2.5 times more CO2 is released during incineration compared to coal fired power plants (See Attachment 2)

It is hard to see how incineration will help towards the carbon emissions reductions that the UK is legally required to comply with as part of the Carbon Budget section of the Climate Change Act.

In order to reach future targets the UK will need to decrease emissions by 3% a year.

WILDLIFE IMPACTS

A waste incineration plant operating 24/7 with the associated noise, light and odour will affect not only the new residents in these developments and beyond – but it must also be considered that a number of wildlife species rely on smell, dark conditions and use calls to communicate.

For example, the Bat Conservation Trust is particularly concerned with Light pollution and its effect on invertebrates, birds and mammals. Noise pollution interferes with the way animals communicate, mate and catch prey.

There is now also evidence that bees and other pollinators are less effective due to odour pollution.

Following on from this, despite reassurances from Incinerator Operators, there are many documented cases of residents experiencing unexpected levels of noise, odour and light both during construction and during 'normal' operating conditions(See Attachment 6).

IMPACT ON RECYCLING

If this Incinerator is allowed to operate, the County Council will find it hard to conform to the Waste Hierarchy of Reduce, Reuse, Recycle, Residual and will have its progress towards the circular economy that will ultimately be required in order to sustainably address the waste issue .

Priority should instead be given to the development of the necessary infrastructure to ensure high re-use, recycling (including composting) rates including the development of the necessary separate collection systems (whether door-to-door or through collection points and/or civil amenities):

https://environment.ec.europa.eu/topics/circular-economy_en#main-content

Published waste statistics show that there is a depression of recycling rates related to percentages that are being sent to incinerator plants. An example of this is shown in the graph below from the Bedfordshire Against Covanta Incinreator campaign group:

REGULATORY COMPLIANCE AND NON-COMPLIANCE

A recent study by the Right Waste Right Place campaign sponsored by Environment Agency, Natural Resources Wales, CIWM and ESAET showed that 56% of UK companies were not complying with the correct processes regarding waste and recyclables. The Study further shows that 1/4 of these Companies were not sorting waste into recyclable and residual at all.

MONITORING

Cambridge Friends of the Earth has serious reservations regarding the Environment Agency's emission monitoring standards, which we assume the Operator will be subjected to, as we consider these standards and their application to be inadequate.

Our concerns are focused on the quality and nature of monitoring and the way it is carried out and can be summarized as follows:

Inadequate range of substances monitored

Only a tiny fraction of the hundreds of chemicals released from an incinerator will be measured. Important pollutants like **dioxins, heavy metals and PM2.5 particulates will be virtually unmonitored during everyday operation of the incinerator**. Usually, only half a dozen pollutants are measured continuously in the stack and about another half dozen are measured occasionally (usually 6 monthly for the first year and then yearly) by spot monitoring – these include heavy metals and dioxins.

***M*onitoring of emissions of waste incinerators is conducted by spot checks rather than continuously.**

A system in which the high emissions that can occur during a period of poor operation can be missed simply because of spot checks only is totally unacceptable.

Infrequent monitoring means that the operator and the public might never find out about unintended periods of high emissions and then steps might never be taken to deal with the consequences.

The Operator will receive advanced notice of inspections from the Environment Agency.

We consider that unannounced visits are necessary. Levels of emissions achieved under test conditions or when inspections occur by prior arrangements are likely to be far lower than under real life conditions. The US Environmental Protection Agency and Occupational Safety and Health Administration conducted **62 unannounced visits** and no less than **69% of inspections led to summons for violations of regulations** (In the UK inspections are by prior arrangement).

Finally, no mention is made of any proposed monitoring of body burdens of possible pollutants from the emissions in the local population or the build-up of pollutants in the locality. **There should be regular monitoring of dioxins in cattle and other farm animals, together with checks for pollutants in dust, vegetation and in the bodies of local inhabitants** otherwise, as with the rest of the Environment Agency’s not fit for purpose monitoring regime, we will assume that the principal of ‘Don’t ask if you don’t want to know’ applies.

Trash Incineration More Polluting than Coal

Trash incinerators are the dirtiest way to make electricity by most air pollution measures. Even with air pollution control equipment, trash incinerators emit more pollution than (less controlled) coal power plants per unit of energy produced. Coal power plants are widely understood as the most air-polluting energy source, but few realize how much worse trash incinerators are for air quality.

> To make the same amount of energy as a coal power plant, trash incinerators in 2018 released 65% more carbon dioxide (CO2), as much carbon monoxide, three times as much nitrogen oxides (NOx), five times as much mercury, nearly six times as much lead and 27 times more hydrochloric acid (HCl).

This is not a radical conclusion. The New York State Department of Environmental Conservation proved, in a 2011 analysis, that the state's 10 trash incinerators are dirtier than the 8 coal-burning power plants that were still operating at the time (all of the coal power plants have since closed, but the 10 incinerators remain). Except for sulfur dioxide, trash incinerators are dirtier than coal on the six other pollutants the state compared (nitrogen oxides, carbon monoxide, hydrochloric acid, mercury, lead, and cadmium).[1] See the chart at the bottom of this page for the summarized New York data.

Dioxins/furans: Trash incinerators are well known to be the largest source of the most toxic man-made chemicals known to science – dioxins. The latest national inventory of dioxin emissions – by the U.S. Environmental Protection Agency in 2006, looking at data from 1987, 1995, and 2000 – shows that trash incineration has gone from the largest source of dioxin emissions in 1987 and 1995 to the 4th largest source in 2000. However, if one accounts for the lack of continuous monitoring and the consequent massive underestimation of dioxin emissions from incineration, trash incineration is still the largest source of dioxins, despite the cleanup or closure of some of the dirtiest incinerators.[2]

Between 2000 and 2005, new dioxin emissions limits were implemented for trash incinerators, requiring the worst dioxin polluting incinerators to clean up or shut down. EPA and the trash incinerator industry tout that dioxin emissions from trash incinerators have been reduced by over 99% between 1990 and 2005. Even with this large reduction, and without even accounting for the aforementioned underestimation from lack of continuous monitoring, trash incinerators release 28 times as much dioxin than coal power plants do to produce the same amount of energy.[2][3][4]

Mercury is another notoriously toxic pollutant released from incinerators. It is a potent neurotoxin

that accumulates in the fatty tissue of fish once in the environment. Mercury emissions from trash incineration were a close second to coal power plants in the early 1990s, which is rather incredible given the much larger size of coal power plants and the fact that there are about five times as many coal plants as incinerators. Pollution controls required on trash incinerators reduced the industry's mercury emissions 96% by 2005.[5] However, even with this dramatic industry-wide reduction, trash incineration still put out 5.3 times as much mercury as coal plants do to produce the same amount of energy, according to the latest available national data from 2018.[5] A state-wide analysis by the New York State Department of Environmental Conservation found that, in 2009, the state's 10 trash incinerators released 14 times as much mercury per unit of energy than the state's 8 coal power plants – high enough that the total amount of mercury coming from the incinerators was higher than the emissions from the coal plants, even without adjusting for size (coal plants are far larger facilities).[1]

Lead is another well-known toxic chemical that diminishes intelligence and – by lowering dopamine levels in the brain – may even be tied to increases in violent behavior and cocaine addiction.[6][7][8] Trash incineration releases more than six times as much lead as coal to produce the same amount of energy.[5]

Nitrogen oxide (NOx) pollution primarily contributes to eye, nose, throat, and lung irritation and respiratory problems like shortness of breath that can trigger asthma. Trash incineration releases 3.3 times as much NOx as coal does to produce the same amount of energy.[9]

Carbon monoxide (CO) is also released from trash incinerators at rates comparable coal power plants per 1 MWh of energy produced.[5] Both NOx (directly) and CO (indirectly) contribute to the formation of ground-level ozone pollution, aggravating asthma.[11][12]

Sulfur dioxide (SO2) – famous as a cause of acid rain – is also bad for lungs, with even short exposures to ambient levels causing "bronchoconstriction and increased asthma symptoms."[13] SO2 is one of the rare pollutants where coal plants are worse. Coal plants release two times more SO2 as trash incinerationbsp;to produce the same amount of energy.[5][9]

Hydrochloric Acid (HCl) is linked to acute bronchitis and lung cancer.[14] Trash incineration releases a whopping 27 times more HCl than coal plants to produce the same amount of energy.[5]

Carbon dioxide (CO2) – the prime global warming pollutant – is released at a rate 1.65 times that of coal power plants.[15][16][17]. By analyzing 2018 data from EPA's Greenhouse Gas Reporting Program (GHGRP), we were able to compare data from continuous emissions monitoring systems (CEMS) on both incinerators and coal plants. Other data sources (such as EPA's eGRID database) estimate incinerator emissions with emissions factors. However, according to EPA, "for heterogeneous fuels such as municipal solid waste, CEMS are generally considered the most accurate emissions estimation method."[18]

While comparing incinerators and coal plants, we ruled out facilities that burn more than 5% of another fuel (to ensure a fair comparison of fuel types) and facilities that weren't generating any energy. We also excluded facilities that aren't primarily making electricity. This was done to ensure that we can make comparisons in terms of pollution per amount of electricity produced without overestimating pollution levels by failing to account for significant amounts of energy produced in the form of steam heat instead of electricity.

CO2 emissions from incinerators and coal plants have not changed much over time. In 2012, trash incinerators also emitted 65% more CO2 than coal plants.[15][17]

CEMS technology tracks the total CO2 coming out of the smokestack and doesn't discriminate between the biogenic and anthropogenic fractions of carbon dioxide emissions.

The industry argues that the "biogenic" portion of CO2 emissions (that from burning paper and other organic material) should not count because trees will regrow and take the CO2 back out of the air.[19] However, studies of the alleged "carbon neutrality" of biomass incineration have shown that biomass is not truly carbon neutral, as it can take many decades for trees to reabsorb the pulse of CO2 emitted by incineration – meanwhile, the climate is heating up at a higher rate.[20] This also presumes that somewhere, trees are being replanted in sufficient numbers to eventually take up this extra carbon pollution (and that those trees aren't being counted toward offsetting some other climate damage… and that the trees will not be cut back down as soon as it's profitable to use them). Trash incinerators are not causing any additional tree and plant growth, so in comparison to landfills or to other energy producers, no credit deserves to be applied to zero out biogenic carbon emissions.

Comparision of pollutants with the strongest data

Using 2018 interim NEI data[5], we conducted a robust analysis of over 73 pollutants tracked by NEI that were emitted and tracked across many different types of power plants (biomass burners, gas plants, oil plants, etc.). From there, we eliminated pollutants that had fewer than 25 facilities reporting emissions (since an outlier could significantly change data) and pollutants based on modeled data. Using reliability scores (with a reliability score of 1 representing a verified measurement and a score of 5 representing the lowest data quality) from EPA's peer-reviewed StEWI software[21], we further restricted the pollutants to those with reliability scores of 1, 2, 3 ensuring that we were only comparing pollutants with verified measurements and calculations.

Pollutant	Coal emissions (lbs/MWh)	Coal Data Points	Incinerator emissions (lbs/MWh)	Incinerator Data Points	x times worse than coal
Lead	4.0E-05	186	2.5E-04	42	6.2
Mercury	7.8E-06	188	4.0E-05	41	5.2
Benzene	3.2E-04	159	1.1E-03	30	3.3
Nitrogen oxides	1.5E+00	220	4.9E+00	51	3.3
Toluene	7.1E-05	148	2.0E-04	30	2.8
Cadmium	1.0E-05	189	2.4E-05	40	2.4
Carbon Monoxide	7.0E-01	189	7.1E-01	42	1.0
PM2.5 Primary	3.1E-01	189	1.6E-01	42	0.5
Nickel	6.3E-05	188	2.5E-05	32	0.4
Sulfur dioxide	2.1E+00	220	7.6E-01	51	0.4

1. New York State Department of Environmental Conservation, "Matter of the Application of Covanta Energy Corporation for Inclusion of Energy from Waste Facilities as an Eligible Technology in the Main Tier of the Renewable Portfolio Standard Program. Case No. 03-E-0188," Aug. 19, 2011. [redacted]
2. "An Inventory of Sources and Environmental Releases of Dioxin-Like Compounds in the

United States for the Years 1987, 1995, and 2000," U.S. EPA, November 2006, Table ES-2. [redacted] The largest sources in 2000 are considered to be backyard burn barrels (498.5 grams), followed by medical waste incineration (378 g), sewage sludge applied to land and emissions from sludge incineration (89.7 g) and trash incineration (83.8 g). The backyard burn barrel estimate is not subject to drastic differences based on test methods. 95% of medical waste incinerators have closed between 2000 and 2009. Most of the sewage sludge dioxin emissions are from land application rather than sludge incineration (since far more is dumped on farm fields than is burned). EPA admits in their inventory report (p 3-23): “Because all tests were conducted under normal operating conditions, some uncertainty exists about the magnitude of emissions that may have occurred during other conditions (e.g., upset conditions, start up, and shut down).” If the medical waste, sludge incineration and trash incineration numbers are adjusted upwards by 30-50 times to account for the fact that a 6-hour stack test each year underestimates dioxin emissions by this amount, compared to using continuous monitoring (and if the aforementioned differences in the medical waste and sludge incineration data are accounted for), trash incineration would still be the largest dioxin pollution source by far. A newer study from the Netherlands (data from 2015 to 2017) found that actual dioxin emissions are 460-1,290 times higher than stack tests indicate. See Arkenbout, A, Olie K, Esbensen, KH. "Emission regimes of POPs of a Dutch incinerator: regulated, measured and hidden issues" [redacted]

3. U.S. Environmental Protection Agency, Emissions & Generation Resource Integrated Database, eGRID2002 and eGRID2007 (for 2000 and 2005 electric generation data). [redacted]
4. “Emissions from Large and Small MWC Units at MACT Compliance,” U.S. Environmental Protection Agency memorandum, August 10, 2007. [redacted]
5. United States Environmental Protection Agency (EPA). 2018. "National Emissions Inventory (NEI)" Interim 2018 data accessed from the Emissions Inventory System (EIS)

European Federation for Transport and Environment AISBL

Diesel machines

Learn more about how diesel machines – like generators, construction equipment and barges – can harm our air quality and what can be done to stop it.

In this section

What problems do diesel machines cause for air quality?

European air pollution rules for off-road diesel machines such as bulldozers, excavators and barges are much more lax than those for cars and lorries. As well as this, some engine types (for example, diesel locomotives) and older machines are excluded from air quality laws. This is a problem because, according to the World Health Organisation (WHO), diesel exhaust is carcinogenic. Diesel machines account for 12% of nitrogen dioxide (NOx) emissions and 15% of fine particles from land-based sources but their importance is increasing as emissions from other land sources are reduced due to more stringent emissions legislation.

In 2016 Europe updated the relevant legislation but, despite making some improvements, lawmakers failed to harmonise the standards with those for lorry engines – currently the most closely comparable road vehicle in terms of emissions and engine configuration. In order to substantially reduce emissions that harm both human health and the environment, the EU should act soon.

Why is particulate matter a problem for diesel machines?

Particulate matter (PM) is the general term used to describe a mixture of suspended particles in the air. They are classified according to their diameter. Ultrafine particles are the most dangerous as they can penetrate deep into the lungs, enter the bloodstream and even reach the brain.

Historically, Europe has measured and regulated PM by the total weight. Unsurprisingly, this approach has led road-vehicle and diesel machinery manufacturers to focus on reducing the bigger and heavier PM while ignoring the smaller and more dangerous ones. In recent years, Europe has also started to regulate the number of particulates, known as particulate matter number (PN). Regulating PM and PN ensures that both large and small particles are cut.

In the latest reform, Europe has finally applied this new parameter to most engines for diesel machines, requiring them to fit particulate traps after 2020. However, some important categories, such as diesel locomotives used for freight operations, are exempt from the new rules. Ultrafine particles are therefore still a major problem for those trains, still used regularly in many parts of Europe.

Why is NOx a problem in diesel machinery?

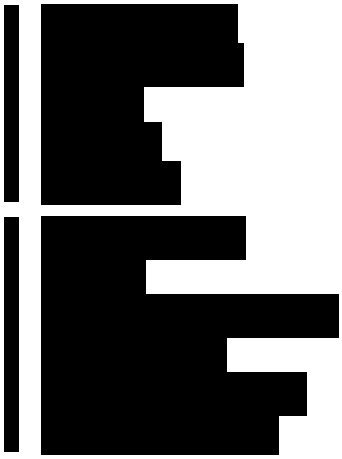
For some diesel machinery, nitrogen oxide (NOx) emissions limits are much higher than those for the equivalent road vehicles. Road vehicles are now equipped with catalysts, which treat and reduce these emissions in the exhaust gas. In contrast, some road machinery and barge standards for NOx are so lax they can be met without any exhaust after-treatment.

The latest reform failed to significantly improve NOx standards for new diesel machines, leaving this to a future review in 2023. This is a missed opportunity as technology to cut such pollution is available and already widely used in other applications such as trucks and buses.

What should Europe do about the impact of diesel machines on air quality?

Future legislation concerning diesel machines should:

- Align, wherever possible, standards for diesel machines to those for road vehicles. In particular, this means bringing down the currently weak NOx standards in line with best practice and WHO air quality guidelines.
- Enlarge the scope of the legislation to cover excluded machines: diesel machines with engines above 560 kW and below 19kW and diesel locomotives.
- Address emissions from existing machines. Diesel machines have a long lifetime and, without retrofitting of catalysts, will continue to pollute for a long time.
- Exemptions and flexibilities should be cut drastically so that it is impossible to sell machinery-equipped engines which comply with an old standard after a limited time from when the standard enters force.
- Finalise, no later than 2017, new provision on real-world testing of diesel machines (such as in-service surveillance) to make sure that these machinery meet air pollution standards when operated in real life (not only in lab testing).
- Transparency should be ensured by mandatory publication of an engine's emissions performance in a publicly available European database.
-

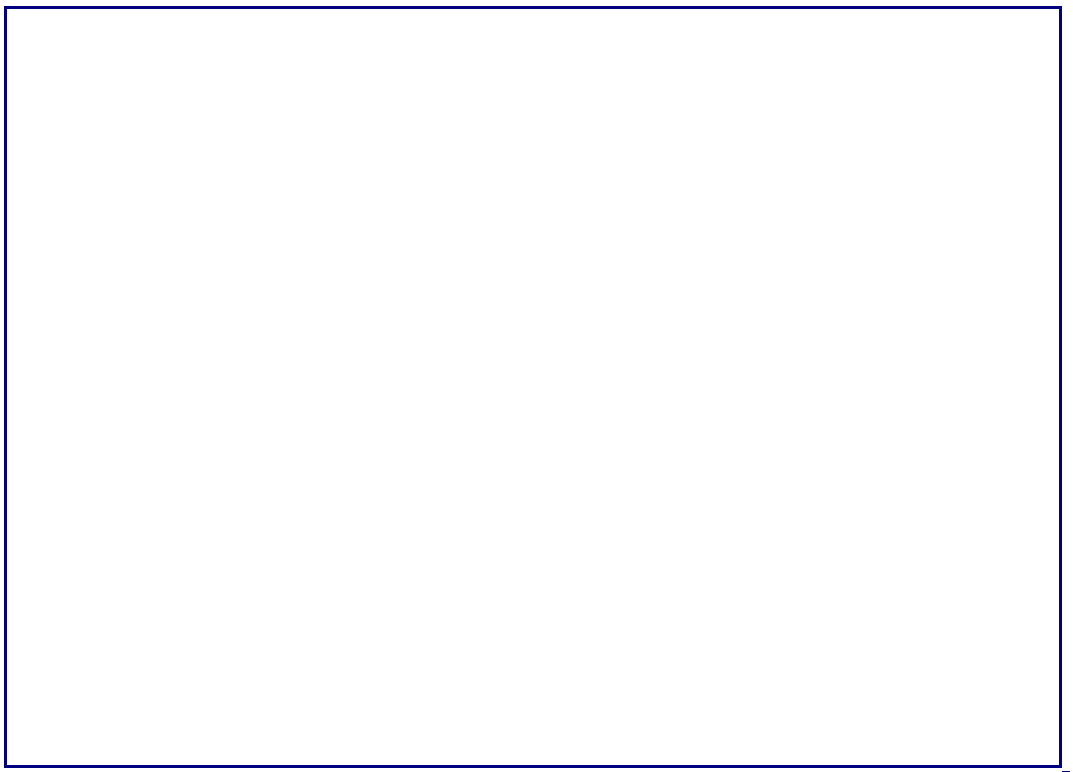


EU Transparency Register number 58744833263-19

Web design for nonprofit organisations by Fat Beehive

The United Kingdom Without Incineration Network (UKWIN)

-



-

Why Oppose Incineration

- United Kingdom Without Incineration Network
- Why Oppose Incineration

There are many reasons why people oppose incineration, including because:

- Incineration harms recycling
- Incineration exacerbates climate change
- Incineration is a barrier to the circular economy
- The UK already faces incineration overcapacity
- Incinerators harms air quality
- Incinerators are bad neighbours

Alternatives to incineration are less costly, more flexible, quicker to implement and better for the environment. Rather than incinerating waste, local authorities should focus on maximising re-use and recycling alongside providing a weekly separate food waste collection for treatment by composting or anaerobic digestion (AD). See the 'promoting alternatives' section of UKWIN's 'Act Locally' page for advice on how councils can avoid relying on incineration.

Incineration harms recycling

For councils with above-average rates of incineration the is a clear correlation between higher rates of incineration and lower recycling rates. Data source: Defra.

Studies indicate that most of what is currently in the 'residual' waste stream is readily recyclable, meaning a significant proportion of what is currently incinerated could have been recycled or composted (Source). A Defra report published in August 2020 stated that: "*Of total residual waste from household sources in England in 2017, an estimated 53% could be categorised as readily recyclable, 27% as potentially recyclable, 12% as potentially substitutable and 8% as difficult to either recycle or substitute*" (Source).

If incinerators limited their feedstock to genuinely residual waste then it would free up more than half of their current capacity, undermining the rationale for building new incinerators in the UK.

Many councils are signed up to long-term waste contracts that involve incineration. These contracts usually ensure that the council takes on the primary risk of the incinerator not getting enough waste to burn, meaning councils are in effect penalised for not sending enough waste for incineration. Incinerators cost around £200m+ to build and that money cannot then be spent on recycling (Source, Source).

Contractual mechanisms such as 'minimum tonnage guarantees', 'put-or-pay' clauses and 'banding mechanisms' undermine the economic incentive to reduce, re-use and recycle even where funds are available (Source, Source, Source).

There is a correlation between high rates of incineration and low rates of recycling (Source, Source, Source, Source). Many councils have told the Government that their low recycling rates are due to their incineration-based waste contracts that undermine their incentive or ability to invest in improvements to their recycling service (Source, Source).

The Government has a target for England to achieve 65% recycling for municipal solid waste by 2035 and no more than 10% landfill (Source). As some residual waste is not combustible, the Government's 65% recycling target implies that the rate of incineration should be no higher than a maximum of around 30%. However, in 2019/20, 45.5% of England's local authority collected waste was incinerated (Source).

> Investing in more EfW can negatively affect long term recycling rates. This investment needs to be paid for by an assured income stream, usually through contracts with local authorities to pay the EfW operator to take waste. Contracts are often lengthy – the majority are over 20 years. The terms of contracts, such as minimum annual payments,

> or a low fee per tonne of waste, can undermine the financial viability for the local authority of reducing waste, or sending it to other destinations such as recycling.
>
> *— London Assembly Environment Committee Report: 'Waste: Energy from Waste' (February 2018)*

Incineration exacerbates climate change

Incineration releases significantly more CO2 for every kWh exported to the electricity grid than the conventional use of fossil fuels, with the incineration of plastics being worse than coal (Source, Source)

According to the Committee on Climate Change: "*Achieving significant emission reductions in the waste sector requires a step-change towards a circular economy, moving away from landfill and incineration (and the associated methane and fossil CO□ emissions), and towards a reduction in waste arisings and collection of separated valuable resources for re-use and recycling*" (Source)

Incineration results in high levels of greenhouse gas emissions. For every tonne of waste burned, typically around one tonne of CO□ is released into the atmosphere, and around half of this is fossil CO□ (Source). This means that incineration has a higher carbon intensity than the conventional use of fossil fuels, and significantly higher than what most people would consider 'low carbon'.

Research on the real world performance information of English incinerators found that they often performed significantly worse than was predicted at the planning and permitting stage with the fossil carbon intensity of electricity exported to the grid being around 49% higher than predicted for the plant's studied (Source).

In 2020 the UK's 55 incinerators released a combined total of around 14m tonnes of CO□e, around 6.4 million tonnes of which were from fossil sources such as plastic (Source). The 6.4 million tonnes of fossil CO□ released by UK incinerators in 2020 resulted in an unpaid cost to society of more than £1.5 billion based on the UK Government's central abatement cost (Source).

Even when methane generation from the landfill of biogenic material is taken into account, over its lifetime a typical waste incinerator built in 2020 is estimated to release the equivalent of around 1.6 million tonnes of CO□ more than sending the same waste to landfill (Source).

Around half of the biogenic material sent to landfill does not rot down and therefore does not exacerbate climate change, whereas were the same waste to otherwise be incinerated then all of the biogenic carbon in the waste would converted into CO□ and released into the atmosphere (Source,

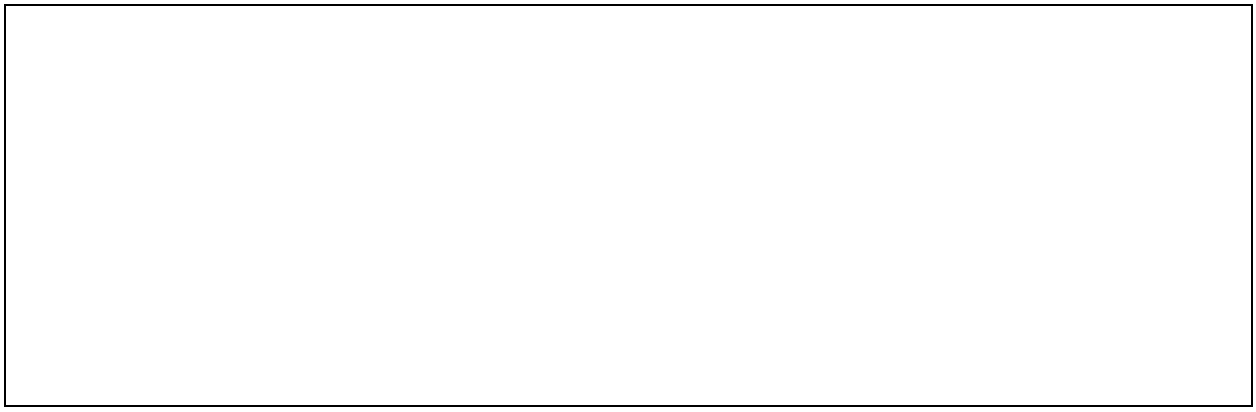
Source, Source, Source). Biostabilisation can be used to significantly reduce the production of methane from landfilled waste. Whilst incineration performs poorly against sending waste to landfill, it performs even worse when compared with sending waste that has been biostabilised to landfill (Source, Source, Source, Source).

Whatever the relative direct emissions are from incineration and landfill, the high cost of building incinerators and their for feedstock create a unique barrier to the environmentally preferable options of reduction, re-use and recycling.

Composition analysis indicates that much of what is currently used as incinerator feedstock could be recycled or composted (see recycling section above), and recycling what would otherwise be incinerated would result in significant carbon savings and other environmental benefits. Thus, incinerating waste comes with a significant 'opportunity cost' that has a significant adverse climate change impact (Source, Source).

> Decarbonisation of the grid has been so successful that EfW technologies can no longer be considered low carbon solutions. Decisions on future management must be based on the most current and accurate data possible to ensure climate change impacts are minimised.
>
> — **The climate change impacts of burning municipal waste in Scotland (technical report) (Zero Waste Scotland, October 2020)**

Incineration is a barrier to the Circular Economy



Moving away from incineration is a key element in moving towards a more circular economy because, instead of being destroyed, materials and nutrients can remain available thereby avoiding the additional extraction of finite resources

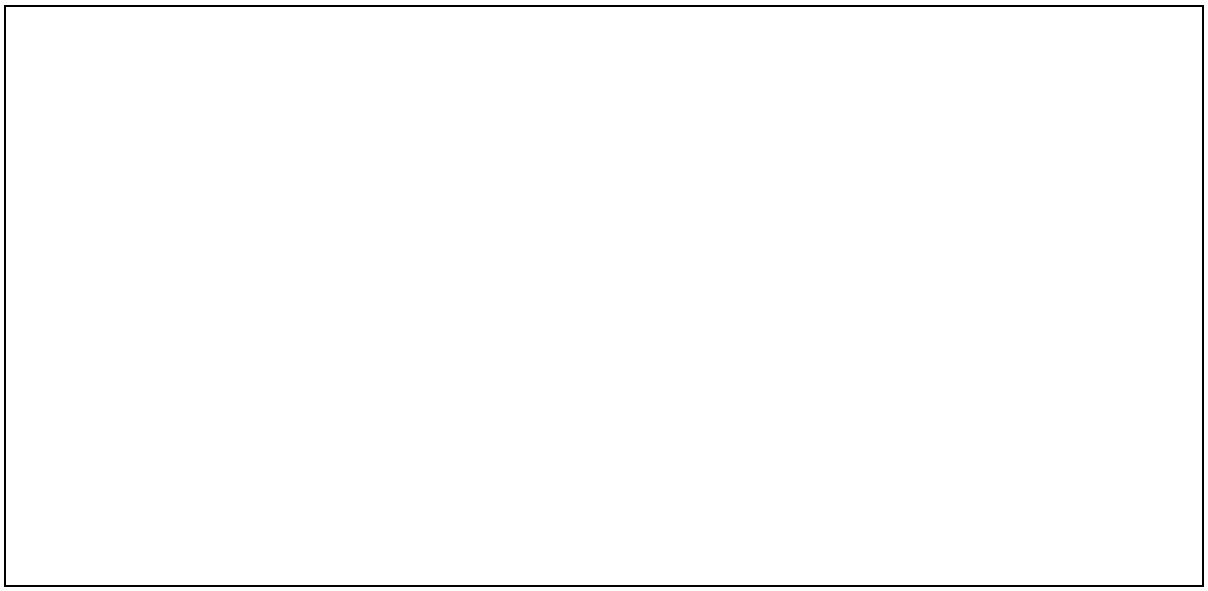
The 'linear economy' relies on extraction and processing, followed by consumption and disposal (via incineration or landfill). Extraction and disposal deplete finite resources and cause environmental and social harm. With a circular economy the value of resources is preserved, material and nutrients that are needed to create new products are maintained, and the most is made of existing resources. (Source)

Incineration has no place in the circular economy towards which we should be working. Incineration waste finite resource, squanders nutrients vital for the health of our soil, and is recognised as a 'leakage' to be minimised (Source, Source, Source). Products currently being incinerated should be treated at a higher tier of the Waste Hierarchy, and where that is not possible they need to be 'designed out'.

> "One of the central pillars of a circular economy is feeding materials back into the economy and avoiding waste being sent to landfill or incinerated, thereby capturing the value of the materials as far as possible and reducing losses"

— Circular economy in Europe – Developing the knowledge base. European Environment Agency, January 2016.

The UK already faces incineration overcapacity



Residual treatment overcapacity in September 2022 (see overcapacity page)

The 'Incinerator Feedstock' section of the chart (above) shows waste volumes are around 25.4Mt in 2022, and that these are expected to fall by 12Mt by 2042 to 13.4Mt. The 'Incineration Capacity' section shows that we do not expect to have enough feedstock to make use of the 18.9Mt of incineration capacity that is already operational and under construction, let alone to feed the 12.8Mt of proposed additional capacity that has yet to enter construction.

The chart above also shows that for a moratorium to capture a significant proportion of the potential overcapacity in the pipeline it must include not only the 4.1Mt of capacity which has yet to obtain planning permission but also the 5.7Mt of capacity which has planning permission but has yet to be granted an Environmental Permit by the Environment Agency.

The UK currently has more incineration capacity existing and under construction than genuinely residual waste to burn, and there are many more incinerator projects in the pipeline.

Incineration overcapacity harms the markets for recycling and reduces the marginal benefits of waste minimisation and re-use schemes, causing significant environmental harm. Locking our valuable materials into incineration creates a serious long-term risk to UK resource security and is a huge waste of money.

Even those who believe that incineration is a good way to treat genuinely residual waste should oppose the construction of new incinerators because they exacerbate long-term overcapacity. This is also a reason why the prospect of 'alternatives' to conventional incineration such as gasification and pyrolysis are a distraction from the need to invest in the higher tiers of the waste hierarchy.

The majority of the material that is currently incinerated is readily recyclable, with a 2020 Defra report stating that: *"Of approximately 13.1 million tonnes of residual waste generated by household sources in England in 2017, around 7 million tonnes could be categorised as readily recyclable, 3.5 million tonnes as potentially recyclable, 1.6 million tonnes as potentially substitutable, and 1.0 million tonnes as difficult to recycle or substitute"* (Source, Source). As more of this material is recycled, and as non-recyclable products are increasingly phased out, more and more capacity at existing incinerators will become available.

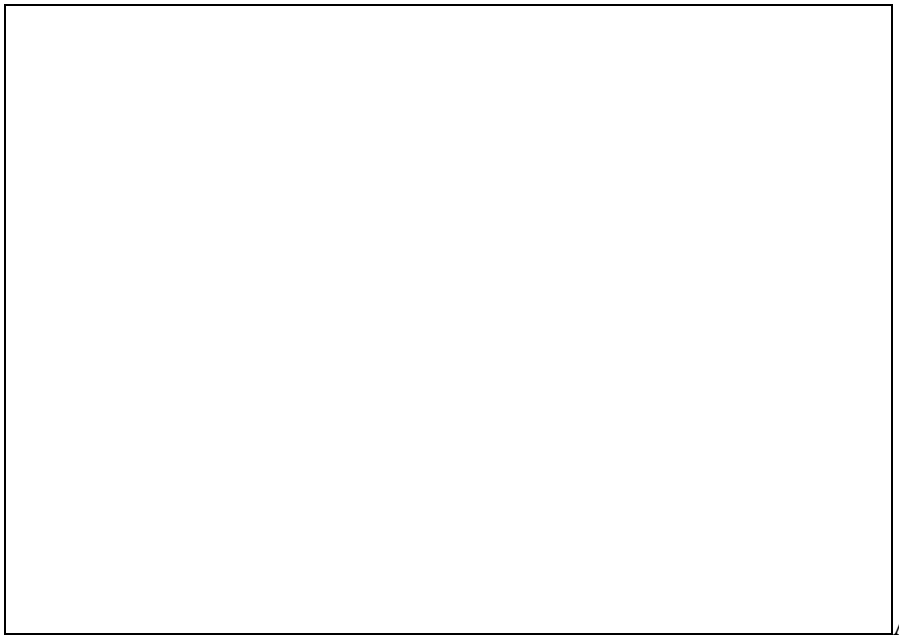
Furthermore, the move away from single-use plastics is increasing the treatment capacity of existing

incinerators, and many existing incinerator permits have been varied to increase the quantity of feedstock they are permitted to burn (Source).

There is widespread acknowledgement across Europe that those countries which pursued incineration with the most vigour, such as Germany, Denmark, and the Netherlands, are now facing incineration overcapacity which is harming recycling (Source, Source, Source, Source). It is vital that we learn from their mistakes, not repeat them.

> It would be wise to limit development of new thermal treatment capacity to that required once any targets have been met to avoid creating overcapacity as recycling increases.
>
> — *Waste markets study (Eunomia report for the Scottish Government, 23 April 2019)*

Incinerators harm air quality


Artistic impression of incinerator emissions, many of which are invisible to the human eye (Credit: F. Howe 2016)

In addition to greenhouse gas emissions that exacerbate climate change, incinerators emit many toxins and pollutants that harm local air quality. Emissions include dioxins, NOx and ultrafine particulate matter that can be harmful to both human health and the natural environment. There is not enough monitoring, not enough enforcement, and not enough transparency.

> "There is no safe level for particulate matter (PM10, PM2.5), while NO2 is associated with adverse health effects at concentrations at and below the legal limits."
>
> — **Air Quality: A Briefing for Directors of Public Health. Defra, Public Health England and the Local Government Association, March 2017.**

Incinerators are bad neighbours

Communities living near incinerators have many complaints that arise during construction, pre-operational testing (commissioning) and full operation, including:

- **Noise, vibration, plume, flies and odours** – These disamenities are often downplayed by operators during the planning and permitting application stages, however when problems do

occur some of these same operators dismiss the problems as inevitable or unavoidable. Press coverage reflecting some of these problems with incinerators include:

- In *Runcorn*, where waste is delivered by rail, it was reported that: *"one resident said she faced daily noise from cargo trains en route to deliver the waste to be burned, well into the evening"* and that: *"It's unbelievable – you can lie in bed at night and feel the vibration of the train as it goes past but it goes that slow it takes about two to three minutes to come past through the station."* (Source)
- It was also reported in *Runcorn* that: *"Around 100 people attended a meeting…to protest over the noise, smell, steam and pollution from the plant."* quoting one resident saying: *"I've been awake most of the night and I'm losing the will to live. Then wagons beeping their horns this morning followed by banging of containers*". The organiser of the meeting is quoted as stating: *"People feel trapped. It's gone from a place where they could sit in their garden to closing doors and windows because it stinks"*. This report also quoted the local MP as follows: *"People have been complaining about a droning noise disturbing their sleep. These are genuine concerns about the vapour, noise and smells."* (Source)
- In *Derby*, one resident stated: *"Where we are, the stench is really strong and smells like rotting food. We have been getting loads of flies around here as well. The summer has been horrendous, we have had to keep our windows closed in the hot weather because when we open them it is just awful."* (Source). It was also reported that: *"Bad smells from the controversial Sinfin waste treatment plant are still plaguing residents almost a year after the stink first started. Last August, residents and businesses near to the plant complained to the Environment Agency about a compost-like smell shortly after waste arrived for pre-opening commissioning. They were told the smell would disappear and was due to waste being stored on the site ahead of testing. But the smell has continued to plague residents – especially during the recent warmer weather – despite earlier promises from the operators that there would be no smell off-site from the facility"* (Source).
- In *Derby*, the operator stated: *"we acknowledge…that some nuisance has been caused especially overnight when background noise levels are lower, and the warm weather leaves residents understandably wishing to have windows open"* (Source).
- In *Gloucestershire*, the operator stated in relation to hot commissioning that: *"During this period, up until the facility is fully operational in summer 2019, there will be occasional loud noises, which sound similar to when you bleed a radiator, and plumes of steam as the first combustion gases are pushed through the ducting to test all systems"* (Source).
- An incinerator in *Plymouth* has also generated numerous complaints from local residents, with one commenting to the Plymouth Herald that: *"The summer was awful, all the flies, the rubbish, the smell. I am looking to move because we have had enough of it"*, and another stating: *"It smells, it makes me feel sick"*. According to an ITV report: *"Residents nearby have complained about the smell, the noise and flies in their homes. They say their worst fears have been realised"* (Source). It was also reported that: *"A 'rotten smell' was frequently emitted when first constructed, and still occurs in the summer"* (Source).

- **Light pollution** – Bright lights are typically placed towards the top of the incinerator stack to reduce the risk of aircraft collision. This is a constant reminder of the incinerator and a source of distress to many residents. For example, it was reported in *Runcorn* that one resident: *"said she now lives with her curtains drawn at night to block the lights from the site, which include a pair of red lights like eyes peering from the top of the main chimney stack, from shining into her home and bedroom, having previously enjoyed looking out at the trees behind her home and the site"* (Source).
- **Visual impact of the chimney stack and building –** Incinerators are often seen as a blot on

the local landscape and a constant reminder of the pollution that they cause. For example, one local newspaper article about an incinerator in **North Yorkshire** described the *Allerton* plant as one which *"dominates the skyline of the main road to the North"* quoting a councillor as stating: *"A lot of people do feel it is a blot on the landscape, I'm astonished that it can be seen from so many places"* (Source).

- **Traffic** – In addition to increases in the general volume of traffic and the pollution that this brings, some of those living near incinerators have observed HGVs ignoring planning conditions designed to control adverse impacts. For example, lorries delivering feedstock sometimes travel along routes that are disallowed by planning conditions, despite assurances made at the planning application stage that this would not happen. In other instances, after planning permission is granted on the basis of strict controls over when and where the HGVs can travel, it is not unusual for operators to seek to change the arrangement to enable increases in the number of vehicles, extensions of the time these vehicles are permitted, and expansion of the routes that they are allowed to take. Such changes are often allowed under delegated powers without any community consultation, even in circumstances where the changes directly break promises made to the community about how traffic impacts will be strictly controlled.
- **Broken promises, misinformation and lack of transparency –** In addition to the broken promises referred to above in relation to disamenities, there are various other instances where operators behave differently to how they said they would during consultations or where operators have not acted with full candour. For example:
    - Operators routinely state that inverse pressure will be used in buildings to avoid noise and odour issues, with doors being mostly shut, but then too often the operators end up leaving doors open for operational reasons which results in disamenities to neighbours.
    - Areas have faced real-world reductions in recycling rates despite assurances that the incineration plant would only be used for "non-recyclable" waste. In some cases, this is a result reduced recycling services once the incinerator is in place.
    - Liaison groups set up with the stated purpose of engaging with the community are often not informed of forthcoming changes to planning permissions and environmental permits, e.g. proposals to increase capacity. Those who ask tough questions are often excluded from liaison groups, and applicants often use participation in the liaison group as evidence of 'community support' for the facility (even in circumstances where the operator promised that they would not do so). In many cases, liaison groups are given the promise of helping to design the proposal but end up having influence over the location, capacity and technology choices adopted by the operator.
    - Operators often try to give the impression that all emissions are continuously monitored when in most cases emissions of concern, such as dioxins, are only monitored a few times a year.
    - Even in cases where operators have carried out compositional analysis of what they are burning, they often do not publish this information and will not release it to the public when this information is requested.
- **Inadequate responses to complaints** – When communities face serious nuisance from an incinerator, residents who reach out to the operator are too often greeted with denials that the problems are caused by the incinerator. Even when the operator is subsequently found to be at fault, these operators rarely apologise for having denied the issues were their responsibility. It is extremely rare for an operator to provide any compensation for the nuisances that they cause.
- **Property values –** Whether or not the loss of property value is a material planning consideration, it is not unusual for houses prices to fall when there is a proposed or actual incinerator. There are numerous instances where residents have reported experiencing

difficulty selling their property due to the threat of an incinerator. Operators do not tend to compensate residents who have suffered financially as a result of incinerators or incinerator proposals.

- **Problems with district heating schemes** including:
    - *Outages*, where residents are left in the cold due with no heating or hot water, e.g. because of an unplanned incinerator shut-down.
    - *Costs*, where residents may be tied into paying above-market-rate prices for their heating. Residents often do not have alternative means of powering their heating system (e.g. they have no boiler), and they are contractually obliged to pay for the heating network.

BBC News 15th March 2005

Foul smells blamed on incinerator

Residents living near an incinerator have made new complaints about foul smells, two months after it re-opened.

The HLC centre at Crymlyn Burrows started trials in January

Trials at the £35m recycling and energy centre at Crymlyn Burrows near Neath started in January, 18 months after it was closed by a fire.

The Environment Agency has traced the smell to the plant and is working with the operator to eradicate it.

The company said recent smells were "very mild indeed" but campaigners want the facility shut down for good.

The plant was given the go-ahead to start operating in May 2002 despite a number of protests.

> **"It's been like shoving your head in wheelie bin - you could not open the windows or doors"**
> Mike Ryan

Last week its operator, HLC Waste Management Services Limited, was fined £4,000 and ordered to pay £4,000 costs after admitting breaching operating conditions which allowed odours to escape from the site prior to a major fire there in August 2003.

Since the blaze, the plant has been partly refurbished and partly re-built and a new senior management team appointed.

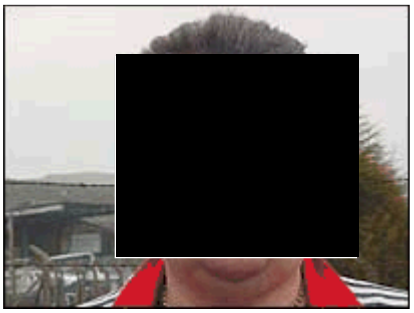
It began trials in January but people living nearby say since then the smells have returned.

Mike Ryan, who lives in Crymlyn Burrows and founded the Stop The Incinerator Campaign, said: "We've had terrible smells of rotting garbage coming into the village.

"It's been like shoving your head in wheelie bin - you could not open the windows or doors.

"It's obvious to me our objections at the start have been proved right.

"My quality of life and all the residents' quality of life has gone."



Mr Ryan said the smell from the plant was like rotting garbage

The Environment Agency confirmed it had received fresh complaints about the plant since it re-opened.

A spokesman said it had not taken any enforcement action but was working with HLC to resolve the problem.

When fully operational the plant would be capable of processing all domestic and non-hazardous commercial waste generated in the counties of Neath Port Talbot and Bridgend.

Seventy-five percent would be recycled, composted or burnt to produce power for the plant with the rest going to landfill.

A company spokesman said it was hoped the plant would be operating fully in the near future.

He said liaison meetings are held regularly with residents to give them a chance to discuss concerns and added he was aware of complaints by Mr Ryan and others.

But he said odours had emanated from the plant a few weeks ago but were 'very mild indeed' and the plant was operating strictly to Environment Agengy rules.

He added: "The trials have been very successful to date."

The Health Effects of Waste Incinerators

4th Report of the British Society for Ecological Medicine

Second Edition
June 2008

Moderators: Dr Jeremy Thompson and Dr Honor Anthony

Preface to Second Edition

Since the publication of this report, important new data has been published strengthening the evidence that fine particulate pollution plays an important role in both cardiovascular and cerebrovascular mortality (see section 3.1) and demonstrating that the danger is greater than previously realised. More data has also been released on the dangers to health of ultrafine particulates and about the risks of other pollutants released from incinerators (see section 3.4). With each publication the hazards of incineration are becoming more obvious and more difficult to ignore.

In the light of this data and the discussion provoked by our report, we have extended several sections. In particular, the section on alternative waste technologies (section 8) has been extensively revised and enlarged, as has that on the costs of

incineration (section 9), the problems of ash (9.4), radioactivity (section 9.5), and the sections on monitoring (section 11), and risk assessment (section 12).

We also highlight recent research which has demonstrated the very high releases of dioxin that arise during start-up and shut-down of incinerators (section 11). This is especially worrying as most assumptions about the safety of modern incinerators are based only on emissions which occur during standard operating conditions. Of equal concern is the likelihood that these dangerously high emissions will not be detected by present monitoring systems for dioxins.

Foreword to the 1st Edition

from **Professor C. V. Howard. MB. ChB. PhD. FRCPath.**

The authors are to be congratulated on producing this report. The reader will soon understand that to come to a comprehensive understanding of the health problems associated with incineration it is essential to become acquainted with a large number of different disciplines ranging from aerosol physics to endocrine disruption to long range transport of pollutants. In most medical schools, to this day, virtually nothing is routinely taught to equip the medical graduate to approach these problems. This has to change. We need the medical profession to be educated to health consequences associated with current environmental degredation.

There are no certainties in pinning specific health effects on incineration: the report makes that clear. However this is largely because of the complexity of exposure of the human race to many influences. The fact that 'proof' of cause and effect are hard to come by is the main defence used by those who prefer the *status quo*. However the weight of evidence, collected within this report, is sufficient in the authors' opinion to call for the phasing out of incineration as a way of dealing with our waste. I agree with that.

There is also the question of sustainability. Waste destroyed in an incinerator will be replaced. That involves new raw materials, manufacture, transport, packaging etc etc. In contrast, reduction, reuse and recycling represent a win-win strategy. It has been shown in a number of different cities that high levels of diversion of waste (>60%) can be achieved relatively quickly. When that happens, there is not very much left to burn, but a number of the products left will be problematic, for example PVC. Incineration, an end of pipe approach, sends the message 'No problem, we have a solution for disposal of your product, carry on business as usual'. What should happen is a 'front end solution'. Society should be able to say 'Your product is unsustainable and a health hazard – stop making it".

Incineration destroys accountability and this encourages industries to go on making products that lead to problematic toxic wastes. Once the waste has been reduced to ash who can say who made what? The past 150 years has seen a progressive 'toxification' of the waste stream with heavy metals, radionuclides and synthetic halogenated organic molecules. It is time to start reversing that trend. We won't achieve that while we continue to incinerate waste.

Vyvyan Howard December 2005

Professor of Bioimaging, Centre for Molecular Biosciences,
University of Ulster, Cromore Road, Coleraine, Co. Londonderry BT52 1SA

Contents

Executive Summary

- Large studies have shown higher rates of adult and childhood cancer and also birth defects around municipal waste incinerators: the results are consistent with the associations being causal. A number of smaller epidemiological studies support this interpretation and suggest that the range of illnesses produced by incinerators may be much wider.
- Incinerator emissions are a major source of fine particulates, of toxic metals and of more than 200 organic chemicals, including known carcinogens, mutagens, and hormone disrupters. Emissions also contain other unidentified compounds whose potential for harm is as yet unknown, as was once the case with dioxins. Since the nature of waste is continually changing, so is the chemical nature of the incinerator emissions and therefore the potential for adverse health effects.
- Present safety measures are designed to avoid acute toxic effects in the immediate neighbourhood, but ignore the fact that many of the pollutants bioaccumulate, enter the food chain and can cause chronic illnesses over time and over a much wider geographical area. No official attempts have been made to assess the effects of emissions on long-term health.
- Incinerators produce bottom and fly ash which amount to *30-50% by volume of the original waste* (if compacted), and require transportation to landfill sites. Abatement equipment in modern incinerators merely transfers the toxic load, notably that of dioxins and heavy metals, from airborne emissions to the fly ash. This fly ash is light, readily windborne and mostly of low particle size. It represents a considerable and poorly understood health hazard.
- Two large cohort studies in America have shown that fine ($PM_{2.5}$) particulate air pollution causes increases in all-cause mortality, cardiovascular mortality and mortality from lung cancer, after adjustment for other factors. A more recent, well-designed study of morbidity and mortality in postmenopausal women has confirmed this, showing a 76% increase in cardiovascular and 83% increase in cerebrovascular mortality in women exposed to higher levels of fine particulates. These fine particulates are primarily produced by combustion processes and are emitted in large quantities by incinerators.
- Higher levels of fine particulates have been associated with an increased prevalence of asthma and COPD.
- Fine particulates formed in incinerators in the presence of toxic metals and organic toxins (including those known to be carcinogens), adsorb these pollutants and carry them into the blood stream and into the cells of the body.
- Toxic metals accumulate in the body and have been implicated in a range of emotional and behavioural problems in children including autism, dyslexia, attention deficit and hyperactivity disorder (ADHD), learning difficulties, and delinquency, and in problems in adults including violence, dementia, depression and Parkinson's disease. Increased rates of autism and learning disabilities have been noted to occur around sites that release mercury into the environment. Toxic metals are universally present in incinerator emissions and present in high concentrations in the fly ash.
- Susceptibility to chemical pollutants varies, depending on genetic and acquired factors, with the maximum impact being on the foetus. Acute

exposure can lead to sensitisation of some individuals, leaving them with life-long low dose chemical sensitivity.

- Few chemical combinations have been tested for toxicity, even though synergistic effects have been demonstrated in the majority of cases when this testing has been done. This synergy could greatly increase the toxicity of the pollutants emitted, but this danger has not been assessed.
- Both cancer and asthma have increased relentlessly along with industrialisation, and cancer rates have been shown to correlate geographically with both toxic waste treatment facilities and the presence of chemical industries, pointing to an urgent need to reduce our exposure.
- In the UK, some incinerators burn radioactive material producing radioactive particulates. Inhalation allows entry into the body of this radioactive material which can subsequently emit alpha or beta radiation. These types of radiation have low danger outside the body but are highly destructive within. No studies have been done to assess the danger to health of these radioactive emissions.
- Some chemical pollutants such as polyaromatic hydrocarbons (PAHs) and heavy metals are known to cause genetic changes. This represents not only a risk to present generations but to future generations.
- Monitoring of incinerators has been unsatisfactory in the lack of rigor, the infrequency of monitoring, the small number of compounds measured, the levels deemed acceptable, and the absence of biological monitoring. Approval of new installations has depended on modelling data, supposed to be scientific measures of safety, even though the method used has no more than a 30% accuracy of predicting pollutants levels correctly and ignores the important problems of secondary particulates and chemical interactions.
- It has been claimed that modern abatement procedures render the emissions from incinerators safe, but this is impossible to establish and would apply only to emissions generated under standard operating conditions. Of much more concern are non-standard operating conditions including start-up and shut-down when large volumes of pollutants are released within a short period of time. Two of the most hazardous emissions – fine particulates and heavy metals – are relatively resistant to removal.
- The safety of new incinerator installations cannot be established in advance and, although rigorous independent health monitoring might give rise to suspicions of adverse effects on the foetus and infant within a few years, this type of monitoring has not been put in place, and in the short term would not reach statistical significance for individual installations. Other effects, such as adult cancers, could be delayed for at least ten to twenty years. It would therefore be appropriate to apply the precautionary principle here.
- There are now alternative methods of dealing with waste which would avoid the main health hazards of incineration, would produce more energy and would be far cheaper in real terms, if the health costs were taken into account.
- Incinerators presently contravene basic human rights as stated by the United Nations Commission on Human Rights, in particular the Right to Life under the European Human Rights Convention, but also the Stockholm Convention and the Environmental Protection Act of 1990. The foetus, infant and child are most at risk from incinerator emissions: their rights are therefore being ignored and violated, which is not in keeping with the concept of a just

society. Nor is the present policy of locating incinerators in deprived areas where their health effects will be maximal: this needs urgent review.

- Reviewing the literature for the second edition has confirmed our earlier conclusions. Recent research, including that relating to fine and ultrafine particulates, the costs of incineration, together with research investigating non-standard emissions from incinerators, has demonstrated that the hazards of incineration are greater than previously realised. The accumulated evidence on the health risks of incinerators is simply too strong to ignore and their use cannot be justified now that better, cheaper and far less hazardous methods of waste disposal have become available. We therefore conclude that no more incinerators should be approved.

1. Introduction

Both the amount of waste and its potential toxicity are increasing. Available landfill sites are being used up and incineration is being seen increasingly as a solution to the waste problem. This report examines the literature concerning the health effects of incinerators.

Incinerators produce pollution in two ways. Firstly, they discharge hundreds of pollutants into the atmosphere. Although some attention has been paid to the *concentrations* of the major chemicals emitted in an effort to avoid acute local toxic effects, this is only part of the problem. Many of these chemicals are both toxic and bio-accumulative, building up over time in the body in an insidious fashion with the risk of chronic effects at much lower exposures. Little is known about the risks of many of these pollutants, particularly when combined. In addition, incinerators convert some of the waste into ash and some of this ash will contain high concentrations of toxic substances such as dioxins and heavy metals, creating a major pollution problem for future generations. Pollutants from landfill have already been shown to seep down and pollute water sources. It is also important to note that incineration does not solve the landfill problem because of the large volumes of the ash that are produced.

There have been relatively few studies of populations exposed to incinerator emissions or of occupational exposure to incinerators (see section 4), but most show higher-than-expected levels of cancer and birth defects in the local population and increased ischaemic heart disease has been reported in incinerator workers. These findings are disturbing but, taken alone, they might only serve to alert the scientific community to possible dangers but for two facts. The first is the acknowledged difficulty of establishing beyond question the chronic effects associated with any sort of environmental exposure. The second is the volume of evidence linking health effects with exposure to the individual combustion products known to be discharged by incinerators and other combustion processes.

The purpose of this report is to look at all the evidence and come to a balanced view about the future dangers that would be associated with the next generation of waste incinerators. There are good reasons for undertaking this review. The history of science shows that it often takes decades to identify the health effects of toxic exposures but, with hindsight, early warning signs were often present which had gone unheeded. It is rare for the effects of environmental exposures to have been anticipated in advance. For instance it was not anticipated that the older generation of

incinerators in the UK would prove to be a major source of contamination of the food supply with dioxins. In assessing the evidence we shall also look at data from a number of other areas which we believe to be relevant, including research on the increased vulnerability of the foetus to toxic exposures, and the risk of synergistic effects between chemicals, the higher risks to people more sensitive to chemical pollution, the difficulties of hazard assessment, the problems of monitoring and the health costs of incineration.

2. Emissions from Incinerators and other Combustion Sources

The exact composition of emissions from incinerators will vary with what waste is being burnt at any given time, the efficiency of the installation and the pollution control measures in place. A municipal waste incinerator will take in a great variety of waste contaminated by heavy metals and by man-made organic chemicals. During incineration more toxic forms of some of these substances can be created. The three most important constituents of the emissions, in terms of health effects, are particulates, heavy metals and combustion products of man-made chemicals; the latter two can be adsorbed onto the smaller particulates making them especially hazardous. The wide range of chemicals known to be products of combustion include sulphur dioxide, oxides of nitrogen, over a hundred volatile organic compounds (VOCs), dioxins, polyaromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs) and furans.

2.1 Particulates

Particulates are tiny particles in the air that are classified by size. PM_{10}s have a diameter of less than 10 microns whereas fine particulates ($PM_{2.5}$s) are less than 2.5 microns and ultrafine particulates ($PM_{0.1}$s) are less than 0.1 micron. Incinerators produce huge quantities of fine and ultrafine particulates. Incinerators are permitted to emit particulates at a rate of 10mg per cubic metre of gaseous discharge. The commonly-used baghouse filters act like a sieve, effectively allowing the smallest particulates to get through and blocking the less dangerous, larger particulates. Only 5-30% of the $PM_{2..5}$s will be removed by these filters and virtually none of the $PM_{0.1}$s. In fact the majority of particles emitted by incinerators are the most dangerous ultrafine particulates[1]. The baghouse filters are least effective at removing the smallest particles, especially those of 0.2 to 0.3 microns, and these will have a considerable health impact. Health effects are determined by the number and size of particles and not the weight. Measurements of the particle size distribution *by weight* will give a false impression of safety due to the higher weight of the larger particulates. Pollution abatement equipment, installed to reduce emissions of nitrogen oxides, may actually increase emissions of the $PM_{2.5}$ particulates[2]. The ammonia used in this process reacts with sulphurous acid formed when steam and sulphur dioxide combine as they travel up the stack, leading to the production of secondary particulates. These secondary particulates are formed beyond the filters and emitted unabated: they can easily double the total volume of particulates emitted[3]. Present modelling methods do not take secondary particulates into account (see section 12).

Studies have shown that toxic metals accumulate on the smallest particulates[3] and that 95% of polycyclic aromatic hydrocarbons (PAHs) are associated with fine

particulates (PM_3 and below) [5-7]. PAHs are toxic and carcinogenic, and it has been estimated that these increase the lung cancer risk by 7.8 times[8].

2.2 Heavy Metals

Incinerators are allowed to emit 10mg per cubic metre of particulates and 1mg per cubic metre of metals. The limits mean little as, even within these limits, the total amount of particulates and metals emitted will vary with the volume per second of emissions generated by the incinerator and this can vary hugely. A further concern is that there are no statutory ambient air quality standards for heavy metals apart from lead, which means the levels of heavy metals in the surrounding air do not need to be monitored.

The proportion of metals to particulates allowed to be emitted by incinerators is very high and much higher than found in emissions from cars. At the high temperatures found in incinerators metals are released from metallic waste, plastics and many other substances. Many of the heavy metals emitted, such as cadmium, are toxic at very low concentrations. The selective attachment of heavy metals to the smallest particulates emitted from incinerators[4] increases the toxicity of these particulates. This fact is likely to make the particulates from incinerators more dangerous than particulates from other sources such as from cars.

2.3 Nitrogen Oxides

Removal of nitric oxide by incinerators is only about 60% effective and the nitric oxide is then converted to nitrogen dioxide to form smog and acid rain. Sunlight acts on nitrous oxides and volatile organic compounds (VOCs) to produce another pollutant, ozone.

2.4 Organic Pollutants

A wide range of organic pollutants are emitted from incinerators. These include PAHs (polycyclic aromatic hydrocarbons), PCBs (polychlorinated biphenyls), dioxins, furans, phthalates, ketones, aldehydes, organic acids and alkenes.

The waste being burnt now differs considerably from that burnt in the past with a higher load of heavy metals and plastics producing far greater potential for health and environmental problems. An example of this is PVC which is more than 90% organic chlorine. It has been used extensively for doors and windows and with an expected life of 40 years it is likely to appear in increasing quantities in the waste stream. This could easily raise the organic chlorine in the waste stream to over 1%, which according to the European Waste Directive would mean the waste would be regarded as hazardous.

Many of the compounds are known to be not only toxic but bio-accumulative and persistent. They include compounds that have been reported to affect the immune system[9], attach to chromosomes[10], disrupt hormone regulation[11], trigger cancer[12], alter behaviour[13], and lower intelligence[14]. The very limited toxicity data on many of these substances is a matter of concern[15]. The changing nature of waste means new substances are likely to be emitted and created. For example polybrominated diphenyl ethers (PBDEs) are found in many electrical goods and are increasingly finding their way into incinerator waste. They have been found to affect brain development and affect the thyroid gland and cause behavioural and learning defects in animals[16,17].

3. Health Effects of Pollutants

3.1 Particulates

A large and growing body of literature has highlighted the dangers of particulates to health. Various studies have confirmed that *the smaller the size of the particles the more dangerous the health effects*[18-21]. The data from the World Health Organisation shown in the graph below clearly illustrates that $PM_{2.5}$ particles have a greater effect on daily mortality than the larger PM_{10}s[18].

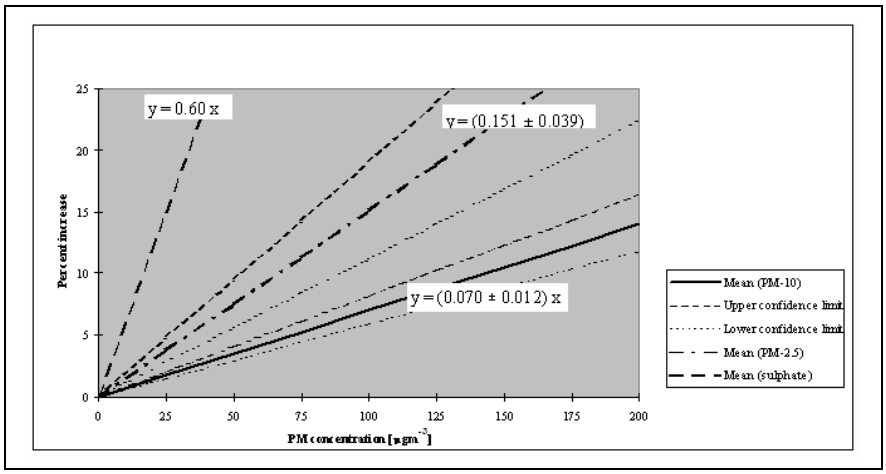


Figure 1. Increase in daily mortality as a function of PM concentration.
(reproduced from ref 18, Figure 3.6)

The smaller particles are not filtered out by the nose and bronchioles and their miniscule size allows them to be breathed deeply into the lungs and to be absorbed directly into the blood stream where they can persist for hours[22]. They can then travel through the cell walls and into the cell nucleus affecting the cell's DNA. The WHO state that there is no safe level of $PM_{2.5}$[18]and health effects have been observed at surprisingly low concentrations with no threshold[23,24]. The smallest particulates, particularly the ultrafine particulates ($PM_{0.1}$) are highly chemically reactive, a property of their small size and large surface area[25]. A further danger of the smallest particulates is that there are thousands more of them per unit weight. In incinerators heavy metals, dioxins and other chemicals can adhere to their surface[26] increasing their toxicity. The body does not have efficient mechanisms for clearing the deeper part of the lung as only a tiny fraction of natural particles will be as small as this.

As incinerators are effectively particulate generators and produce predominately the smaller particulates that have the biggest effect on mortality it is clear that incinerators have considerable lethal potential.

a) Epidemiological Studies of Particulate Pollutants

Fine particulates have been associated with both respiratory and cardiovascular disease[27] and with lung cancer[19,28].

Two large cohort studies in the USA showed increasing mortality with increasing levels of $PM_{2.5}$ pollution. In the Six City Study published in 1993[19] , 8,111 individuals were followed for 14-16 years (1974-1991), involving a total of 111,076 person years, to examine the effect of air pollution, allowing for smoking and other individual factors. As expected, the greatest risk factor was smoking (adjusted mortality-rate ratio 1.59) but, after allowing for individual factors, mortality rates showed highly significant associations ($p<0.005$) with the levels of fine particles and sulphate particles in the cities, with the most polluted city giving an adjusted all-cause mortality rate of 1.26 compared to the least. This related to a $PM_{2.5}$ difference of 18.6µg per cubic metre: cardiopulmonary mortality was increased by 37% and lung cancer mortality was also 37% higher.

In the American Cancer Society study[20], 552,138 adults (drawn from the Cancer Prevention II study) were followed from 1982 to 1989 and deaths analysed against mean concentrations of sulphate air pollution in 1980 and the median fine particulate concentration from 1979-1983, both obtained for each participant's area of residence from Environmental Protection Agency (EPA) data. Again, the strongest correlation was between lung cancer and smoking (adjusted mortality risk ratio 9.73), but both pollution measures showed highly significant association with all-cause mortality and with cardiopulmonary mortality: sulphates were also associated with lung cancer. After adjusting for smoking and other variables, higher fine particulate pollution was associated with a 17% increase in all-cause mortality and a 31% increase in cardiopulmonary mortality for a 24.5 µg per cubic metre difference in $PM_{2.5}$s. These results are highly significant and led the EPA to place regulatory limits on $PM_{2.5}$s, establishing the National Ambient Air Quality Standards in 1997. These regulations were challenged by industry but ultimately upheld by the US Supreme Court[29] after the data from all the studies had been subjected to intense scrutiny including an extensive independent audit and a re-analysis of the original data[30].

The health benefits of bringing in these new regulations have been estimated as $32 billion annually[31] based on mortality and chronic and acute health effects, and a White House report from the Office of Management and Budget in September 2003 calculated the benefits in terms of reductions in hospitalizations, premature deaths and lost working days as between $120 and $193 billion over the last 10 years (see section 9.1). As this study looked at only three health indicators it is likely to underestimate the true benefits.

It follows from this data that incinerators and all other major sources of $PM_{2.5}$ particulates will generate substantial health costs as well as increasing mortality.

b) Further Studies

An analysis published in 2002 of the Cancer Prevention II study participants linked the individual factors, pollution exposures and mortality data for approximately 500,000 adults as reported in the ACS study above, bringing the follow-up to 1998 [28]. The report doubled the follow-up period and reported triple the number of deaths, a wider range of individual factors and more pollution data, concentrating on fine particles. Smoking remained the strongest factor associated with mortality, but fine particulate pollution remained significantly associated with all-cause, and cardiopulmonary mortality with average adjusted RRs of 1.06 and 1.09. In addition, after the longer follow-up period, fine particulates were significantly associated with lung cancer mortality with an adjusted RR of 1.14. The authors reported that exposure to a 10µg per cubic metre higher level of $PM_{2.5}$s was associated with a 14% increase in lung cancer and a 9% increase in cardiopulmonary disease[28].

c) Cardiovascular Disease

Researchers were surprised to find that the increased cardiopulmonary mortality associated with particulate pollution was primarily due to cardiovascular disease. This was found in both the Six City and ACS studies when they were re-analysed[30]. When the causes of death in the Cancer Prevention II Study were looked at in more detail[32] to look for clues to possible pathophysiological mechanisms, the link was strongest with ischaemic heart disease: a 10µg per cubic metre increase in $PM_{2.5}$s was associated with an 18% increase in deaths from ischaemic heart disease (22% in never smokers). A more recent prospective study, the Women's Health Initiative (WHI), followed 65,893 postmenopausal women (initially free of cardiovascular disease) over 6 years, to examine the effects of the fine particulate pollution in the neighbourhood of each participant on the first cardiovascular or cerebrovascular incident and on mortality. The results for mortality and morbidity were consistent. Each increase of 10µg per cubic metre in fine particulate pollution was associated with a 76% increase in deaths from cardiovascular disease and an 83% increase in deaths from cerebrovascular disease[33]. The effect was independent of other variables but obese women and those who spent more time outdoors were more vulnerable to the effect. The WHI involved a more homogeneous study population and had a number of other methodological advantages over the earlier studies, resulting in greater sensitivity, and more reliable estimates. However, part of the greater effect in this study may be due to gender: there has been some evidence in other studies that women are more susceptible to the cardiovascular effects of fine particulates than men.

These results imply that the increase in fine particulate pollution associated with larger incinerators can be expected to increase mortality. It is probably safe to extrapolate from the WHI assuming that the effect on mortality in the WHI was genuine for women, and that the risk to men would be half as great. In that case, if the incinerator increased $PM_{2.5}$ particulates by as little as 1µg per cubic metre, cardiovascular mortality would be increased by 5-10%, with similarly increased cerebrovascular mortality.

Acute myocardial infarctions have been found to rise during episodes of high particulate pollution, doubling when levels of $PM_{2.5}$s were 20-25µg per cubic metre higher[34]. Particulates also increased mortality from stroke[35,36]. One study concluded that 11% of strokes could be attributed to outdoor air pollution[37]. Episodes of increased particulate pollution also increased admissions with heart disease[38]. A recent study found that each 10µg per cubic metre rise in PM_{10} particulates was associated with a 70% increase in DVT risk.[39] Mortality from diabetes[27] and admissions for diabetic heart disease are also increased[40] and these were double the non-diabetic CHD admissions, suggesting that diabetics were particularly vulnerable to the effect of particulate pollution[40]. Higher levels of particulates have been associated with life-threatening arrhythmias[41] exercise-induced ischaemia[42], excess mortality from heart failure[36,43] and thrombotic disease[36].

d) Effect on Children and the Foetus

Particulates carry various chemicals including polycyclic aromatic hydrocarbons (PAHs) into the human body. Frederica Perera from the Columbia Center for Children's Environmental Health has found that the foetus is 10 times

more vulnerable to damage by these substances[44]. She also found that $PM_{2.5}$ particulates have an adverse effect on the developing foetus with significant reductions in weight, length and head circumference and reiterated the importance of reducing ambient fine particulate concentrations[45]. In addition further studies have shown an adverse effect on foetal development at levels currently found in cities today, such as New York[46]. Air pollution has been found to cause irreversible genetic mutations in mice. Researchers found, in contrast, that if mice breathed air which had been freed of particulates by filtration they developed only background levels of genetic mutations, confirming that particulates were causative[47]. At the fourth Ministerial Conference of Environment and Health in June 2004, the WHO announced that between 1.8 and 6.4% of deaths in the age group from 0 to 4 could be attributed to air pollution[48].

e) Acute Respiratory Incidents

Elevated particulate air pollution has been associated with increased hospital admissions with asthma[24] and with COPD[49], increases in respiratory symptoms[50,51], higher incidence of asthma[52], reduced immunity[53,54], higher rates of ear, nose and throat infection[52], loss of time from school in children through respiratory disease[55,56], and declines of respiratory function[57-59]. A sad aside to the above is that children who did more outdoor sport had greater declines in respiratory function[59]. We are doing a great disservice to our children if they cannot pursue healthy activities without damaging their health.

f) Mortality from Particulate Pollution

Episodes of increased particulate pollution have been associated with increased cardiovascular mortality[19,20,27,28,36,43,60] and increased respiratory mortality[43,44]. About 150 time-series studies around the world have shown transient increases in mortality with increases in particulates. Cohort studies have shown a long-term effect on mortality[19,20,28] (see section 3.1a).

Can we quantify this mortality? It has been estimated that the increased mortality works out as about a 0.5-1% increase in mortality for each 10µg per cubic metre rise in PM_{10}s[61] for acute exposures and a 3.5% rise for chronic exposures[31]. For $PM_{2.5}$s the increase in mortality is much greater, especially for cardiopulmonary mortality (see Table).

Table 1 Cardiopulmonary Mortality and Fine Particulate Pollution

Study	Reference & Year	No of Participants	Follow up	Adjusted excess c/p mortality	Difference in $PM_{2.5}$s in µg/m^3	Adjusted excess c/p mortality for rise of 10µg/ m^3
Six Cities	[19] 1993	8,111	1974-1991	37%	18.6	19.8%
ACS Cancer Prevention II	[20] 1995	552,138	1982-1989	31%	24.5	12.7%
Cancer Prevention II	[28] 2002	500,000	1982-1998	9%	10	9%

Women' Health Initiative	[33] 2007	65,893	1994-2002	76%	10	76%

When the data from the Six Cities Study and the ACS study were subject to audit and re-analysis (see section 3.1a) the cardiopulmonary deaths were separated into pulmonary and cardiovascular[30]. Unexpectedly most of the excess deaths due to particulates had been from cardiovascular causes. This was apparent in each of the analyses performed giving figures for the increase in cardiovascular mortality in the Six Cities study of between 35% and 44% for an 18.6 µg per cubic metre difference in $PM_{2.5s}$ and in the ACS study between 33% and 47% for a 24.5µg per cubic metre. This was much higher in each case than the increase in respiratory deaths of 7%. In the ACS data it was later found that the excess cardiovascular deaths were primarily due to an 18% increase in deaths from ischaemic heart disease for each 10µg per cubic metre rise in $PM_{2.5}$s[32]. The Women's Health Initiative study has demonstrated an even stronger statistical relationship between raised levels of fine particulates and cardiovascular deaths with a 76% increase in cardiovascular mortality for each 10µg per cubic metre increase in $PM_{2.5}$ particulates, and this depended not just on which city a woman lived in but in which part of that city[33]. This study, more than any other, demonstrates the great dangers posed by fine particulates and the highlights the urgent need to remove major sources of these pollutants.

As incinerators selectively emit smaller particulates and cause a greater effect on levels of $PM_{2.5}$s than PM_{10}s, they would therefore be expected to have a significant impact on cardiopulmonary mortality, especially cardiovascular mortality. This has not so far been studied directly.

g) Studies Involving Ultrafine Particles

Ultrafine particles (0.1µg per cubic metre and below) are produced in great numbers by incinerators[1]. They have been less studied than $PM_{2.5}$ and PM_{10} particulates but there has been enough data available for the WHO to conclude that they produce health effects immediately, after a time lag and in association with cumulative exposure. They have been found to have a more marked effect on cardiovascular mortality than fine particulates, with a time lag of 4-5 days[62]. Stroke mortality has been positively associated with current and previous day levels of ultrafine particulates and this has occurred in an area of low pollution suggesting there may be no threshold for this effect[63]. Ultrafine particulates have also been reported to be more potent than other particulates on a per mass basis in inducing oxidative stress in cells[64] and they have the ability to cross the blood-brain barrier and lodge in brain tissue[65]. They represent another largely unknown and unexplored danger of incineration.

h) Assessment by the WHO and Other Authorities

Based on the World Health Organisation Air Quality Guidelines[66] we have estimated that a 1µg per cubic metre increase in $PM_{2..5}$ particulates (a very conservative estimate of the level of increase that would be expected around large incinerators) would lead to a reduced life expectancy of 40 days per person over 15 years (this equals a reduction of life expectancy of 1.1 years for each 10µg per cubic metre increase in $PM_{2.5}$ particulates). Although this figure appears small they note that

the public health implications are large and the effect on a typical population of 250,000 surrounding an incinerator would be a loss of 27,500 years of life over a 15 year time period. This figure gives an indication of the likely loss of life from any major source of $PM_{2.5}$ particulates. In addition, incinerators normally operate for much longer periods than the 15 years quoted here. Note that the estimated loss of life here is likely to be an underestimate as it is from particulates alone and not from other toxic substances.

The European Respiratory Society[67] has published its concern about the mismatch between European Union policy and the best scientific evidence. They state that a reduction in the yearly average $PM_{2.5}$ particulates to 15µg per cubic metre * would result in life expectancy gains, at age 30, of between 1 month and 2 years. They point out that the benefits of implementing stringent air pollution legislation would outweigh the costs. These recommendations are sensible and based on sound science. A programme of building incinerators would unfortunately achieve the opposite: they would increase particulate pollution, reduce life expectancy and would be at odds with the best science.

Statements by leading researchers include the following: "*the magnitude of the association between fine particles and mortality suggests that controlling fine particles would result in saving thousands of early deaths each year*" (Schwartz)[61] and "*there is consistent evidence that fine particulates are associated with increased all cause, cardiac and respiratory mortality. These findings strengthen the case for controlling the levels of respiratory particulates in outdoor air*" [60].

*** The National Ambient Air Quality Standard for $PM_{2.5}$ particulates was introduced into the USA in 1997 with a mean annual limit of 15µg per cubic metre. This had measurable health benefits. An annual mean limit for $PM_{2.5}$ particulates is to be introduced into Scotland in 2010 and this will be 12µg per cubic metre. An annual mean target for $PM_{2.5}$ particulates is to be introduced into the UK in 2020 and this will be will be 25µg per cubic metre. Many will wonder why the difference is so vast when the science is the same.**

i) Summary

In summary there is now robust scientific evidence on the dangers to health of fine particulates and of the substantial health costs involved. Recent studies have shown the risk to be considerably greater than previously thought**.** For these reasons it is impossible to justify increasing levels of these particulates still further by building incinerators or any other major source of $PM_{2.5}$ particulates. The data makes it quite clear that attempts should be made to the reduce levels of these particulates whenever possible. However particulates are not the only reasons to be concerned about incinerators. There are other dangers:-

3.2 Heavy Metals

Pope reported that hospital admissions of children with respiratory disease fell dramatically in the Utah valley when a steel mill was closed for a year due to a strike. Air pollution analysis showed that the metal content of particulates was lower that year and that the type of inflammation found in the lungs while the steel mill was working could be reproduced in both rat and human lung tissue by using air pollutants of the type emitted by the steel mill[68,69]. This is a very clear illustration of the dangers of pollution of the air with heavy metals. Exposure to inhaled metals, similar to the type produced by incinerators, have been shown to mediate cardiopulmonary injury in rats[70] and small amounts of metal (<1%) in particulates are known to cause pulmonary toxicity[71]. Salts of heavy metals such as iron and copper act as catalysts for dioxin

formation causing rapid rates of dioxin formation[72] increasing the dangers from burning metals.

Incinerator emissions to air and ash contain over 35 metals[73]. Several are known or suspected carcinogens. Toxic metals accumulate in the body with increasing age[74]. Breathing in air containing toxic metals leads to bioaccumulation in the human body. They can remain in the body for years: cadmium has a 30 year half-life. Incineration adds to the burden of toxic metals and can lead to further damage to health.

Mercury is a gas at incineration temperatures and cannot be removed by the filters. Incinerators have been a major source of mercury release into the environment. In theory mercury can be removed using activated carbon but in practice it is difficult to control and, even when effective, the mercury ends up in the fly ash to be landfilled. Mercury is one of the most dangerous heavy metals. It is neurotoxic and has been implicated in Alzheimer's disease[75-77], learning disabilities and hyperactivity[78,79]. Recent studies have found a significant increase in both autism and in rates of special education students around sites where mercury is released into the environment[80,81].

Inhalation of heavy metals such as nickel, beryllium, chromium, cadmium and arsenic increases the risk of lung cancer[12]**.** Cumulative exposure to cadmium has been correlated with lung cancer[82]. Supportive evidence comes from Blot and Fraumeni who found an excess of lung cancer in US counties where there was smelting and refining of non-ferrous metals[83]. Inhaled cadmium also correlates with ischaemic heart disease[84].

So what are the dangers caused by toxic metals accumulating in the body? They have been implicated in a range of emotional and behavioural problems in children including autism[85], dyslexia[86], impulsive behaviour[87] attention deficit and hyperactivity disorder (ADHD)[88,89] as well as learning difficulties[14,78,90-93], lowered intelligence[89] and delinquency[94,89], although not every study reached standard significance levels. Many of these problems were noted in the study of the population round the Sint Niklaas incinerator[95]. Exposed adults have also been shown to be affected, showing higher levels of violence[13,96], dementia[97-103] and depression than non-exposed individuals. Heavy metal toxicity has also been implicated in Parkinson's disease[104].

Heavy metals emitted from incinerators are usually monitored at 3 to 12 monthly intervals in the stack: this is clearly inadequate for substances with this degree of toxicity.

3.3 Nitrogen Oxides and Ozone

Nitrogen dioxide is another pollutant produced by incinerators. It has caused a variety of effects, primarily on the lung but also on the spleen, liver and blood in animal studies. Both reversible and irreversible effects on the lung have been noted. Children between the ages of 5 and 12 years have been estimated to have a 20% increase in respiratory symptoms for each 28 µg per cubic metre increase in nitrogen dioxide. Studies in Japan showed a higher incidence of asthma with increasing NO_2 levels and that it synergistically increases lung cancer mortality rates[41]. It has also been reported to aid the spread of tumours[105,106]. Increases in NO_2 have been associated with rises in admissions with COPD[107], asthma in children and in heart disease in those over 65[18]. Other studies have shown increases in asthma admissions[108] and increased mortality with rising NO_2 levels[109].

Rising ozone levels have led to increasing hospital admissions, asthma and respiratory inflammation and have been reported to lower immunity[110]. Higher levels have been significantly associated with increased mortality[111] and with cardiovascular disease. Both ozone and nitrogen dioxide are associated with increasing admissions with COPD[107.]

When it comes to incinerator emissions the health effects of nitrous oxides are likely to compound the negative health effects of particulates

3.4 Organic Toxicants

Hundreds of chemical compounds are released from incinerators. They include a host of chemicals produced from the burning of plastic and similar substances and include polycyclic aromatic hydrocarbons (PAHs), brominated flame retardants, polychlorinated biphenols (PCBs), dioxins, polychlorinated dibenzofurans (furans). These substances are lipophilic and accumulate in fatty tissue and remain active in the living organisms and the environment for many years. They have been linked with early puberty[112], endometriosis[113], breast cancer[114,115], reduced sperm counts[116] and other disorders of male reproductive tissues[117], testicular cancer[118] and thyroid disruption[11]. It has been claimed that about 10% of man-made chemicals are carcinogenic (see section 5.1), and many are now recognised as endocrine disrupters. Most of these health effects were not anticipated and are only now being recognised. No safety data exist on many of the compounds released by incinerators.

PAHs are an example of organic toxicants. Although emission levels are small these substances are toxic at parts per billion or even parts per trillion[73] as opposed to parts per million for many other pollutants. They can cause cancer, immune changes, lung and liver damage, retarded cognitive and motor development, lowered birth weight and lowered growth rate[73].

a) Organochlorines

The most detailed analysis to date on incinerator emissions has identified several hundred products of incomplete combustion (PICs) including 38 organochlorines – but 58% of the total mass of PICs remained unidentified[119]. Organochlorines, which include dioxins, furans and PCBs, deserve special attention, because of their known toxicity, because they bioaccumulate, and because of the likelihood that they will increase in the waste stream. Their major precursor, PVC, presently makes up 80% of organically bound chlorine and the amount of PVC in waste is likely to increase significantly in the future[120]. Clearly organochlorines will be an important component of incinerator emissions.

Organochlorines as a group are associated with six distinct types of health impact and these often occur at low concentrations. They are associated with 1) reproductive impairment in males and females 2) developmental damage 3) impaired cognitive ability and behaviour 4) neurological damage 5) suppressed immunity and 6) hormonal disruption and hormonal cancers. Each of these six effects has been demonstrated in three separate fields: in humans, in laboratory animals and in wildlife[121]. The American Pubic Health Association (APHA) concluded "virtually all organochlorines that have been studied exhibit at least one of a range of serious toxic effects, such as endocrine disruption, developmental impairment, birth defects, reproductive dysfunction and infertility, immunosuppression and cancer, often at extremely low doses"[122]. Other organohalogens such as bromides and fluorides have many similar properties.

A common misconception is that these pollutants have little effect if dispersed into the environment. This is wrong for several reasons. Firstly they are persistent as there is no mechanism in the environment to break them down and so they accumulate. Secondly as they are fat soluble they concentrate in living matter, often dramatically, at progressively higher concentrations (bioaccumulation). For example dioxin has been found in fish at levels 159,000 times that found in the water[123]; PCBs have been found in North Pacific Dolphins at 13 million times the concentration in the water[124] and trichloroacetic acid is found in North European conifers at 3-10,000 times that in the ambient air[125]. Thirdly they are concentrated by the foetus so a typical polar bear cub has a body burden double that of its mother[126] and at a level known to cause reproductive failure, altered brain development and immune suppression[127]. Fourthly they are nearly all toxic. In short the ability of ecosystems to assimilate organochlorines and other persistent bioaccumulative compounds is close to zero and they should simply never be released into the environment.

b) Dioxins

Dioxins are the organochlorines compounds most associated with incinerators and inventories have consistently shown that incinerators are the major source of emissions of dioxins into the air[128-30] though these are decreasing*. Dioxin releases over the last few decades have caused widespread contamination of food, significant toxic body burdens in nearly all human beings and severe pollution of the Arctic. None of this was foreseen. The damage already done by incinerators has been incalculable.

Eighteen separate assessments of dioxin's carcinogenicity have involved five different routes of exposure, five different species, low and high doses and long or short exposure times. In *every* case dioxins have caused cancer, involving nine different types of cancer, including lymphomas, cancers of the lung, liver, skin, soft tissue and of the oral and nasal cavities[131]. The National Institute of Environmental Health have looked for, but been unable to find, any threshold for the toxicity of dioxin. At the lowest detectable concentrations it can induce target genes and activate a cascade of intracellular molecular effects and can promote pre-malignant liver tumours and disrupt hormones[132]. Even doses as low as 2.5 parts per quadrillion can stop cultured cells from showing changes characteristic of immune responses[133].

The US Environmental Protection Agency's current estimate of dioxin's carcinogenicity, derived from animal studies, is that the average person's exposure to dioxin, which is 3-6 picogram per kilogram per day** gives a lifetime cancer risk of between 500 and 1000 per million[134]. (An acceptable cancer risk is considered to be between 1 in a million and 1 in 100,000). In comparison, a German study[135], derived from human dioxin exposure, found that each additional unit dose of dioxin (one picogram per kilogram of body weight per day) is associated with an increase in lifetime cancer risk of between 1000 and 10,000 per million.

The average infant receives doses of dioxins of 60-80 picograms (TEQ) per kilogram per day[136,137] which is 10- 20 times higher than those of the average adult and exceeds by a factor of 6 – 10,000 every government in the world's acceptable daily intake.*** This dioxin intake in the first year has been calculated to pose a cancer risk to the average infant of 187 per million (187 times the acceptable level)[138].

All these figures demonstrate that dioxins already in the environment are at unacceptable levels and are likely to be causing up to 6% of all cancers and to be having a range of adverse impacts on health including subtle effects.

Rats given dioxin to produce a body burden of dioxin at about half the average in the human population had male offspring whose sperm count was reduced by 25%[139] and rhesus monkeys given dioxin equivalent to twice the average human body burden had increased foetal death in their offspring and cognitive impairment which was transgenerational (passed on to their offspring) and abnormally aggressive behaviour[140,141]. This data indicates that releasing even a small amount of dioxin into an already overloaded environment can simply not be justified.

*An assessment of dioxins by the European Dioxin Inventory in 2005 found that in the UK, the biggest single source of dioxins in 2000 and in 2005 (projected figure) was the incineration of municipal waste, producing 20 times as much dioxin as road transport[142].

** a picogram is 1,000,000,000,000 gram, ie. a billionth of a gram in the UK, but more typically described in US literature as a trillionth of a gram.

*** Tolerable daily intake (TDI) is set at 0.006 picograms/kg per day in the US and 2 picograms/kg per day in the UK.

3.5 Effects on Genetic Material

Both heavy metals and many chemicals form covalent bonds with DNA called DNA adducts. This can increase the risk of cancer by activating oncogenes and blocking anti-tumour genes. This raises a very serious concern. This concern is that by releasing chemicals into the environment we may not only be poisoning this generation but the next. Carcinogenesis from chemicals being passed on through several generations is not just a horrifying scenario but has been demonstrated to occur in animals[143,144]. Incinerator emissions would greatly increase this risk.

DNA adducts to PAHs increase with exposure to pollution and patients with lung cancer have higher levels of adducts (see below). This is one demonstration of how pollutants alter genes and predispose to cancer. Other chemicals, such as vinyl chloride interfere with DNA repair and yet others such as organochlorines are tumour promoters.

3.6 Effects on the Immune System

Starting in the late 1980s a series of dramatic marine epidemics killed off thousands of dolphins, seals and porpoises. Many were found to have been affected by a distemper-like virus. Autopsies of the dead animals showed weakened immune systems and high levels of pollutants including PCBs and synthetic chemicals. A virologist, Albert Osterhaus and his co-workers, demonstrated that when seals were fed contaminated fish containing organochlorines (which were, however, considered fit for human consumption) they developed immune suppression and were unable to fight viruses[145-7]. Their natural killer cells were 20-50% below normal and their T cell response dropped by 25-60%. The immune suppression was due to dioxin-like chemicals, PCBs and synthetic chemicals. An immunologist Garet Lahvis found immunity in dolphins in the USA dropped as PCBs and DDT increased in their blood[148]. The immune system appeared most vulnerable during prenatal development. This demonstrates that the immune system may be damaged by exposure to synthetic chemicals and that we have seriously underestimated the dangers of these chemicals.

Animal experiments have shown immunotoxicity with heavy metals, organochlorine pesticides and halogenated aromatics[149] and accidental exposure data on humans has shown immunotoxicity with PBBs, dioxins and aldicarb. In fact whole volumes have been written on immunotoxicity[150]. Note these are the type of pollutants released by incinerators. Environmental toxins have been shown to decrease T-lymphocyte helper-suppressor ratios in four different exposed populations[151]. Nitrogen dioxide exposure leads to abnormally elevated immune and allergic responses. $PM_{2.5}$

particulates themselves can cause mutagenic and cytotoxic effects and the smallest particulates cause the greatest effects[152].

In utero exposure to dioxins results in thymus atrophy and weakened immune defences[153]. When female rhesus monkeys were exposed to PCBs at very low levels producing a body burden typical of general human population, their offspring's ability to mount a defence against foreign proteins was permanently compromised[154].

In summary there is abundant evidence that a large number of the pollutants emitted by incinerators can cause damage to the immune system[155]. As is demonstrated in the next section the combination of these is likely to have an even more potent and damaging effect on immunity than any one pollutant in isolation.

3.7 Synergistic Effects

Various studies have shown that a combination of substances can cause toxicity even when the individual chemicals are at a level normally considered safe. The report "Man's Impact on the Global Environment" by the Massachusetts Institute of Technology stated "synergistic effects among chemical pollutants are more often present than not"[156]. Testing has been minimal and most of the synergistic effects are likely to remain unknown. Toxicologist Prof Vyvyan Howard has calculated that to test just the commonest 1,000 toxic chemicals in unique combinations of three would require 166 million different experiments and even this would disregard varying doses[157].

Synergy has been demonstrated when organic chemicals are combined with heavy metals,[158,159] and with combinations of pesticides[160,161] and food additives[162]. The last study is of particular concern. Rats fed with one additive were unharmed. Those fed two developed a variety of symptoms whereas those fed all three all died within two weeks. In this case the chemicals appeared to amplify each other's toxicity in logarithmic fashion. In a recent experiment scientists dosed animals with a mixture of 16 organochlorine pesticides, lead and cadmium at "safe levels" and found they developed impaired immune responses, altered thyroid function and altered brain development[163]. Another study in 1996, published in Science, reported on the dangers of combinations of pesticides and their ability to mimic oestrogen. They found that combinations could increase the toxicity by 500 to 1000 times[164]. Mice exposed to 25 common groundwater pollutants, all at levels well below those that produce any effects in isolation, developed severe immunosuppression[165]. The level of concern about the multiplicity of pollutants released into the air by incinerators is enhanced by the fact that even when the probable effects of the single pollutants involved are known, no one has any idea what damage the combinations can cause.

The population living round an incinerator is being exposed to multiple chemical carcinogens, and to fine particulates, to carcinogenic heavy metals (in particular cadmium) and in some cases to radioactive particles, all known to increase lung cancer. Nitrogen dioxide has also been shown to synergistically increase lung cancer. When all these are combined, the effects are likely to be more potent, and, in fact, an increase in the incidence of lung cancer has been reported around incinerators (see section 4.1).

The potential for multiple pollutants to cause other serious health effects is illustrated by the results of a key study on rats exposed to the dust, soil and air from a landfill site. These animals developed abnormal changes in the liver, thyroid and reproductive organs within only two days of exposure[166]. Although effects in animals do not always mimic those in humans, the authors concluded that present methods of

calculating health risks underestimate the biological effects. This has obvious relevance to the dangers of exposing people to multiple pollutants from incinerators.

4. Increased Morbidity and Mortality near Incinerators

4.1 Cancer

There have been a number of studies of the effect of incinerators on the health of the surrounding population, mainly concentrating on cancer incidence. In most studies, the incinerators were situated near other sources of pollution and often in areas of deprivation, both likely to confound the findings since both are associated with higher cancer incidence. The study of an incinerator burning 55,000 tonnes of waste a year and built in 1977 in the middle of a residential area of a town of 140,000 with no heavy industry (Sint Niklaas) is scientifically unsatisfactory because funds were not made available for the study of controls[95]. However, the investigators mapped a convincing cluster of 38 cancer deaths immediately surrounding and to leeward of the incinerator, and this area also showed high concentrations of dioxin in soil samples when tested in 1992. They noted that the cancer SMR for this town for 1994-1996 (national statistics) was high (112.08 for males and 105.32 for females), supporting the genuine nature of their findings.

In 1996, Elliott et al. published a major study[167] in which they compared the numbers of registered cancer cases within 3 km and within 7.5 km of the 72 municipal waste incinerator sites in the UK with the number of cases expected. It involved data on over 14 million people for up to 13 years. Expected numbers were calculated from national registrations, adjusted for unemployment, overcrowding and social class. No account was taken of prevailing winds, or of differences between incinerators. They first studied a sample of 20 of the incinerator sites, replicating the analysis later with the other 52. If the results of two sets like this concur, it strengthens the data. In each set there was an excess of all cancers near the incinerators, and excesses separately of stomach, colorectal, liver and lung cancers, but not leukaemias. The first set gave adjusted mortality ratios for all cancers of 1.08 for within 3km and 1.05 within 7.5 km; for the second these were 1.04 and 1.02. These risks, representing an additional risk of 8% and 5% for the first set and 4% and 2% for the second, seem small **but represented a total of over 11,000 extra cancer deaths near incinerators** and were highly significant ($p < 0.001$ for each).

For each of the main cancer sites the excesses were higher for those living within 3 km than for all within 7.5 km[167,168], suggesting that the incinerators had caused the excess. The authors doubted this and attributed the findings to additional confounding in spite of the fact that they had already adjusted (possibly over-adjusted) for unemployment, overcrowding and social class, which give a partial correction for pollution. Moreover, the effect on people living to leeward of the incinerator would be substantially higher than shown by this study as the true number of people affected was diluted by those living at the same distance but away from the wind plume coming from the incinerator.

Knox et al. looked at the data from 22,458 children who died of cancer between 1953 and 1980 in the UK[169]. For each child they compared the distance of the

birth and death addresses from the nearest source of pollution and found a consistent asymmetry: more had moved away from the nearest hazard than towards it[169]. They deduced that the excess of migrations away from the hazard (after allowing for social factors) was evidence that the children had been affected by the cancer-causing pollution before or shortly after birth.

Later they applied the method to the set of incinerators studied by Elliott et al. and again showed the same asymmetry in the children's birth and death addresses, indicating that the incinerators had posed a cancer risk to children[170]. Of the 9,224 children for whom they had found accurate birth and death addresses, 4,385 children had moved at least 0.1 km. Significantly, more children had migrated away from incinerators than towards. For all those who had at least one address within 3 km of an incinerator, the ratio was 1.27. When they limited the analysis to children with one address inside a 5 km radius from the nearest incinerator and the other address outside this radius the ratio was 2.01; this indicated a doubling of cancer risk. Both these findings were highly significant ($p < 0.001$ for each). The excess had only occurred during the operational period of each incinerator and was also noted round hospital incinerators but not landfill sites. This is strong evidence that the incinerators' emissions contributed to the children's cancer deaths.

Biggeri et al. in 1996 compared 755 lung cancer deaths in Trieste with controls in relation to smoking, probable occupational exposure to carcinogens and air pollution (measured nearest to their homes) and the distance of their home from each of four pollution sites. The city centre carried a risk of lung cancer but the strongest correlation was with the incinerator where they found a 6.7 excess of lung cancer after allowing for individual risk factors[171].

Using a spatial scan statistic, Viel et al 2000 looked at the incidence of soft tissue sarcoma and non-Hodgkin's lymphoma from French Cancer Registry data, in two areas close to an incinerator with high emission of dioxin[172]. They found highly significant clusters of soft tissue sarcoma (RR 1.44) and of non-Hodgkins lymphoma (RR 1.27) but no clusters of Hodgkins disease (used as negative control). This study was interesting in that it was designed to look both in a focussed way at the area round the incinerator, and to check the association by looking for space time relationships which should be present if the relationship was causal. In addition they looked in an unfocussed way for other clusters in the wider area which contained other areas of deprivation. Both the first two analyses were positive close to the incinerator - demonstrating that a causal relationship was likely - and since no other clusters were found they concluded that deprivation could be virtually excluded as a factor.

According to Ohta et al, Japan built 73% of all the municipal waste incinerators in the world and by 1997 had become very concerned about their health effects: in the village of Shintone, 42% of all deaths between 1985-95 in the area up to 1.2 km to leeward of an incinerator (built in 1971) were due to cancer, compared to 20% further away and 25% overall in the local prefecture[173]. Their data on soil contamination reinforced the importance of considering wind directions in evaluating the health effects of incinerators.

Comba found an increased incidence of soft tissue sarcoma in an Italian population living within 2 km of an incinerator[174]. Zambon et al looked at cases of sarcoma from a different perspective. They calculated dioxin exposure from incinerators and other industrial sources in patients with sarcoma using a dispersion model and found the risk of sarcoma increased with the extent and duration of exposure to dioxin[175].

In 1989 Gustavsson reported a twofold increase in lung cancer in incinerator workers in Sweden compared to the expected local rate[176]. In 1993 he reported a 1.5 fold increase in oesophageal cancer in combustion workers, including those working in incinerators[177].

4.2 Birth Defects

There have been five reports of increases in congenital abnormalities around incinerators. The investigators at Sint Niklaas noted multiple birth defects to leeward of the incinerator[95]. Orofacial defects and other midline defects were found to be more than doubled near an incinerator in Zeeburg, Amsterdam[178]. Most of these deformed babies were born in an area corresponding to wind-flow from the incinerator and other defects included hypospadius and spina bifida. In the Neerland area, Belgium, there was a 26% increase in congenital anomalies in an area situated between two incinerators[179]. A study of incinerators in France has shown chromosomal defects and other major anomalies (facial clefts, megacolon, renal dysplasias)[180]. A recent British study looked at births in Cumbria between 1956 and 1993 and reported significantly increased lethal birth defects around incinerators after adjusting for year of birth, social class, birth order, and multiple births. The odds ratio for spina bifida was 1.17 and that for heart defects 1.12. There was also an increased risk of stillbirth and anencephalus around crematoriums[181]. The study pointed out that the figures for birth defects are likely to be substantial underestimates since they do not include spontaneous or therapeutic abortions, both increased by foetal anomalies.

In addition, several studies have noted an increase in birth defects near waste sites, particularly hazardous waste sites. The pattern of abnormalities was similar to the pattern found with incinerators, with neural tube defects often being the most frequent abnormality found, with cardiac defects second[182-85]. Harmful chemicals are normally stored in fatty tissue: in the foetus there is little or no fatty tissue except for that in the brain and nervous system, which may explain the pattern of damage. A review of this subject stated "*the weight of evidence points to an association between residential proximity to hazardous waste site and adverse reproductive outcomes.*"[186]

4.3 Ischaemic Heart Disease

Gustavsson found an excess of ischaemic heart disease[176] in incinerator workers who had been exposed for longer. We have not found any epidemiological studies of cardiovascular disease in the neighbourhood of incinerators, but in view of the research on particulates (see section 3.1) this should be investigated.

4.4 Comment

The authors of some of these reports did not consider that they had sufficient grounds for concluding that the health effects round incinerators were *caused* by pollution from the incinerators. However, statistically their findings were highly significant and, taking the studies together, it is difficult to believe that all their results could have been due to unrecognised confounding variables. This is even less likely when you consider the nature of the pollutants released from incinerators and the scientific evidence for the health effects of those compounds (see sections 2 and 3). The concordance of increased cancer incidence in local areas demonstrated to be more polluted also points to a causal association, although it does not necessarily imply that the pollutant measured contributed to the increase.

The studies may have underestimated the risks. At 13 years, the follow-up period of the large British study was probably too short: at Sint Niklaas adult cancer cases seemed to increase from 13 years onward (although children's cancers occurred earlier), and in Japan, Ohta noted that cancer caused 42% of all deaths in the lee of incinerators from 14 to 24 years after the incinerator was commissioned[173]. The reported risks were higher in the studies in which allowance was made for the direction of prevailing winds, possibly because of dilution elsewhere by relatively unexposed persons.

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

Taking into account these results and the difficulty in identifying causes of cancers and other chronic diseases, it is a matter of considerable concern that incinerators have been introduced without a comprehensive system to study their health effects, and that further incinerators are being planned without comprehensive monitoring either of emissions or of the health of the local population.

5. Disease Incidence and Pollution

5.1 Cancer

Studies linking cancer with incinerators cannot be seen in isolation. It is important to obtain an overall picture and look at other studies which link pollutants with cancer. And there is another aspect to this. Many types of cancer, including lung, pancreatic and stomach cancer, have a very poor prognosis and our only hope lies in prevention. Prevention means reducing our exposure to carcinogenic substances and we should take every opportunity to do this.

Cancer has shown an unrelenting rise over the last century, and is affecting younger people. The rise has been gradual, steady and real. Cancer incidence has been increasing by 1% per annum with an age standardized increase in mortality of 43% between 1950 and 1988[187]. Put another way, the chance of dying from cancer at the turn of the 20th century was 1 in 33. It is now 1 in 4. WHO data has demonstrated that 80% of cancers are due to environmental influences,[188] and evidence from migrant studies confirms that it is mainly the environment rather than the genes that determine the cancer risk[188].

Many people have noted that the rise in cancer has paralleled the rise in the production and use of synthetic chemicals, all the more remarkable since there has been a simultaneous large drop in smoking in males in many countries. In the second half of the twentieth century synthetic chemical production doubled every 7 to 8 years with a 100 fold increase over the last 2 generations[189]. Many converging pieces of evidence link chemicals to the relentless rise of cancer.

a) Links between exposure to pollutants and cancer in man

- Cancer is commonest in industrialised countries with 50% of cases in the industrialised 20% of the world[190] and the WHO has noted that cancer incidence rises with the GNP of a country.
- There is the same correlation within countries. The highest mortality from cancer in the USA is in areas of highest industrialised activity. There is also a correlation in the USA between cancer incidence and the number of waste sites in the county[191,192]. Counties with facilities for treating toxic waste have four times as much breast cancer[193]. Cancer is also commoner in counties with chemical industries[194]. Public Data Access in the USA shows a close correlation between cancer mortality and environmental contamination[195].
- Numerous studies have shown higher cancer incidence in both industrial workers and in populations living in polluted areas.[196,197]
- One of the three most rapidly rising cancers, non-Hodgkin's lymphoma, has been clearly linked with exposure to certain chemicals (for instance phenoxyherbicides and chlorophenols).[198,199]

b) Links between exposure to pollutants and cancer in animals

Three decades of studies of cancers in wildlife have shown that these are intimately associated with environmental contamination. This is particularly important as animals do not smoke, drink or eat junk food and cannot be accused of living in deprived areas. This strengthens the long-suspected link between environmental pollution and cancer. In a recent study of outbreaks of liver cancer in 16 different species of fish at 25 different sites, cancers were always associated with environmental contamination[200]. Dogs have been found to have higher rates of bladder cancer in industrialised counties in the USA[201]. It is inconceivable that we are not affected in the same way. Furthermore cancer rates in animals rapidly decline when the pollutants are removed showing the critical importance of an uncontaminated environment for good health.[202]

c) Large increases in cancer in certain tissues

Steep rises in cancer have occurred in tissues directly exposed to the environment: the lung and skin. But some of the steepest rises have occurred in parts of the body with high fat content, including cancers of the brain, breast, bone marrow and liver. This again points to toxic chemicals which are predominantly stored in the fatty tissues.

d) Genetic mutation

Many chemicals are known to attach to DNA causing genetic change in the form of DNA adducts. The research of molecular epidemiologist, Dr Frederica Perera, of Columbia Centre for Children's Environmental Health, has shown consistent associations between exposures to pollution and DNA adduct formation on the one hand and adduct formation and cancer risk on the other[203,204]. Perera found two to three times the level of DNA adducts to polycyclic aromatic hydrocarbons in people in polluted areas and also found higher levels of adducts in people with lung cancer than in those without. Mothers exposed to pollution form DNA adducts but their babies have even higher adduct levels potentially putting them at increased risk of cancer from birth[44].

e) Cancers and Environmental pollution

Several studies have already given direct evidence of a link between environmental pollution and cancer. These include the Long Island Study showing a link between airborne carcinogens and breast cancer[205,206] and the Upper Cape Study showing that tetrachloroethylene in the water was associated with elevated rates of several types of cancer[207-9]. It is noteworthy that initial investigations were negative in both these places and it was only demonstrated after detailed and sophisticated studies by scientists from many fields. Numerous other studies have shown links between cancer and chemicals: these include associations between volatile organic chemicals (VOCs) in the water and increases in leukaemia in New Jersey[210], increases in lymphoma in counties in Iowa where drinking water was contaminated with dieldrin[211], elevated levels of leukaemia in children at Woburn, Massachusetts coinciding with a known period of water contamination with chlorinated solvents[212], a cancer cluster linked to consumption of river water contaminated by industrial and agricultural chemicals in Bynum, North Carolina[213] and high rates of non-Hodgkin's lymphoma from water contamination with chlorophenols in Finland[214].

f) Spread of cancer and pollutants

Airborne pollutants not only affect the chance of contracting cancer but may also influence the chance of the cancer spreading. Animal studies showed that inhalation of ambient level nitrogen dioxide, or polluted urban ambient air, facilitated blood-borne cancer cell metastasis[105].

g) Levels of Carcinogens in the body

The reality about most chemicals is that their risks are largely unknown. This is particularly true of chemicals new to the market. What we do know is that about 5 to 10% are probable carcinogens. The International Agency for Cancer Research tested 1000 chemicals in 1993 and found that 110 were probable carcinogens[215]. The National Toxicity Program tested 400 chemicals in 1995 and found that 5-10% were carcinogenic[216]. Only 200 of the 75,000 synthetic chemicals in existence are regulated as carcinogens whereas, from this data, between 3,000 and 7,500 might be expected to be. We have even less knowledge about the carcinogenic potential of combinations of toxic chemicals but what evidence we do have suggests combinations may be more dangerous and yet these are what we are routinely exposed to.

Although the UK figures are not available we know that 2.26 billion pounds of toxic chemicals were released in the USA in 1994: about 177 million pounds of these will have been suspected carcinogens. But what happens to all these chemicals? The reality is that much of this chemical pollution ends up inside us. The evidence for this is as follows:-

In a study, a group of middle aged Americans were found to have 177 organochlorine residues in their bodies.[217,218] This is likely to be an underestimate as EPA scientists consider that the fatty tissues of the US general population contain over 700 additional contaminants that have not yet been chemically characterized[219]. A recent study by the Mount Sinai School of Medicine measured chemicals in the blood and urine of healthy volunteers and found an average of 52 carcinogens, 62 chemicals toxic to the brain and nervous system and 55 chemicals associated with birth defects[220]. They point out that these were chemicals that could be measured and that there were many more that could not, making this again a considerable underestimate. A study of pollutants in amniotic fluid found detectable levels of PCBs and pesticides at levels equivalent to the foetus's own sex hormones[221]. What these studies demonstrate is that what we put out into the world sooner or later comes back

to us and will be stored in our bodies, particularly the lipophilic, bioaccumulative compounds which are particularly damaging. This effect is slow, insidious and real. To allow carcinogens and other poisonous substances into our bodies in this way must be to gamble with our health.

Incinerators emit carcinogens. Particulates themselves are known to be carcinogenic, many heavy metals are known or suspected carcinogens, up to 10% of the chemical pollutants are carcinogenic and there is abundant evidence that carcinogens are far more dangerous when combined than when in isolation.

Common sense dictates that it is reckless to continue to pour more carcinogens into the air at a time when cancer is steadily increasing. Recent studies suggest that we already have to cope with 65 carcinogens in food, 40 carcinogens in water and 60 carcinogens in the air we breathe[222]. They should not be there at all. They should certainly not be increased. If we seriously want to prevent cancer it is of paramount importance that we rapidly decrease the levels of all carcinogens that we are exposed to.

5.2 Neurological Disease

Most toxic compounds are preferentially stored in fatty tissue and this includes the brain – making the brain a key target organ for pollutants. There is now compelling evidence that heavy metals and other compounds such as PCBs and dioxins cause cognitive defects, learning problems and behavioural disturbances in children and these effects occur at levels previously thought to be safe[223]. It is inconceivable that these same pollutants have no impact on adult brain function. In fact, some organochlorines, especially those with toxic metabolites and those that dissolve in the cell membranes are known to kill brain cells.[224,225] We note also the ability of ultrafine particulates to carry pollutants across the blood-brain barrier[65]. If neurones were lost at the undetectable rate of 0.1% annually this would lead to a major decline in brain function by middle age[226].

Of great concern is the developing crisis of Alzheimer's disease which now affects 4.5 million patients in the USA and nearly 700,000[227] in the UK. This is a disease which had never been diagnosed until 1907 and in the UK had only reached 150 cases by 1948. At the present rate of increase, the numbers will double by 2030. These statistics are alarming but need to be seen as part of an overall trend of increasing neurological disease. A recent study has noted substantial increases in neurological diseases in the last two decades coupled with earlier onset of these illnesses. Increases were noted in Alzheimer's disease, Parkinson's disease and motor neurone disease[228]. The increase in Alzheimer's disease was found in almost all developed countries, and rises varied across countries from 20% (which was defined as substantial) to 1200%. The paper suggested environmental factors were likely to be responsible.

It is notable that these diseases of older people have increased at the same time that diseases affecting the brain (including ADHD, autism and learning difficulties) have also shown large increases at the other end of the age spectrum, to the order of 200-1700%[229]. It is very likely that these diseases have aetiological factors in common.

Heavy metal exposure is known to correlate with both Parkinson's disease[103,230] and Alzheimer's disease[75,76,98-102]. Both diseases have increased dramatically over the last 30 years. In addition we have already noted that the average person's body contains at least 62 chemicals which are toxic to the brain and nervous system[220]. It is crucial to look at every possible way to prevent Alzheimer's because of

its huge care costs (US figures are $60 billion annually) and because of its dire effect on both patients and carers.

Although multiple factors are probably involved in its causation, there is evidence of a link to heavy metal exposure and it is therefore imperative to reduce our exposure to these toxic metals and other neurotoxic chemicals by all means possible. To deliberately increase our exposure to these pollutants, at a time when these diseases are showing huge increases, shows a worrying lack of foresight.

5.3 Mental Diseases

Many pollutants pass straight from the nose to the brain where they affect brain function. Air pollution correlates with inpatient admissions with organic brain syndrome, schizophrenia, major affective disorders, neurosis, behavioural disorder of childhood and adolescence, personality disorder and alcoholism[231]. Increases in the total number of psychiatric emergency room visits and in schizophrenia[232] have been noted on days when air pollution has been high. Depression has also been linked to inhaled pollutants[233,234]. Clearly something very profound occurs when we pollute the air.

5.4 Violence and Crime

An increasing number of studies, including studies of murderers[235], case-control and correlation studies[13,94,236,237] and prospective studies[96,238] have shown links between violence and heavy metals and these include lead, cadmium and manganese. The majority of the studies have investigated lead. Violence and crime have been associated with both increased body levels of lead and with increased levels of lead in the air. For instance Denno[239] found early lead exposure was one of the most important predictors of disciplinary problems from ages 13 to 14, delinquency from ages 7 to 17 and adult criminal offences, from ages 18 to 22. Stretesky found an association between air lead levels and murder rates in US counties[240]. It is interesting that air lead levels were a much stronger predictor of both violent and property crime than unemployment, which has often been considered an important cause for crime[241]. The likely mechanism is that these substances alter neurotransmitters such as dopamine and serotonin and reduce impulse control.

This growing literature should serve as a warning about the dangers of allowing heavy metals to be emitted into the environment. Crime, especially violent crime, can have a dramatic effect on people's quality of life. We need to consider the effect of incinerators, not only on health, but on education and on quality of life, including the impact of violence and crime.

6. High Risk Groups

6.1 The Foetus

The unborn child is the most vulnerable member of the human population. The foetus is uniquely susceptible to toxic damage and early exposures can have life changing consequences. Why is the foetus so vulnerable? There are two main reasons. Firstly most of these chemicals are fat soluble. The foetus has virtually no protective fat stores until very late pregnancy so the chemicals are stored in the only fatty tissues it has, namely its own nervous system and particularly the brain. Secondly many pollutants are actively transported across the placenta from the mother to the foetus. This occurs with heavy metals which the body mistakes for essential minerals. This is

particularly critical for mercury where one tenth of women already have body stores of mercury which can lead to neurodevelopmental problems in the newborn[242]. Other factors that increase foetal susceptibility are higher rates of cell proliferation, lower immunological competence and decreased capacity to detoxify carcinogens and repair DNA[243].

Safety limits currently do not take into account this increased risk to the foetus. Only 7% of high volume chemicals have been tested for neurodevelopmental toxicity[244] and very few pollutants have been tested for teratogenicity.

During a narrow window of time, in the first 12 weeks in utero, the foetus's body is affected by miniscule amounts of hormone measured in parts per trillion. Tiny amounts of chemicals can upset this delicate balance. It is now generally accepted that chemicals that are not toxic to an adult can have devastating effects on the newborn. Porterfield has shown that small amounts of chemicals such as dioxins and PCBs, at doses that are not normally regarded as toxic, can affect thyroid hormones and neurological development[11]. A single exposure is enough and timing is critical[245]. Small doses of oestrogenic chemicals can alter sexual development of the brain and the endocrine system[246].

It is estimated that 5% of babies born in the USA have been exposed to sufficient pollutants to affect neurological development[247]. It has also been shown that exposure to oestrogenic chemicals affects immunity, reduces the immune response to vaccines, and is associated with a high incidence of middle ear and recurrent respiratory infections[248]. The amount of chemical that the baby takes in relates to the total persistent contaminants that have built up in the mother's fat over her lifetime[249]. This will increase in areas around incinerators. Exposure to fine particulate pollution during pregnancy can have an adverse effect on the developing foetus and lead to impaired foetal growth[74].

In July 2005, in a ground-breaking study[250], researchers at two major laboratories in the USA looked at the body burden in the foetus. They reported an average of 200 industrial chemicals and pollutants (out of 413 tested) in the umbilical cord blood of 10 randomly chosen babies. These included 180 carcinogens, 217 chemicals that are toxic to the brain and nervous system and 208 that can cause birth defects and abnormal development in animals. A statement by scientists and paediatricians said that the report raised issues of substantial importance to public health, showed up gaping holes in the government's safety net and pointed to the need for major reform to the nation's laws that aim to protect the public from chemical exposures.

Two months later, scientists at the University of Groningen, released the results of a European study, commissioned by WWF and Greenpeace, on the foetal body burden. They tested for the presence of 35 chemicals in the umbilical cord blood of newborns[251]. At least five hazardous chemicals were found in all babies and some had as many as 14 different compounds. The report questioned the wisdom of allowing the foetus to be exposed to a complex mixture of persistent, bio-accumulative and bioactive chemicals at the most critical stage of life.

Incinerators can only have the effect of increasing the foetal body burden and their use is therefore a retrograde step for society. It is particularly important to apply the precautionary principle in issues that affect the foetus, infant and child.

6.2 The Breast-fed Infant

It is a major concern that breast milk, perhaps the greatest gift a mother can give for the future health of her child, has now become the most contaminated food on

the planet, in terms of persistent organic pollutants[252]. In the USA studies of human breast milk have shown that 90% of samples contained a disturbing 350 chemicals. This was higher in industrialised areas showing that inhalation of these toxic substances is an important factor[253]. The dose taken in by a breast-feeding baby is 50 times higher than that taken in by an adult[254].

The incinerator would add to the total load of chemicals in the mother's fat and those toxins accumulated over a lifetime by the mother will then be transferred to the tiny body of her baby through her milk. Six months of breast feeding will transfer 20% of the mother's lifetime accumulation of organochlorines to the child[255]. From 1979 one in four samples of breast milk have been found to be over the legal limit set for PCBs in commercial feeds[249] and these are known to impair intellectual development-[256-8]. Contamination with persistent organic pollutants (POPs) in breast milk in animals has consistently shown structural, behavioural and functional problems in their offspring[259]. For instance, in monkeys it has shown that it decreases their ability to learn[260-2]. Polybrominated diphenyl ethers (PBDEs) are toxic chemicals which have been doubling in breast milk every five years, and have also been rapidly increasing in the waste fed to incinerators as they are now present in many common electrical and electronic goods. PBDEs cause cancer, birth defects, thyroid dysfunction and immune suppression.[263,264] It is truly tragic that one of the few ways of removing these contaminants from the mother's body is by breast-feeding.

6.3 Children

Toxic and carcinogenic exposures in early life, including prenatal exposures, are more likely to lead to cancer than similar exposures later[265-7]. At the First International Scientific Conference of Childhood Leukaemia, held in September 2004, Professor Alan Preece suggested that pollutants crossing the placenta, were damaging the immune system and could be linked with soaring rates of leukaemia, which were being initiated in utero. This theme was expanded by Professor George Knox in his recent study which found that **children born in "pollution hotspots" were two to four times more likely to die from childhood cancer.** The "hotspots" included sites of industrial combustion, and sites with higher levels of particulates, VOCs, nitrogen dioxides, dioxins and benz(a)pyrenes – in other words just what would be found around incinerators. He said that, in most cases, the mother had inhaled these toxic substances and they were then passed on to the foetus through the placenta[268]. This is supported by animal studies which have already confirmed that cancer in young can be initiated by giving carcinogens before conception (to the mother), in utero or directly to the neonate[269,270].

Developing systems are very delicate and in many instances are not able to repair damage done by environmental toxicants[271]. In one study there was an age-related difference in neurotoxicity for all but two of 31 substances tested; these included heavy metals, pesticides and other chemicals[272]. Children are not just a vulnerable group but the current inhabitants of a developmental stage through which all future generations must pass. This fact is recognised in the passage of the Food Quality Protection Act in the USA. It requires that pesticide standards are based primarily on health considerations and that standards are set at levels which will protect the health of children and infants.

Developmental disorders including autism and attention deficit syndrome are widespread and affect 3-8% of children. The US National Academy of Sciences concluded in July 2000 that 3% of all developmental disorders were a direct consequence of toxic environmental exposures and another 25% are the result of

interactions between toxic exposures and individual susceptibility. The causes included lead, mercury, PCBs, certain pesticides and other environmental neurotoxicants[273], substances that are all discharged from incinerators

Recently associations have been reported in case control studies between the body burden of mercury and the risk of autism[274]. In other studies in Texas, associations have been found between the amount of mercury discharged into the air and water by chemical plants and the local incidence of autism[80] and an inverse relationship between the distances of schools from the plants discharging mercury and autism in their youngest pupils 4 years later; this is the lag expected from the fact that the greatest sensitivity to neurotoxicity is seen before birth and in neonates[81]. This suggests that mercury could be responsible but the contribution of other neurotoxins was not excluded.

The study of the Sint Niklaas incinerator found a multitude of problems in children, including learning defects, hyperactivity, autism, mental retardation and allergies[95] and this is exactly what would be anticipated from the above and research already done on the health effects of heavy metals, PCBs and dioxins on children. Animal studies show similarities, with a recent study demonstrating autistic-like behavioural changes in rats whose mothers has been exposed to PCBs whilst pregnant; they had developed abnormal plasticity in the cortex of the brain[275].

We need also to consider subclinical toxicity. The pioneering work of Herbert Needleman showed that lead could cause decreases in intelligence and alteration of behaviour in the absence of clinically visible signs of toxicity[92]. This has also been shown to be the case with PCBs[276] and methyl mercury[79]. These effects are all the more likely when children are exposed to multiple pollutants, notably the heavy metals, which will be found in the cocktail of chemicals released by incinerators.

Although this has only minor implications for an individual it can have major implications for a population. For instance a 5 point drop of IQ in the population reduces by 50% the number of gifted children (IQ above 120) and increases by 50% the number with borderline IQ (below 80)[277]. This can have profound consequences for a society, especially if the drop in IQ is accompanied by behavioural changes.

6.4 The Chemically Sensitive

In the book, Chemical Exposures, Low Levels and High Stakes by Professors Ashford and Miller[151], the authors noted that a proportion of the population react to chemicals and pollutants at several orders of magnitude below that normally thought to be toxic. For example research has discovered individuals who react to levels of toxins previously considered to be safe. Two examples are benzene[278] and lead[93]. It has been demonstrated that there is a tenfold difference between different individuals in the metabolism of the carcinogenic PAH benz(a)pyrene[279].

Ashford and Miller also noted that studies in both toxicology and epidemiology have recognised that chemicals are harmful at lower and lower doses and that an increasing number of people are having problems. A significant percentage of the population have been found to react this way (15 to 30% in several surveys with 5% having daily symptoms).[151] Research has shown 150 to 450 fold variability in response to airborne particles[280]. Friedman has stated that environmental regulation requires the protection of these sensitive individuals[281]. This highlights the dangers of incinerators which emit a multitude of chemical compounds. Chemical sensitivity is typically triggered by an acute exposure after which symptoms start to occur at very low levels of exposure[151]. Faults are all too common with modern incinerators leading to discharges of pollutants at levels that endanger health – giving

a very real risk of long-term sensitisation. Certain susceptible individuals will be highly affected by these pollutants and these effects will be difficult to anticipate. In addition, people affected this way are extremely difficult to treat.

7. Past Mistakes and The Precautionary Principle

7.1 The Precautionary Principle

The Precautionary Principle has now been introduced into national and international law including that of the European Union[282]. This principle involves acting in the face of uncertain knowledge about risks from environmental exposures. This means public health measures should be taken in response to limited, but plausible and credible, evidence of likely and substantial harm[283]. It is summed up in the 1998 Wingspread statement: "When an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically. In this context, the proponent of the activity, rather than the public, should bear the burden of proof." In the case of incinerators a recent review of health effects found two thirds of studies showed a positive exposure-disease association with cancer (mortality, incidence and prevalence)[284] and some studies pointed to a positive association with congenital malformations. In addition without exact knowledge of what pollutants are produced by incinerators, their quantities, their environmental fate or their health effects, it is impossible to assure their safety. It is absolutely clear from this and from the evidence presented here that building municipal waste incinerators violates the Precautionary Principle and perhaps European Law.

7.2 Learning from Past Mistakes

Time and time again it has been found that what we did not know about chemicals proved to be far more important than what we did know. As an incinerator generates hundreds of chemicals, including new compounds, we can expect many unpleasant future surprises. Here are a few examples from the past:

- **Chlorofluorocarbons (CFCs)** These chemicals were touted as the safest chemicals ever invented when first synthesised in 1928. Thomas Midgeley received the highest award from the chemical industry for his discovery. After 40 years on the market suspicion fell on them. They were producing holes in the ozone layer exceeding the worst case scenario predicted by scientists.
- **Polychlorinated biphenyls (PCBs)** These chemicals were introduced in 1929. Toxicity tests at the time showed no hazardous effects. They were on the market for 36 years before questions arose. By that time they were in the body fat of every living creature in the planet and evidence began to emerge of their endocrine disrupting effects.
- **Pesticides** Early pesticides included arsenical compounds but these killed farmers as well as pests. They were replaced by DDT. Paul Muller was awarded the Nobel Prize for this discovery as it was considered a milestone in human progress. But DDT brought death in a different way and it was another two decades before it was banned. Less persistent pesticides then came onto

the market but they had yet another unanticipated problem – endocrine disruption.

- **Tributyl tin (TBT)** In the early seventies scientists noted irreversible damage was occurring to the reproductive system of fish and shellfish, especially clams, shrimps, oysters, Dover Sole and salmon. It was 11 years before the cause was found and it was found to be due to be tributyl tin, a chemical added to paint to stop barnacles growing. Incredibly the damage was occurring at a concentration of just five parts per trillion. By the end of the eighties more than one hundred species of fish were known to have been harmed.

This pattern of unanticipated disasters and long latent intervals before their discovery characterises the history of many toxic chemicals and warrants great caution in the use of new compounds. Animal studies almost never warn us of the uniquely human neurotoxic effects on behaviour, language and thinking. In the case of lead, mercury and PCBs the levels of exposure needed for these effects to occur have been overestimated by a factor of 100 to 10,000[285]. To quote Grandjean[283] "*Past experiences show the costly consequences of disregarding early warnings about environmental hazards. Today the need for applying the Precautionary Principle is even greater than before*"

8. Alternative Waste Technologies

An ideal waste strategy would produce no toxic emissions, no toxic by-products, no residues that need landfilling (zero waste), good recovery of materials and be capable of dealing with all types of waste. This might seem a tall order but with a combination of approaches, it is now possible to come quite close to this goal.

Once this aim is made clear then incineration becomes a poor choice. The potentially dangerous emissions to air, the high volume of ash that needs landfilling and the very toxic nature of the fly ash would rule it out. Similarly pyrolysis produces toxic by-products and is best avoided.

The most important component of an integrated strategy must be some form of separation and recycling. We must also look at methods of dealing with residual waste that produce no ash, such as Mechanical-Biological Treatment, Anaerobic Digestion and Advanced Thermal Technologies.

8.1 Recycling, Re-use and Composting

Both government guidance and the European Union Waste Hierarchy make it clear that recycling and re-use are the highest priorities in waste management and that this should take precedence over incineration and landfill. This hierarchy has been described as reduction, reuse, recovery and disposal. Many fine words have been spoken, but the reality is, that without incentives to support recycling, both the increase in landfill tax and the European Directives to reduce the amount of biodegradable waste going to landfill are driving waste management towards its lowest priorities, principally incineration. This has now becoming the easiest option for local authorities. Waste policy is veering away from its stated highest priorities with their low environmental impact towards the least sustainable options which have the highest environmental impact.

The net effect of this is that incineration, with its large appetite for high calorific recyclable materials, is now in direct competition with recycling and has

become an obstacle to sound waste policy. This is an inversion of the Waste Hierarchy and removes the motivation to re-use and recycle. One way forward would be to use the strategy already employed by several countries such as Sweden and the Netherlands, where waste cannot be delivered to landfill or incinerators without having undergone separation or treatment. In effect, this stops the sending of recyclable items to landfill and incineration.

About 46% of municipal waste consists of paper, cardboard, fabrics, glass and metals – all of which could be recycled. Metals are becoming more valuable and are already being mined in dumps in parts of the world. About 32% consists of garden and food waste which could be composted. Several commentators have emphasised that, for recycling programs to work successfully, it is important to have systems in place that are easy to use. Doorstep collections of organic waste are especially important. Another 13% of waste is plastics which are discussed below.

The UK presently recycles about 23% of its waste. Many other countries recycle a far higher proportion of their waste with Norway, Austria and Holland achieving over 40% and Switzerland over 50%. St Edmundsbury in the UK has reached 50%. Below is a table showing that many areas have achieved high rates of municipal waste diversion (recycling, re-use and composting) and this demonstrates that diversion rates of 50-70% are realistic targets.

Locality	Diversion Rate (percent)
Zabbaleen-served areas of Cairo, Egypt	85
Opotiki District, New Zealand	85
Gazzo (Padua), Italy	81
Trenton, Ontario	75
Bellusco (Milan), Italy	73
Netherlands	72
Northumberland County, Ontario, Canada	69
Sidney, Ontario	69
East Prince, Prince Edward Island, Canada	66
Boothbay, Maine, U.SA	66
Halifax, Canada	65
Chatham, New Jersey, U.SA	65
Falls Church, Virginia, U.SA	65
Galway, Ireland	63
Belleville, Ontario	63
Canberra, Australia	61
Bellevue, Washington, U.SA	60
Guelph, Ontario, Canada	58
Gisbome District, New Zealand	57
Cfifton, New Jersey, U.SA	56
Loveland, Colorado, U.SA	56
Denma~	54
Bergen County, New Jersey, U.SA	54
Worcester, Massachusetts, U.SA	54
Leverett, Massachusetts, U.S.A.	53
Ann Arbor, Michigan, U.S.A.	52
Crockett, Texas, U.S.A.	52
Dover, New Hampshire, U.SA	52
Kaikoura District, New Zealand	52
Switzerland	50
Nova Scotia, Canada	50
Portland, Oregon, U.SA	50
Madison, Wisconsin, U.SA	50
Fitchburg, Wisconsin, U.SA	50
Visalia, California, U.SA	50

8.2 Producing Less Waste

However efficiently we recycle, re-use and compost, these cannot solve the waste problem without another vital step; namely producing less waste in the first place. To emphasise this point, the amount of municipal and business waste in the UK is still growing[286] in spite of higher rates of recycling.

Various solutions to this are gaining popularity. One is Extended Product Responsibility (EPR) where firms take physical and financial responsibility for products even after they are sold, collecting their products and packaging after use. This encourages firms not to produce non-recyclable and non re-usable products. It has been applied to packaging, tyres, and electronics. EPR needs to be extended but where this is not practical, such as where products are hazardous or non-recyclable, then a product ban might be appropriate. A further solution would be to tax non-recyclable items to discourage their production.

There is a further aside to this issue which has yet to be addressed by governments. The developed world is producing, and disposing of, increasing amounts of goods of all kinds, including large amounts of synthetic materials unknown a century ago. The rest of the world is not unnaturally wanting to share the prosperity, but we are rapidly reaching a point where continuing even at the present level will become impossible because we are running out of both energy and of essential materials, particularly oil.

We have finite sources of oil from which so many materials are made. We are probably close to reaching peak production and this resource will diminish over the next few decades at a time when demand is increasing internationally. Natural gas will peak a decade or two later and then diminish. The only other two major sources of energy would be coal and nuclear power. Nuclear energy, even in the unlikely event that a safe way could be found to deal with the radioactive waste, would last between 8 [287] and 17 years [288] if it was supplying 20-25% of the world's energy because uranium is also a finite resource. Burning coal could cause a disastrous increase in greenhouse gases. Again it could not make up for the shortage of energy and would last less than a century[289]. At present it appears that genuinely renewable sources of energy could provide, at the very most, 40% of our present energy requirements[289]. (In reality it is likely to be much less and it has been estimated renewable sources will produce 4¾ % of total energy and 22% of electricity by 2020 in the UK).[290] Different experts will have their own opinions on all of these figures, but one thing is certain: - we are running out of energy. We can anticipate a 20% reduction in energy from all sources in 40 years and a 40% reduction in 60 years[289]. Long before this happens the price of energy and of goods made from oil will soar.

There is only one possible solution to this problem in the long term and that is to reduce our use of energy which means reducing our production and consumption of goods, and preserving our resources, including the valuable components in our waste.

8.3 Zero Waste

Zero waste, initially introduced in New Zealand has been taken up successfully by other regions and cities such as San Francisco, The Philippines, Flanders, Canberra, Bath and North East Somerset. In the UK, 71% of councils have committed to zero waste as part of their plan. This means working towards a goal of producing zero waste and avoiding disposal in landfill and incineration. The policy of the European Union is already on the path towards zero waste. **Zero waste and incineration are mutually incompatible.**

There are some difficulties with zero waste. One is that not all materials can be recycled and there will be some residual waste, notably plastics. Other goods contain mixed ingredients (for example envelopes containing plastic windows) and cannot easily be recycled. These could be taxed or banned. Some areas such as Flanders in Belgium have recognised this problem and have innovatively set a target for residual waste, currently 150kg per capita per year (UK: 400kg per capita per year). This is a useful idea and the policy sends out a strong signal to manufacturers to produce recyclable products.

8.4 The Problem of Plastics

A large amount of our waste is plastics and related materials such as PVC. Presently only two types of plastics can be recycled. The first key question is what will we do with these non-recyclable plastics? The second key question is how do we make chlorinated plastics safe for the future, taking into account that their highly persistent and toxic nature? The third key question is can we use plastics as a future resource? These are not small issues. For example, we use 500 billion carrier bags each year. They are used for an average of 20 minutes and are virtually indestructible, lasting for centuries. Many end up as microscopic tilth in the oceans. They then find their way into the food chain via lugworms and barnacles.

Incineration is a poor answer to these issues as many plastics are organochlorines and form toxic products, notably dioxins, when burnt. In addition an important resource is wasted. We use about 3-4% of our oil to produce these plastics and it makes no sense to simply burn them. The best solution would be to stop making chlorinated plastics in the first place in view of their persistence and toxicity. Instead we could make biodegradable plastics (but note these will break down to form the greenhouse gas methane). Another answer is plasma gasification. Plasma gasification, unlike incineration can convert chlorine-based plastics back to their original starting material, namely salt and water and synthesis gas (carbon monoxide and hydrogen). Further procedures can be used to convert synthesis gases into highly useful materials: fuels such as ethanol and Fischer-Tropsch diesel (a cleaner form of diesel) or ethylene to produce more plastics. It other words it could be used to both detoxify and reform plastics.

8.5 Anaerobic Digestion of Organic Matter

The problems of landfills are threefold. One is the production of greenhouse gases, principally methane. The second is the seeping of chemicals from landfill sites into aquifers. The third is lack of space. The former is the most urgent problem to solve. The methane is produced by organic waste, in other words rotting organic matter, but not by plastics (except bio-degradable ones) or metals. At present the methane is burnt in a flare tower or gas generator plant at the landfill site. However this is very inefficient. A far better option is to remove the paper, plastics and metals and allow the waste to break down in an anaerobic digester. The methane can then be burnt in a combined heat and power plant to produce electricity and heat. As this occurs in a sealed unit the environmental impact is much less than a landfill gas power plant. If this type of facility was used for the majority of agricultural waste and sewage then it could supply 3% of the UK's electricity and would also displace carbon emissions[284.]

8.6 Mechanical Biological Treatment (MBT)

This treatment is used extensively in Germany, Italy and Austria, has been in use for over 10 years and is due to be introduced into the UK. The process involves a mechanical stage in which the waste is chopped up into fragments and then separated by being put through screens of various sizes and past magnets. This process will separate the waste into fractions which can be used for different purposes. For instance metals, minerals and hard plastics can then be recycled. Paper, textiles and timber can also be recovered. Organic matter can then be broken down by composting – this is the biological treatment. This can be achieved by exposing the waste to atmospheric oxygen or it can be broken down in the absence of oxygen (anaerobic digestion). The remaining rubbish can then be landfilled. This process is virtually pollution-free **unless the remaining pellets are burnt** with all the risks this entails. With MBT most of the original goals are being met. It fails on two counts only. Firstly there is some residue that needs landfilling – this is a minor point but the second is more serious: MBT cannot cope with all types of waste as it is not suitable for hazardous waste. This is important as the amount of hazardous waste is likely to increase. So MBT needs to be part of a system.

Note that residues from MBT have had the organic matter removed, so they will not produce the problematic greenhouse gases. For this reason we believe it is wrong that it incurs the full landfill tax as happens at present.

8.7 Advanced Thermal Technologies (ATT) and Plasma Gasification

In contrast with non-thermal methods, any thermal method of dealing with waste carries an inherent risk of causing fatalities. Because of this thermal methods should only be used for residual waste after full separation of recyclables has taken place. If thermal methods are used, these should always be the safest ones available. In effect this means plasma gasification or gasification using the Thermoselect process. Japan has more experience of incineration than any other country and has started to use plasma gasification as a safer alternative to incineration. Plasma gasification is also in use in Canada.

Plasma gasification achieves the final objective by disposing of the residual waste after separation and recycling and other separating technologies such as mechanical-biological treatment. It can deal safely with the most hazardous types of waste and can produce up to three times as much energy as incineration.

Gasification has been employed by the natural gas industry for over 80 years but has not, so far, been used extensively for dealing with waste, although such plants are now in operation in Italy, Switzerland, Germany and Japan. Gasification produces high temperatures and can thermally decompose complex and hazardous organic molecules into gases and benign simple substances. Plasma refers to the gas when it has become ionized and this happens when an electric current is passed through the gas. **A very important distinction from incineration is that it does not produce ash**. The gas cleaning process can convert many contaminants into environmentally benign and useful by-products. The abatement equipment of incinerators and gasification units is very different. If the abatement equipment in an incinerator fails, as is all too common, people downwind from the installation will be subjected to dangerous pollution. If the abatement equipment in a gasification unit fails it will cause serious damage to the plant itself – so the plant has to be built to a much higher quality.

In a plasma gasification plant, the residual toxic substances including metals become encapsulated in silicate which is like being encased in stone. The plant will remove the toxic and persistent compounds from plastics and other chemicals and

reform them. A good quality plasma gasification unit will not produce any adverse residues or by-products, only synthesis gas, silica, sulphur and salt. Synthesis gas is a useful by-product which can be used as a fuel; ─ a major financial advantage which allows the capital costs of the unit to be paid within a 7 year period. Although it is a relatively expensive process, it is far cheaper than incineration once the health costs are taken into account (see section 9.1). Note also that it would not incur costs under the European Union Emissions Trading Scheme, potentially saving millions of pounds annually. A recent review of plasma gasification considered it to be a promising alternative to older technologies and that the present climate favoured the adoption of advanced technologies for waste treatment[291]. If it is combined with MBT and recycling, then only a small unit would be needed.

It is important to realise that gasification systems can vary in quality and therefore safety. It is crucial that there is a good gas cleaning system which goes through 7 or 8 stages. It is also essential that temperatures of 1500 C are achieved - enough to break down organochlorines and convert them back to their original safe form, salt and water.

Organochlorines are probably the most problematical group of chemicals on the planet so a real benefit of this technology is that this process reverses of the chlor-alkali process that produces organochlorines in the first place

8.8 Greenhouse Gases

Incineration has been sold as a source of green energy and even more bizarrely as a source of renewable energy. This is far from the truth. In a recent report, incineration was found to be second only to coal fired power stations as a producer of greenhouse gases.

However this is only part of the problem. With incineration there are two releases of greenhouse gases – once when the material is burnt and another when it is re-manufactured. Once we add to the equation the carbon and other greenhouse gases produced when these products are remade, as opposed to being recycled, then it becomes obvious how wrong it would be to regard incineration as a source of green energy. In fact, between two to five times more energy goes into remaking products than the energy recovered from incinerating them[292].

Recycling is far more energy efficient than incineration and has greater carbon benefits. With the high rates of methane capture assumed by DEFRA, landfill has similar CO_2 emissions to incinerators.

All incinerators should be routinely assessed for their effect on global warming.

9. The Costs of Incineration

9.1 Direct and Indirect Costs

Incineration has been reported to be more expensive than alternative waste strategies even when health costs are not considered. A recent document from the Scottish Environmental Protection Agency estimated that the disposal costs to process a tonne of waste would be £50-80 for incineration compared to £30-40 for aerobic digestion. These costs include high transportation costs and the equivalent figure for England would be £20-30 lower per tonne (making it approx £25-55 per tonne for incineration and £5 per tonne for aerobic digestion). The capital costs of aerobic digestion would be about half that of incineration[293].

It is likely that the waste industry will come under the European Union Emission Trading Scheme (ETS) within the next 10 years, in an effort to offset carbon emissions. This would greatly increase the cost of incineration. Two tonnes of carbon are produced for every tonne of waste burned. The present cost per tonne of carbon, under ETS, will be around €20 and this cost will gradually increase, which would add approximately £30 to each tonne of waste burned. Councils will then be committed to paying an escalating cost, starting at £12 million per annum (for a 400,000 tonne a year incinerator) for up to 25 years*. It is a travesty that this cost should fall on local taxpayers subjected to this pollution which they did not ask for and which could be putting their own health at risk. We believe that many councils may be unaware of the implications of Emissions Trading Scheme.

Another consideration councils may be unaware of is the financial impact of Renewable Obligation Certificates. Basically some waste disposal systems will attract these certificates, whilst others will not. The systems that attract ROC credits could produce very significant increases in income. These would be worth millions of pounds per annum for the waste companies operating such plants and for council taxpayers in areas where waste companies operate such equipment on their behalf.

Incinerators generally attract no ROC payments. An exception to this is a CHP (combined heat and power) incinerator which attracts a payment of 1 ROC, or a fraction of an ROC, per megawatt hour of power generated **. Plasma gasification and anaerobic digestion attract a payment of 2 ROCs, or associated fraction, per megawatt watt hour of power generated. These technologies are not only far safer but this payment also makes them a much more attractive financial proposition.

The implication of this is that a 200,000 tonne per year incinerator would attract no payment but a 200,000 tonne per year plasma gasification unit would attract a payment of £4.9 million per annum ***. This would allow the waste company to offer a substantial reduction in their charge to the council for each tonne of waste received. This would, in turn, lead to large savings for both council taxpayers[3].

However, calculation of the total costs of different methods of getting rid of waste must not only include the set-up and running costs but also the environmental, human and health costs. In the case of incineration, human and health costs are substantial but tend to be overlooked because they come out of another budget. However the health costs will have to be paid for and must be included in the equation. Dealing with the ash produced by incinerators represents another major cost to society, which again will come out of someone else's budget. These are not small costs and to give some idea of the magnitude of the costs involved, it was estimated that in 1992 the bill for remediating all the contaminated waste sites in the USA was $750 billion[294].

* Although these charges will be directed at the waste producer, contract clauses protecting them will ensure these high costs are passed on.

** ROC payments related to renewable energy generated by waste facilities are based on the percentage of feedstock that can be classed as renewable. Waste is not a wholly renewable substance and is deemed by Ofgem to contain 50% renewable content. Therefore, only half a megawatt of renewable electricity will be generated when one megawatt overall is generated. As a consequence of this, the megawatt generated will only attract half an ROC.

*** a 200,000 tonne per annum plasma gasification unit would burn 24 tonnes per hour producing 14 megawatts per hour or 122,640 megawatt hours per annum. It is assumed that 50% of this fuel is renewable and hence there will be a rebate of 50% on the 122,640 megawatts of electricity produced (2 ROCs per MWh x 0.5). Each megawatt would attract a payment of approximately £40. This amounts to a saving of £4.9 million pounds per annum.

9.2 Health Costs of Incineration

The health costs of incineration are huge. A 1996 report by the European Commission suggested that for every tonne of waste burnt there would be between £21 and £126 of health and environmental damage, meaning that a 400,000 tonnes per year incinerator would cost the tax-payer between £9,000,000 and £57,000,000 per year[295]: this figure was based on earlier data when emissions to air were somewhat higher so now these costs would be expected to be less. (However note the corresponding increase in costs that is now needed to make fly ash safe. The better the pollution control the more toxic the residues will be and the more expensive they will be to deal with.)

Studies that have tried to estimate the combination of all these costs of incineration have come up with astonishingly high figures. DEFRA's report in 2004 found that the health costs from PM_{10} particulates from incinerators alone, using a central to high estimate, would be £39,245 per tonne of particulates emitted (NB not per tonne of waste burnt)[296]. A 400,000 tonne per year incinerator would produce about 24,000kg (24 tonnes) of particulates per year and the DEFRA estimate of health costs would be £941,000 per annum.

However DEFRA looked at 13 studies of $PM_{2.5}$ and PM_{10} particulates and noted that the health costs ranged from £2,000 - £300,000 per tonne for $PM_{2.5}$s and £1,800 - £226,700 for PM_{10}s. These estimates were based on modelling data which for reasons described in section 12 are likely to underestimate particulate emissions. In particular they do not take into account recent data demonstrating high levels of pollutants emitted during start-up and shut-down. It is therefore reasonable to assume that the actual health costs would be at the higher end of the range, with a cost of £226,700 per tonne for PM_{10}s and £300,000 per tonne for $PM_{2.5}$s giving a total health cost per annum for particulates alone of £6.5 million ****. To give a realistic estimate of the health costs of incineration, the additional costs from the other pollutants must be added to this.

In a review of health costs of incineration Eshet[297] noted the complexity and difficulty of these calculations, with estimates varying between $1.3 and $171 per tonne of waste burnt. A study of British incinerators estimated the cost to be between $2.42 and $13.16 per tonne of waste burnt[298]. Most of these studies do not take into account the cost of ash, the cost of clean-up of accidents or water contamination or the more subtle health effects such as behavioural changes, reduction in IQ, reproductive and hormonal effects which have become apparent in recent years with many pollutants such as lead and organochlorines. For this reason it is likely the costs are considerably higher than estimated. Based on the findings of all these studies we can estimate that a 400,000 tonne a year incinerator will cause millions of pounds worth of health damage annually. These large health costs alone clearly demonstrate that incinerators make a poor choice for waste management. When a single incinerator can generate health costs of many millions of pounds every year, according to the government's own data, it is absurd to argue that incinerators are safe.

It is hard to see any justification for these huge health costs when other methods such as mechanical biological treatment (MBT), aerobic digestion and plasma gasification with low environmental and health costs (see section 8) are available. These methods have not being given sufficient consideration in the UK. MBT is relatively cheap but plasma gasification is more expensive to install. However, if the health costs are taken into account plasma gasification is very much cheaper than incineration. It makes no logical sense to use a method of waste disposal

that has a total cost far in excess of other methods. And we must ask is it morally acceptable to knowingly incur such high health costs.

**** This calculation is as follows. The Quality of Urban Air Review Group has estimated that the $PM_{2.5}$ fraction of total particulates is between 28% and 100%. Leaving aside the likelihood that the $PM_{2.5}$ fraction is higher from incinerator emissions an average figure of 60% $PM_{2..5}$s would be likely. This calculation therefore estimates that a 400,000 tonne incinerator would produce 24 tonnes of particulates, that 60% would be $PM_{2.5}$ particulates at a cost of £4.32 million per annum and 40% would be at the lower cost for other PM_{10}s costing £2.18 million per annum. The total cost in health damage from particulates would therefore be £6.5 million per annum.

9.3 Financial Gains from Reducing Pollution

The EC Okopol report of 1999[299] calculated that every pound spent on pollution abatement saved £6 in health care costs and £4 in social security costs. A report from the US Environmental Protection Agency also reckoned that every dollar spent on abatement saved 10 dollars in health costs.

In addition, a White House study by the Office of Management and Budget in 2003 concluded that enforcing clean air regulations led to reductions in hospitalisations, emergency room visits, premature deaths and lost workdays which led to a saving of between $120 and $193 billion between October 1992 and September 2002. This is an underestimate as it did not look at other health savings such as prescription costs and primary care costs. Few other measures today would give so dramatic a health benefit and such a large saving in health costs[300].

9.4 Other Studies of the Health Costs of Pollution

Recent studies have drawn attention to the huge unanticipated costs to society of pollution from other sources. The International Joint Commission's Science Advisory Board, the Workgroup on Ecosystem Health (SAB-WGEH) looked at a series of health problems where there was hard evidence for environmental causation. Reasoned arguments suggested that the contribution made by toxic substances to these health problems was between 10 and 50%. Four health problems which they considered concern us here, because they involve pollutants similar to those released from incinerators. These are neurodevelopmental defects, hypothyroidism, loss of 5 IQ points and Parkinson's disease. The cumulative costs in the USA for these disorders alone were considered to be between $370 and $520 billion per year. Even using the lowest estimate of environmental contribution (10%), the costs due to pollutants was $40 billion dollars annually[301].

The WWF investigated three conditions ─ mental retardation, cerebral palsy and autism ─ to assess the impact of chemical pollution, and calculated the cost of toxic chemicals on children's brain development to be approximately £1 billion annually[302].

10. Other Considerations of Importance

10.1 The Problem of Ash

The incineration of waste produces a large amount of ash, amounting to 30% of the weight of the original waste; 40-50% of the volume of compacted waste. This is important as landfill sites are becoming less and less available so there is an urgent need for a workable alternative. It is clear that incineration will not solve the landfill problem since it can only reduce the bulk by just under half. Little thought has been

given to this and incinerator operators are still being given 20 to 30 year contracts creating problems for the future.

Incinerators produce two types of ash, bottom ash and fly ash, sometimes called air pollution control (APC) residues. The latter is highly toxic and listed as an absolute hazardous substance in the European Waste Catalogue. It has high concentration of heavy metals and dioxins. Many substances such as metals have little toxicity before incineration but become hazardous once converted to particulates or fine particles in the ash. In fact, the combination of pollutants in the fly ash can amplify the toxicity. Using a biological test, researchers found that the toxicity in fly ash was five times greater than could be accounted for by the content of dioxins, furans and PCBs[303].

There is a basic problem with modern incinerators. The less air pollution produced, the more toxic the ash. Early incinerators emitted large volumes of dioxins. These emissions have been significantly reduced, but at the cost of a corresponding increase in the fly ash, with similar increases in heavy metals and other toxic chemicals. An incinerator burning 400,000 tonnes of waste annually for its 25 years of operation would produce approximately half a million tonnes of highly toxic fly ash[3]. Apart from vitrification, no adequate method of disposing of fly ash has been found. The EU Commission have stated that leaching from landfill sites may be one of the most important sources of dioxins in the future. Heavy metals are known to have high leachability. The US Environmental Protection Agency considers that all landfills eventually leach through their liners. As most of these pollutants are persistent, probably lasting for centuries, they will sooner or later threaten the water table and aquifers where their removal would be near impossible. Allowing this to take place is an abdication of our responsibility to future generations.

In spite of the massive health risks associated with fly ash it is poorly regulated. At Byker, near Newcastle-upon-Tyne, 2000 tonnes of fly ash laden with dioxins was spread over allotments, bridle paths and footpaths for six years between 1994 and 2000. This cavalier approach to managing toxic waste appears to have changed little. In January 2008, a recently permitted hazardous waste site at Padeswood (for storing fly ash from a cement kiln) was flooded. Fortunately no hazardous waste had been stored at the time otherwise it would have carried the toxic waste into brooks and thence into the River Alyn from where drinking water is extracted.

Workers are often exposed to this ash without protective gear. Even today this material has been foolishly used for construction purposes ignoring its toxic properties and the potential for the release of pollutants during use and from ordinary wear and tear.

Fly ash needs to be transported away from the incinerator and this can involve lengthy journeys. These represent an important hazard. An accident could potentially make an area uninhabitable, as happened at Times Beach, Missouri, due to dioxin-contaminated oil. These potential costs have yet to be factored into the cost calculations of incinerators.

Bottom ash is a less severe hazard, but still contains significant quantities of dioxins, organohalogens and heavy metals. It is extraordinary that whereas regulations have tightened in recent years to reduce dioxin emissions to air, bottom ash, which contains 20 times more dioxin, is unregulated and bizarrely is regarded as inert waste. This misclassification had allowed it to be charged at the lowest rate at landfill sites. We believe this is wrong: it is not inert and should not be classified as such. It should be charged at a rate that is in keeping with its toxicity.

The Stockholm Convention makes it clear that dioxins and furans should be destroyed, which currently means using vitrification. In Japan, this is done responsibly and much of the fly ash is now treated by plasma gasification but this essential safety step has been neglected in the UK. Because of the toxicity of bottom and fly ash there should be a full assessment of the cost of a clean-up operation for both water and land contamination. Environmental clean-up costs should be shown as part of the cost of incineration, and, when relevant, of other waste disposal strategies.

10.2 Radioactivity

a) Associated with Incinerators

Over thirty sites in the UK incinerate radioactive waste. Most countries consider this too hazardous.

The majority of radioactive waste incinerated in the UK is alpha or beta emitting radiation. These types of radiation are not very dangerous outside the human body due to their short range (within tissues this is millimetres for alpha particles and centimetres with beta particles), although beta radiation can penetrate the skin. Once incinerated this relatively safe material is converted into a highly dangerous and sinister pollutant. During incineration, billions of radioactive particulates will be formed and emitted into the air. These may be inhaled by anyone unfortunate enough to be downwind at the wrong time, and pass through the lungs and circulation and then into the cells. Once inside the body it will continue to emit radiation. Alpha radiation has a very short range but great destructive power. Both alpha and beta radiation will be highly destructive and carcinogenic to nearby tissues. Each one of the billions of radioactive particulates emitted represents a very real danger. There can be no safe threshold for this material. The risk from this policy is obvious.

Safety regulations bizarrely make no distinction between internal and external radiation even though these are markedly different. For instance Beral found that prostate cancer was higher in workers in the nuclear industry. There was no correlation with external radiation but a highly significant correlation with internal radiation[304]. Animal studies make this even more clear and rats injected with 0.01mGy of Strontium 90 were found to have pathological damage even though the dose was 200 times less than background radiation[305]. Of more concern is the fact that transgenerational effects have also been demonstrated. Mice two generations from a male injected with this Strontium 90 suffered lethal genetic damage, demonstrating that chromosomal damage was passed through the genes to the offspring of irradiated mice[306].

Many people would be surprised to know just how small a dose of radiation is needed to cause harm. After Chernobyl sheep were monitored for Strontium 90 and the limit set was 0.00000000019 grams per kilograms of meat, so small it would be invisible[307]. And yet regulations allow billions of particulates containing similarly minute quantities of radioactive material to be emitted into the air from incinerators. In contrast, natural background radiation is, at most, a minor hazard. For instance Aberdeen has double the level of natural background radiation but no increased risk of leukaemias or cancers.

b) Associated with Other Sites

Increased incidence of leukaemias and cancers around sites releasing radioactive material are well documented. At Seascale a public health enquiry found children were more than ten times more likely to get leukaemia and three times more

likely to get cancer[308,309]. The incidence of leukaemias in children living within 5 kilometres of the Krummel and Goesthact nuclear installations in Germany is much higher than in Germany as a whole. Significantly, the first cases of leukaemia only appeared five years after Krummel was commissioned. At Dounreay there was a sixfold increase in children's leukaemia[310] and at Aldermaston there was also an increase in leukaemias in the under fives[311]. Sharply rising leukaemia rates were noted in five neighbouring towns surrounding the Pilgrim nuclear plant in Massachusetts in the 1980s. It was thought to be linked to radioactive releases from the Pilgrim nuclear plant ten years earlier where there had been a fuel rod problem. 'Meteorological data showed that individuals with the highest potential for exposure to Pilgrim emissions had almost four times the risk of leukaemia compared to those having the lowest potential for exposure'[312,313]. A recent meta-analysis of 17 published reports that covered 136 nuclear sites across the world took a global look at the problem. They found death rates from leukaemia in children under the age of 9 were increased by 21% and in those under 25 by 10%[314]. They noted that discharges from these plants have been too low to account for the leukaemias using standard criteria (based on single or intermittent high dose radiation). The likely explanation here is internal radiation where a minute dose taken internally would be enough to trigger a cancer or leukaemia. This should be seen as a strong warning about the danger of incinerating and dispersing radioactive matter into the environment.

The weight of evidence here strongly suggests that airborne radioactivity is a potent carcinogen and likely to be extremely hazardous. To allow it at all is foolhardy but to combine this with a cocktail of other carcinogens is reckless.

10.3 Spread of Pollutants

The National Research Council, an arm of the National Academy of Sciences, that was established to advise the US government, concluded that it was not only the health of workers and local populations that would be affected by incinerators. They reported that populations living more distantly are also likely to be exposed to incinerator pollutants. They stated "*Persistent air pollutants, such as dioxins, furans and mercury can be dispersed over large regions – well beyond local areas and even the countries from which the sources emanate. Food contaminated by an incinerator facility might be consumed by local people close to the facility or far away from it. Thus, local deposition on food might result in some exposure of populations at great distances, due to transport of food to markets. However, distant populations are likely to be more exposed through long-range transport of pollutants and low-level widespread deposition on food crops at locations remote from an incineration facility.*"[315]

They later commented that the incremental burden from all incinerators deserves serious consideration beyond a local level. This has obvious relevance to the present policy of promoting incinerators in the UK. An important point is that the more toxic smaller particulates, which typically have more toxic chemicals and carcinogens attached, will travel the furthest.[316]

Most chemical pollutants are lipophilic and are therefore not easily washed away by the rain after they settle. When they land on crops they enter the food chain where they bioaccumulate. It has already been admitted that most dioxin in food today in the UK came from the older generation of incinerators. All chemicals capable of entering the food chain will sooner or later reach their highest concentration in the foetus or breast fed infant.

A striking example of the unforeseen and tragic consequences of releasing pollutants into the air has been seen in Nunavut, in the far North of Canada in the Polar Regions. The Inuit mothers here have twice the level of dioxins in their breast milk as Canadians living in the South, although there is no source of dioxin within 300 miles. At the centre of Biology of Natural Systems in Queen's College, New York, Dr Commoner and his team used a computer programme to track emissions from 44,000 sources of dioxin in North America. This system combined data on toxic releases and meteorological records. Among the leading contributors to the pollution in Nunavut were three municipal incinerators in the USA[317,318].

10.4 Cement Kilns

Although this report is primarily about incinerators it is useful to compare incinerators with cement kilns. Both produce toxic emissions of a similar type and much of the report is relevant to both. Cement kilns convert ground limestone, shale or clay into cement. They require large quantities of fuel to produce the high temperatures needed and this lends itself to the use of non-traditional fuels such as tyres, refuse-derived fuel and industrial and hazardous wastes variously called Cemfuel, secondary liquid fuel (SLF) and recycled liquid fuel (RLF).

However, pollution and planning controls are significantly weaker than those for hazardous waste incinerators. Cement kilns produce a number of toxic emissions similar to incinerators. Burning tyres produces emissions with dioxins and zinc and burning petroleum coke produces vanadium and nickel. Releases of mercury and arsenic are uncontrolled as these are vapourised. The risk from dioxins is considerably greater as most cement kilns do not have the activated charcoal needed to remove them.

The risk from $PM_{2.5}$ particulates is extremely serious. The limit set for the weight of all particulates emitted by incinerators is 10mg per cubic metre. However cement kilns are allowed to emit 30-50 mg per cubic metre. This would be excessive by itself but the volumes of emissions from cement kilns can be up to five times greater than incinerators. Therefore some cement kilns can produce emissions of particulates and other toxic substances which are in excess of 20 times that of incinerators under normal operating conditions. Worse still they have poorer abatement equipment and usually lack the activated charcoal needed to reduce emissions of metals and dioxins.

The electrostatic precipitators need to be shut off when carbon monoxide levels build up due to the risk of explosion. This leads to unabated emissions. This has happened 400 times a year in one plant. The quantities of particulates released at these times are immense reaching 20,000mg per cubic metre which are the highest level that can be measured. Recent research has demonstrated unequivocally that small increases in $PM_{2.5}$ particulates will increase cardiovascular and cerebrovascular mortality, so to allow releases of this order therefore borders on the negligent. Incredibly $PM_{2.5}$ particulates are not routinely measured.

Independently-audited monitoring by a registered charity at one cement kiln in the UK has continuously recorded levels of particulates, using 15 minute average readings[319]. They have found extremely high surges of particulates, typically with peak readings occurring at night, sometimes several times a week, with maximum PM_{10} particulates reaching levels of over 4500 μg per cubic metre and maximum $PM_{2.5}$ reaching over 170μg per cubic metre. Current scientific knowledge on particulates suggests that these levels would be expected to cause cardiovascular deaths and the findings demonstrate the urgent need for independent monitoring

around all cement kilns. This monitoring has exposed major deficiencies in the present monitoring and regulatory system.

Thermal treatment of hazardous waste is always a highly dangerous activity and the very best available technology needs to be used. Cement kilns are effectively being used to burn hazardous waste on the cheap. Sadly hazardous waste typically finds its way to the least regulated and cheapest disposal methods, in practise those that create the most health risks and the most environmental damage.

Cement kiln technology has remained virtually unchanged since the turn of the twentieth century. They can only be refitted or retrofitted to a minimal degree to improve efficiency and toxic waste destruction. The Select Committee for the environment recommended studies on the safety of cement kilns over 10 years ago and this has been ignored. Why?

Cement kilns are therefore capable of extremely serious health consequences. Incredibly some of these cement kilns have been sited in the middle of towns where they would be expected to have a major effect on the health of the local population. The fact that they are allowed at all is astonishing, for the maximum impact will inevitably be on the most vulnerable members of society, and in particular the unborn child.

11. Monitoring

At the heart of the problems with incineration is the poor quality and unsatisfactory nature of monitoring at these installations, unsatisfactory in the way it is done, the compounds monitored, and the levels deemed acceptable, and the lack of monitoring of body burdens in the local population. The problems are as follows:

Very Few Pollutants are being measured

Out of the hundreds of chemicals released from an incinerator only a tiny proportion are measured. On current data, the three most important pollutants released by incinerators are dioxins, heavy metals and $PM_{2.5}$ particulates. Incredibly these are virtually unmonitored. Only half a dozen pollutants are measured continuously in the stack and about another half dozen are measured occasionally (usually 6 monthly for the first year and then yearly) by spot monitoring – these include heavy metals and dioxins. This is clearly unsatisfactory and since waste operators are warned in advance of a visit, they are handed an opportunity to change to burning cleaner waste which is unrepresentative of the toxic risk, making the exercise largely pointless.

The Most Dangerous Pollutants are hardly being Monitored

Accidental by-passing of pollution control devices by incinerators present very real dangers to people living in the vicinity of incinerators and this danger is compounded by the near absence of monitoring of dioxins. Two episodes serve to illustrate this. A modern state of the art incinerator in Rotterdam was found to be by-passing its pollution control devices 10% of the time emitting dioxins equivalent to 5 times the national limit over the city. In Norfolk, Virginia a similar incident led to dioxin emissions greater than the allowable combined limits for traffic, incinerators and industry for Sweden, Germany and the Netherlands combined. This would cause widespread pollution of an area with dioxin and other persistent pollutants that could last for decades, if not centuries, putting many generations at risk.

Start-ups and shut downs of incinerators give rise to a similar danger. A recent study found that a single incinerator start-up would, on average, generate, *over a 48 hour period*, 60% of the total *annual* dioxin emissions produced during steady state

conditions – in other words 7 months worth of dioxin release within 2 days of a typical start-up. They also found that the levels of dioxins produced by start-ups at some of the incinerators they studied could be twice the annual dioxin emissions under steady state conditions (this is the equivalent of 24 months of dioxin release within 2 days)[320]. The danger to people living in the area is obvious and serious. High levels of dioxins can also be produced during shut-downs and during commissioning (when they are not monitored).

Dioxins are only monitored at 3-12 month intervals and then only for a few hours. This means that dioxins are not monitored 99% of the time. It could therefore be many months before high levels of dioxin emissions were detected perhaps allowing enough dioxin to be released to threaten the health of a whole community and render farms in the vicinity unfit for growing vegetables or rearing livestock. In fact, the operator and the public might never find out and then steps would never be taken to deal with the consequences.

An added problem is that spot monitoring (as is used currently) has been shown in a recent study to be unrepresentative and to underestimate dioxin levels by 30-50 times[321]. The situation is no better with heavy metals. Like dioxins, they are unmonitored for 99% of the time.

Clearly, continuous dioxin monitoring is essential and without such monitoring, incinerators must be regarded as unsafe and a hazard to anyone living in the area. Continuous dioxin monitoring should be mandatory as is the case in some other European countries**.** Currently, monitoring of the three most important and dangerous pollutants, namely dioxins, heavy metals and $PM_{2.5}$ particulates is virtually non-existent in the UK. In the case of $PM_{2.5}$ particulates they are not monitored at all – only the far less relevant PM_{10} particulates.

Independent monitoring of cement kilns has already demonstrated very high particulate emissions that could seriously endanger health[319]. These releases have been frequent (sometimes 3 times a week), dangerous (reaching 4500µg per cubic metre of PM_{10} particulates) and have escaped detection by the regulatory authorities. Clearly, the present regulatory system is not protecting the public.

The Standard of Monitoring on the Ground is also Unacceptable

In addition to monitoring in the stack, there is a requirement to monitor pollutants in the surrounding air. This is normally done by the local council with monitors at ground level. However this is also unsatisfactory. For instance to monitor for safe levels of particulates it would require at least 24 monitors placed at strategic points around an incinerator (assuming the wind is distributed evenly) to achieve a 25% sampling rate, which is the minimum that can be considered acceptable[3]. Typically, there are less than three monitors around most incinerators today. Measurement of heavy metals in the surrounding air, with the exception of lead, is not even required.

No Monitoring of Pollutants which have accumulated in the Neighbourhood

Measuring concentration of pollutants released in the stack gives no information about the levels of toxic material that have accumulated in the vicinity. When the rate of discharge of pollutants into the environment is greater than the ability of the ecosystems to break them down then they must accumulate. We already know that many do not break down for centuries. The excretion rates of many pollutants from the human body are also very poor, for example the *half life* of cadmium in the body is 30 years and for PCBs it is 75 years. Many pollutants, being

fat soluble, will bio-accumulate in living matter at far high concentrations than in the ambient air. A US EPA memo admitted that the risk from accumulation of dioxin in farm animals "could result in unacceptable health risks". Using a type of risk assessment called screening analysis[322] they calculated that dioxin would accumulate in cattle downwind from an incinerator and that the risk from beef and milk consumption would be 40,000 times the risk from inhalation. This is a massive increase in risk and is in keeping with what we already know about bioaccumulation in other species (see Section 3.4). Monitoring of dioxins in cattle and other farm animals regularly is essential for these reasons. Regrettably it is not being done and therefore consumers of these products are being put at risk. Checks for pollutants in dust, vegetation and in the bodies of local inhabitants are also necessary.

It is sometimes argued that these pollutants don't matter as they will be carried away in the wind and be someone else's problem. Sadly this is partly true and that is the reason there is so much pollution in the fragile ecosystem in the Arctic where much of the toxic material ultimately ends up.

Monitoring relies on Safety Data derived from Animal Studies

Animal studies commonly underestimate human vulnerability because of the obvious difficulty in testing cognitive, behavioural and language deficiencies and conditions such as fatigue. In the case of lead, mercury and PCBs, animal studies have underestimated the neurotoxic effect on humans by a factor of 100 to 10,000 times[285].

Monitoring Gives Little Protection to the Foetus

Average levels or spot monitoring ignores exposures at critical times. The timing of the exposure is often more important than the concentration. Exposures at critical times during foetal growth or infancy are known to produce more serious effects than similar exposures in adulthood and this damage can be permanent. This is well recognised, especially with lead, mercury and PCBs. None of the safety limits has been demonstrated to protect against foetal damage. We know from animal and human studies that toxins have the greatest impact on the foetus and young child. The most vulnerable members of the community are likely to bear the brunt of these toxic releases.

Many Pollutants have No Safe Threshold or show Low Dose Toxicity

Some pollutants such as $PM_{2.5}$ particulates, lead and dioxin have no safe thresholds. Most organochlorines are endocrine disruptors and thresholds may not exist for these effects. Monitoring gives little or no protection in these situations. Sometimes low dose studies have shown toxic effects at levels far below the "no effect" level in high dose studies. An example of this is bisphenol A, a plasticizer. Studies showed health effects at levels 2,500 times lower than American EPA's lowest observed effect, with adverse outcomes including aggressive behaviour, early puberty and abnormal breast growth[220]. Perchlorate produces changes in the size of parts of the brain at 0.01 mg/kg/day but not at 30mg[220]. Aldicarb was found to suppress the immune system more at 1 ppb than it did at 1000ppb. Other chemicals also produce different effects at low dose to what they do at high dose. This shows how very little we know about the dangers of exposing whole populations to chemical pollution.

Pollution Offences are Commonplace and Regulation is Poor

Ten incinerators in the UK committed 553 pollution offences in a two year period, documented in Greenpeace's "A Review of the Performance of Municipal Incinerators in the UK". This appalling record led to only one prosecution by the Environment Agency. There is little point in tighter regulations if they are not enforced. Fines received for pollution offences have been compared to a person on a £50,000 salary receiving a £20 parking fine. This clearly gives waste companies a green light to ignore regulations and pollute with little fear of the consequences. The above data was based on self assessment by the companies concerned.

Levels of emissions achieved under test conditions or when inspections occur by prior arrangements are likely to be far lower than under real life conditions. This was demonstrated in the United States in 1990 when the EPA and Occupational Safety and Health Administration conducted 62 unannounced visits and no less than 69% of inspections led to summons for violations of regulations[323]. (In the UK inspections are by prior arrangement). This makes a strong case for making all visits unannounced.

When an environmental group investigated an incinerator in Indianapolis the situation was even worse. They found it had violated its permits 6,000 times in two years and bypassed its own air control pollution devices 18 times.

In effect, incinerators present inherent and unavoidable hazards to public safety but the extent of the hazards depends on how well incinerators are run. The evidence is strong that they are often run badly. The situation is made worse by weak regulators with little appetite for enforcing public safety.

12. Risk Assessment

One might reasonably expect that, when the decision to build an incinerator is made, all the above information would be carefully taken into account. Sadly this is not necessarily the case. Directors of Public Health, who usually have little knowledge of environmental health, are asked to write an IPPC (Integrated Pollution Prevention and Control) Application Report and give their opinion on the health risks from the proposed incinerator. Typically this decision is based on an inexact method called risk assessment. They tend to rely almost exclusively on this type of assessment and often have little understanding of its limitations.

Risk assessment is a method developed for engineering but is very poor for assessing the complexities of human health. Typically it involves estimating the risk to health of just 20 out of the hundreds of different pollutants emitted by incinerators. It masquerades as a scientific measure but has all the hallmarks of pseudoscience. By pseudoscience we mean assumptions based on false premises:

1) **It makes the assumption that any substance emitted but not assessed (this means 99% of all pollutants) should be treated as if they have zero risk**. This assumption is obviously untrue.
2) **It assumes wrongly that all pollutants have thresholds below which they are safe.** Science contradicts this. Many pollutants, including dioxins, lead and radioactive particulates do not have thresholds and some may even be more dangerous at lower concentrations (see section 11). An international meeting of neurologists and endocrinologists concluded "Chemical challenges in early life can lead to profound and irreversible abnormalities in brain development at exposure levels that do not produce permanent effects in an adult; there may not be definable thresholds for response to endocrine disruptors"[324]. The

National Research Council concluded in 1992 that "the assumption of thresholds for neurotoxicity was biologically indefensible"[225].

We might also note that the accepted thresholds for many pollutants have been progressively reduced over the last few decades (including vinyl chloride, ethylene dichloride and six chlorinated solvents) with reductions to between one half and one tenth of the original limits. We can expect further reductions as science progresses.

3) **It assumes wrongly that only air emissions need to be considered and bioaccumulation in food can be ignored.** However air emissions may be only the tip of the iceberg. Most food today is contaminated with dioxins, predominantly from past incinerator emissions. As noted in section 11, a leaked report in 1993 from the US Environmental Protection Agency calculated that dioxin would accumulate in cattle in a farm downwind of an incinerator in Ohio posing a risk to the frequent beef consumer which was 40,000 times higher than from inhalation alone. If the incinerator operated for 30 years the cancer risk from eating this beef regularly was calculated to be a massive 1,200 per million, far beyond acceptable risk[322]. We can assume this sort of risk from food produced near most incinerators occurs routinely and yet it is being sold to the public and regulators are turning a blind eye to the danger.
4) **It misconstrues lack of evidence on the danger of pollutants as evidence of safety**. The toxic effects of 88-90% of chemicals and pollutants are unknown[325]. It is impossible to assess the risk of substances we barely understand. This is particularly true in relationship to birth and developmental defects. Many pollutants have not even been characterised, let alone assessed for risk.
5) **It assumes that health effects such as infertility, immune suppression, altered behaviour and reduced intellectual capacity which are not included in the risk assessment can be ignored**. However there is ample and increasing evidence that many pollutants have just these impacts.
6) **It assumes wrongly that ecosystems have the ability to absorb and degrade all environmental pollutants**. Again science contradicts this: many pollutants are known to be persistent and bioaccumulative. In fact, if the rate of input, however small, is greater than the rate at which they break down they must accumulate. It is equivalent to filling up a bucket under a slow dripping tap: sooner or later the water will overflow unless the source of water is stopped.
7) **It assumes wrongly that the hazard posed by each individual compound tested out of context and in isolation can predict the hazard of complex mixtures of chemicals.** In the real world pollutants typically occur in combinations and abundant evidence now exists that increased toxicity is common with multiple exposures.
8) **It assumes wrongly that the cumulative pollution burden of all the emissions produced by all these facilities can safely be ignored and each facility can be considered in isolation.** It is this type of limited thinking that has led to the contamination of entire ecosystems such as the Great Lakes, Baltic Sea, Mediterranean and Arctic. These pollutants pose global and multigenerational threats to health and ecosystems.
9) **It assumes wrongly that we have a comprehensive understanding of the complexity of biological processes and chemical toxicity when in reality**

there are vast information gaps. This is why we have been constantly surprised by unpleasant discoveries like endocrine disruption and high body burdens in newborns.

10) **It wrongly assumes all people will react in the same way** to pollutants and in particular ignores the fact that the foetus is at far greater risk.

Hidden within this type of assessment is a value judgement about what is an acceptable level of risk[326] and this is not made explicit. For instance what is an acceptable number of birth defects and who is it acceptable to? A cancer risk of 1 per million is typically considered acceptable but may not be acceptable to the person affected by the cancer.

Risk assessment usually involves "modelling"; – dispersion models use an estimation of exposure data, rather than actual exposure data, to assess the impacts of pollutants and their likely distribution. These reports are typically produced by the polluter. The models are not accurate - modelling has a 30% confidence level – this means this technique has only a 30% chance of accurately predicting the ground level concentrations of pollutants - in other words less accurate than tossing a coin. Only about half the predictions are within a factor of two of actual (observed) concentrations and the rest are even less accurate. The models attempt to predict a worst case scenario but the models cannot accurately represent real worse case scenarios which typically occur when there is little or no wind leading to a build-up of pollutants. This means real worst case scenarios can be much worse than predicted[327]. Different models can give very different results.

In addition, present modelling methods are not only inaccurate in estimating ground level pollutant concentration once emitted but they also seriously underestimate the quantities of pollutants emitted. In particular, modelling almost never takes into account secondary particulates formed as the products of combustion rise up the stack. These secondary particulates can double the total volume of particulates (see section 2.1).

Modelling produces the illusion of a scientific knowledge and a certainty that is entirely unjustified by the imprecise nature of modelling and it is based on substantial scientific uncertainty and limited scientific data. It produces a mass of complex mathematical data, which implies unjustified precision, and it is difficult for people not familiar with the mathematics to disentangle the inaccuracies. This was summed up by the head of the EPA Carcinogen Assessment Group, Roy Albert, when he said "Individuals with very different institutional loyalties can produce very different risk assessments from the same materials, where large uncertainties exist." In other words it is very easy to bias it towards the waste operator. It is often treated by regulators[328] and Directors of Public Health as if it was an accurate assessment. In spite of these severe limitations it is extensively used.

These risks assessments have almost always concluded that incinerators are safe which flies in the face of epidemiological data which shows the opposite. It also flies in the face of the history of chemical use. The latter is littered with examples of chemicals once said to be safe which were later found to have devastating and unanticipated effects, often beyond the worst case scenario (eg DDT, PCBs, CFCs) (see section 7.2).

13. Public Rights and International Treaties

In 2001 the United Nations Commission on Human Rights stated that "*everyone has the right to live in a world free from toxic pollution and environmental degradation*".

It is unethical that people should die from the emissions from incinerators when safe alternatives are available and for this reason incineration violates Article 2 of the European Human Rights Convention, the Right to Life.

The Stockholm Convention, agreed to by over 100 countries including Britain, in 2001, commits countries to eliminating persistent organic pollutants, including PCB, dioxins and furans, calling for countries to prevent not just the *release* of these pollutants but also their *formation*. The formation of these substances is an inevitable consequence of the use of incinerators. The Convention also requires parties to take measures to reduce the ***total releases*** of these substances (which includes releases to fly ash). It identifies incinerators as primary sources of these compounds. Incineration is, in all these ways, a flagrant violation of the Stockholm convention.

Incineration is also a violation of the Environmental Protection Act of 1990 which states that the UK must prevent emissions from harming human health.

14. Conclusions

1) **Incineration does not remove waste**. It simply converts it into another form (gas, particulates, ash) and these new forms are typically more hazardous though less visible than in the original form.
2) **Large epidemiological studies have shown higher rates of adult and childhood *cancers* and of *birth defects* around incinerators**. Smaller studies and a large body of related research support these findings, point to a causal relationship, and suggest that a much wider range of illnesses may be involved.
3) **Recent research has confirmed that particulate pollution, especially the *fine particulate* ($PM_{2.5}$) pollution, which is typical of incinerator emissions, is an important contributor to *heart disease, lung cancer*, and an assortment of other diseases, and causes a *linear increase in mortality*.** The latest research has found there is a much greater effect on mortality than previously thought and implies that incinerators will cause increases in cardiovascular and cerebrovascular morbidity and mortality with both short-term and long-term exposure. Particulates from incinerators will be especially hazardous due to the toxic chemicals attached to them.
4) Other pollutants emitted by incinerators include heavy metals and a large variety of organic chemicals. **These substances include known carcinogens, endocrine disruptors, and substances that can attach to genes, alter behaviour, damage the immune system and decrease intelligence.** There appears to be no threshold for some of these effects, such as endocrine disruption. The dangers of these are self-evident. Some of these compounds have been detected hundreds to thousands of miles away from their source.
5) The danger of incinerating radioactive waste deserves special mention. Incineration converts radioactive waste into billions of radioactive particulates. These particulates make a near perfect delivery system for introducing the radioactive matter into the human body, where it can then act as an internal emitter of alpha or beta radiation. **This type of radiation is qualitatively different, far more dangerous and far more sinister, than background**

radiation. There can be no justification for using this method of dealing with radioactive waste.

6) **Modern incinerators produce fly ash which is much more toxic than in the past,** containing large quantities of dioxin-rich material for which there is no safe method of disposal, except vitrification, a method not being used in the UK. Disposal of incinerator ash to landfill sites is associated with long-term threats to aquifers and water tables and the potential for accidents serious enough to require evacuation of an area.
7) **The risks to local people that occur when incinerators operate under non-standard working conditions have not been addressed,** particularly the emissions at start-up and shutdown which may be associated with the release, within 2 days, of more dioxin than over 6 months of working under standard conditions.
8) **The greatest concern is the *long-term* effects of incinerator emissions on the developing embryo and infant, and the real possibility that genetic changes will occur and be passed on to succeeding generations.** Far greater vulnerability to toxins has been documented for the very young, particularly foetuses, with risks of cancer, spontaneous abortion, birth defects or permanent cognitive damage. A worryingly high body burden of pollutants has recently been reported in two studies of cord blood from new-born babies.
9) **Waste incineration is prohibitively *expensive* when health costs are taken into account.** A variety of studies, including that from the government, indicate that a single large incinerator could cost the tax payer many million of pounds per annum in health costs. Put simply, the government's own data is demonstrating that incinerators are a major health hazard. With the predicted inclusion of the waste industry within the EU European Emissions Trading Scheme, local taxpayers, in areas with incinerators, will not only have to live within a polluted area but will be saddled with costs, under ETS, of millions of pounds per annum to pay for it.
10) **Waste incineration is unjust because its maximum toxic impact is on the most vulnerable members of our society, the unborn child, children, the poor and the chemically sensitive.** It contravenes the United Nations Commission on Human Rights, the European Human Rights Convention (the Right to Life), and the Stockholm Convention, and violates the Environmental Protection Act of 1990 which states that the UK must prevent emissions from harming human health.

15. Recommendations

1) The safest methods of waste disposal should be used.

2) Health costs should be routinely taken into account when deciding on waste disposal strategies.

3) The present limited method of risk assessment by which the safety of proposed installations is judged, is inadequate, can easily be biased towards the waste operator, cannot be relied on, and should be reviewed.

4) Tackling the problems of both the amount and the nature of waste generated is of critical importance, with the emphasis on reducing the production of waste, and on recycling.

5) The serious health consequences of fine particulate pollution have become apparent in the last ten years: incinerators are a significant source and, for this reason alone, in our considered opinion, incineration is the least preferred option for getting rid of waste. Taking into consideration all the information available, including research indicating that there are no safe levels for fine particulates, the increasing amount of plastic and related substances in the waste stream and the highly toxic ash produced by modern incinerators, we can see no reason to believe that the next generation of incinerators would be substantially safer than the previous ones.

6) **Far safer alternative methods are now available** including recycling, mechanical biological treatment, aerobic digestion and plasma gasification: a combination of these would be safer, would produce more energy, would be cheaper than incineration in the long run and would be much cheaper when health costs are taken into account. Thermal methods should only be used for residual, non-recyclable waste and the safest thermal method should be chosen: currently this is plasma gasification. This not only produces more energy but can use plastics as a resource. These more advanced methods should be employed.

7) **This report draws attention to the many deficiencies and poor quality of the present monitoring procedures.** We recommend the introduction of a far stricter and more comprehensive system for the monitoring of all waste-burning plants by a fully independent body, including random unannounced visits: the monitoring should include:

a) **Continuous monitoring of dioxins – this is an absolute essential and, not surprisingly, is mandatory in some countries.** This vital step is essential because of the extremely toxic nature of the pollution emitted when incinerator pollution control devices are by-passed. The UK should not have the second rate safety standards that they have at present.

b) Continuous monitoring of $PM_{2.5}$ particulates and monitoring of PBDEs.

c) A comprehensive system of monitors set up by Councils around all incinerators to measure particulates and heavy metals.

d) **Monitoring of dioxin in all livestock within a 5 mile radius of incinerators due to the known and serious risk from bioaccumulation in food**.

e) Periodic monitoring of the heavy metals and dioxins in the fly ash

f) A programme of monitoring the body burdens of some key pollutants in local inhabitants.

g) Periodic monitoring of the content of dust in homes in the locality

8) It is particularly important that incinerators should not be sited in deprived areas or areas with high rates of mortality where their health impact is likely to be greatest. This can only add to health inequalities. (NB. Presently 9 out of 14 incinerators have been built in the most deprived 20% of wards[329]).

9) The present subsidies and tax advantages, which favour incineration, should be removed. A ban or tax on recyclable material going to incinerators or landfill deserves

serious consideration. It is nonsense to regard bottom ash, with its significant dioxin content, as an inert substance and it should incur landfill tax at a higher rate.

10**) We recommend that no further waste incinerators be built**.

References:

Section 2. Emissions from Incinerators and other Combustion Sources

2.1 Particulates

1) EC (1998) Proposal for a Council Directive on the incineration of waste. Brussels 07.10.1998 COM (1999) 558final. 98/0289 (SYN).
2) Howard C V (2000) *In* Health Impacts of Waste Management Policies. Hippocrates Foundation, Kos, Greece 12-14 Nov 1998. Academic Publishers.
3) Personal communication, Peter Rossington BSc (Hon), MRSC, Chemical Consultant. 2005.
4) Espinosa AJ, Rodriquez MT, Barragan de la Rosa FJ et al. Size distribution of metals in urban aerosols in Seville (Spain). Atmos Environ 2001; 35: 2595-2601.
5) Baek SO, Field RA, Goldstone ME et al. A review of atmospheric polycyclic aromatic hydrocarbons: sources, fate and behaviour. Water, Air Soil Pollution, 1991; 60: 279-300.
6) Pistikopoulos P, Mascelet P, Mouvier G. A receptor model adapted to reactive species – polycyclic aromatic hydrocarbons - evaluation of source contributions in an open urban site. Atmos Environ A-Gen 1990; 24: 1189-97.
7) Venkataraman C, Friedlander SK. Source resolution of fine particulate polycyclic aromatic hydrocarbons – using a receptor model modified for reactivity. J Air Waste Management; 1994; 44: 1103-08.
8) Zmirou D, Masclet P, Boudet C, Dechenaux J. Personal exposure to atmospheric polycyclic hydrocarbons in a general adult population and lung cancer assessment. J Occup Environ Med 2000; 42(2): 121-6.

2.4 Organic Pollutants

9) Kerkvliet NI. Immunotoxicology of dioxins and related compounds. *In* Schecter, Dioxins and Health p 199-225.
10) Whyatt RM, Santella RM, Jedrychowski W et al. Relationship between ambient air pollution and DNA damage in Polish mothers and newborns. Environ Health Perspect, 1998; 106 Suppl 3: 821-6
11) Porterfield SP. Vulnerability of the developing brain to thyroid abnormalities and environmental insults to the thyroid system. Environ Health Perspect 1994; 102 Supp 2: 125-30.
12) Peters JM, Thomas D, Falk H et al. Contribution of metals to respiratory cancer. Environ Health Perspect 1986;70: 71-83.
13) Gottscalk LA, Rebello T, Buchsbaum MS et al. Abnormalities in hair trace elements as indicators of aberrant behaviour. Comp Pyschiatry 1991; 32 (3): 229-37.
14) Tong S, Baghurst P, McMichael A et al. Lifetime exposure to environmental lead and children's intelligence at 11 – 13 years: the Port Pirie Cohort Study. BMJ 1996; 312 (7046): 1569-75.
15) Sedman RM, Esparza JR. Evaluation of the public health risks associated with semivolatile metal and dioxin emissions from hazardous waste incinerators. Environ Health Perspect 1991; 94: 181-7.
16) Ericksson P, Jakobsson E, Fredriksson A. Brominated flame retardants: A novel class of developmental neurotoxicants in our environment? Environ Health Perspect, 2001; 109(1): 903-908.

17) Olsson P-E, Borg B, Brunstrom B, Hakansson H, Klasson-Wehler E. Endocrine disrupting substances. ISBN 91-620-4859-7, Swedish EPA, Stockholm 1998.

Section 3. Health effects of Pollutants

3.1 Particulates

18) WHO Air Quality Guidelines, 1999, Chapter 3.
19) Dockery DW, Pope Ca 3rd, Xu X et al. An association between air pollution and mortality in six US cities. N Eng J Med 1993; 329(24): 1753-9.
20) Pope CA, Thun MJ, Namboodiri MM et al. Particulate air pollution as a predictor of mortality in a prospective study of US adults. Am J Respir Crit Care Med 1995; 151 (3 pt 1): 669-74.
21) de Hartog JJ, Hoek G, Peters A, et al. Effects of fine and ultrafine particles on cardiorespiratory symptoms in elderly subjects with coronary heart disease: the ULTRA Study. Am J Epidemiology 2003; 157(7): 613-23.
22) Nemmar A, Hoet PH, Vanquickenborne B et al. Passage of inhaled particles into the blood circulation in humans. Circulation 2002; 105(4): 411-4.
23) Maynard RL, Howard CV, Air Pollution and Health, London: Academic Press 1999: 673-705.
24) Ponka A, Virtanen M. Asthma and air pollution in Helsinki. J Epidemiol Community Health 1996; 50 Suppl 1: s59-62.
25) Particulate Matter: Properties and Effects upon Health, BIOS Scientific Publishers Ltd, Oxford p 63-84.
26) Airborne Particulate Matter in the United Kingdom. Third Report of the Quality of Air Review Group (QUARG) May 1996, ISBN 0 9520771 3 2.
27) Goldberg MS, Burnett RT, Bailar JC et al. The association between daily mortality and ambient air particle pollution in Montreal, Quebec. 2. Cause-specific mortality. Environ Res 2001: 86(1): 26-36.
28) Pope CA, Burnett RT, Thun MJ, et al. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA 2002; 287(9): 1132-41.
29) Whitman v American Trucking Assoc Inc 532 US 457 (2001).
30) Re-analysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality: Special Report. Cambridge, Mass: Health Effects Institute July 2000, led by Dr Daniel Kreweski.
31) Ostro B, Chestnut L. Assessing the benefits of reducing particulate matter and pollution in the United States. Environ Res 1998; 76(2): 94-106.
32) Pope CA, Bumett RT, Thurston GD et al. Cardiovascular Mortality and Long-Term Exposure to Particulate Air Pollution: Epidemiological Evidence of General Pathophysiological Pathways of Disease. Circulation 2004; 109: 71-77.
33) Miller KA, Siscovick DS, SheppardL, et al. Long term exposure to air pollution and incidence of cardiovascular events in women. N Eng J Med 2007, 356:447-458
34) Peters A, Dockery DW, Muller JE et al. Increased particulate air pollution and the triggering of myocardial infarction. Circulation 2001; 103 (23): 2810-5.
35) Hong YC, Lee JT, Kim H, Kwon HJ. Air pollution: a new risk factor in ischemic stroke mortality. Stroke 2002; 33(9): 2165-9.
36) Hoek G, Brunekreef B, Fischer P et al. The association between air pollution and heart failure, arrhythmia, embolism, thrombosis and other cardiovascular causes of death in a time series. Epidemiology 2001; 12(3): 355-7.
37) Maheswaran R, Haining RP, Brindley P et al. Outdoor air pollution and Stroke in Sheffield, United Kingdom, Small-Area Geographical Study. Stroke 2005; 36(2): 239-43.
38) Schwartz J. Air pollution and hospital admissions for heart disease in eight US counties. Epidemiology 1999; 10(1): 17-22.
39) Baccerelli A, Martinelli I, Zanobetti A et al. Exposure to particulate air pollution and risk of deep venous thrombosis. Arch Int Med 2008; 168(9):920-7

40) Zanobetti A, Schwartz J. Cardiovascular damage by airborne particles: are diabetics more susceptible? Epidemiology 2002; 13(5): 588-92.
41) Peters A, Liu E, Verrier RL et al. Air pollution and incidence of cardiac arrhythmia. Epidemiology 2000; 11(1): 11-7.
42) Pekkanen J, Peters A, Hoek G, et al. Particulate air pollution and risk of ST segment depression during submaximal exercise tests among subjects with coronary heart disease: the Exposure and Risk Assessment for Fine and Ultrafine Particles in Ambient Air (ULTRA) study. Circulation 2002; 106: 933-38.
43) Goldberg MS, Burnett RT, Bailar JC 3rd et al. Identification of persons with cardiorespiratory conditions who are at risk of dying from the acute effects of ambient air particles. Environ Health Perspect 2001; 109 Supp 4: 487-94.
44) Perera FP, Tang D, Tu YH et al. Biomarkers in maternal and newborn blood indicate heightened fetal susceptibility to procarcinogenic DNA damage. Environ Health Perspect 2004; 112(10): 1133-6.
45) Jedrychowski W, Bendkowska I, Flak E et al. Estimated risk for altered fetal growth resulting from exposure to fine particles during pregnancy: an epidemiologic prospective cohort study in Poland. Environ Health Perspect 2004; 112(14): 1398-1402.
46) Perera FP, Rauh V, Whyatt RM et al. Molecular evidence of an interaction between prenatal environmental exposures and birth outcomes in a multiethnic population. Environ Health Perspect 2004; 112(5): 626-30.
47) Somers CM, McCarry BE, Malek F et al. Reduction of particulate air pollution lowers the risk of heritable mutations in mice. Science 2004; 304(5673): 1008-10.
48) Burden of disease attributable to selected environmental factors and injury among children and adolescents in Europe (no authors listed). Child Care Health Dev 2004; 30(6): 731-732.
49) Morgan G, Corbett S, Wlodarczyk J. Air pollution and hospital admissions in Sydney, Australia, 1990-1994. Am J Public Health 1998; 88(12): 1761-60.
50) Vichit-Vadakan N, Ostro BD, Chestnut LG et al. Air pollution and respiratory symptoms: result from three panel studies in Bangkok, Thailand. Environ Health Perspect 2001; 109 Supp3: 381-7.
51) Dockery DW, Speizer FE, Stram DO et al. Effects of inhalable particles on respiratory health of children. Am Rev Respir Dis 1989; 139(3): 587-94.
52) Brauer M, Hoek G Van Vliet P et al, Air pollution from traffic and the development of respiratory infections and asthmatic and allergic symptoms in children. Am J Respir Crit Care 2002; 166(8): 1092-8.
53) Seaton A, MacNee W, Donaldson K et al. Particulate air pollution and acute health effects. Lancet 1995; 345(8943): 176-8.
54) Boezen HM, van der Zee SC, Postma DS et al. Effects of ambient air pollution on upper and lower respiratory symptoms and peak expiratory flow in children. Lancet 1999; 353 (9156): 874-8.
55) Gilliland FD, Berhane K, Rappaport EB et al. The effects of ambient air pollution on school absenteeism due to respiratory illness. Epidemiology 2001: 12(1): 43-54.
56) Peters A, Dockery DW, Heinrich J, Wichmann HE. Short term effects of particulate air pollution on respiratory morbidity in asthmatic children. Eur Respir J 1997; 10(4): 872-9.
57) Gauderman WJ, McConnell R, Gilliland F et al. Association between air pollution and lung function growth in Southern Californian children. Am J Respir Crit Care Med 2000; 162 (4 Pt 1); 1383-90.
58) Brunekreef B, Hoek G. The relationship between low-level air pollution and short-term changes in lung function in Dutch children. J Expo Anal Environ Epidemiol 1993; 3 Suppl 1: 117-28.
59) Gauderman WJ, Gilliland GF, Vora H, et al. Association between air pollution and lung function growth in Southern Californian children: results from a second cohort. Am J Respir Crit Care Med 2002; 166(1): 76-84.
60) Samet JM, Dominici F, Curriero FC et al. Fine particulate air pollution and mortality in 20 US cities 1987-1994. N Eng J Med 2000; 343(24): 1742-9.

61) Schwartz J, Laden F, Zanobetti A. The concentration-response relation between PM2.5 and daily deaths. Environ Health Perspect 2002; 110(10): 1025-9.
62) Stolzel M, Breitner S Cyrys J et al. Daily mortality and particulate matter in different size classes in Erfurt, Germany. J Expo Sci Environ Epidemiol.Research, 2007; 17(5): 459-67
63) Kettunen J, Lanki T, Tiittanen P et al. Associations of fine and ultrafine particulate air pollution with stroke mortality in an area of low pollution levels. Stroke, 2007; 38(3): 918-22
64) Li N, Sioutas C, Cho A et al. Ultrafine particulate pollutants induce oxidative stress and mitochondrial damage.Environ Health Perspect 2003,111:455-460
65) Oberdorster G, Sharp Z Atudorei V et al. Translocation of inhaled ultrafine particles to the brain.Inhalation Toxicology, 2004, 16:437-445
66) Air Quality Guidelines for Europe, Section 7.3 p19, Second Edition, World Regional Publications, Regional European Series No 91, World Health Organisation, Regional Office for Europe, Copenhagen.
67) Annesi-Maesano I, Forastiere F, Kunzli N et al. Partciculate matter, Science and EU Policy. Eur Resp J 2007; 29: 428-431

3.2 Heavy Metals

68) Proceedings of the Third Colloquium on Particulate Air Pollution and Human Health 6-8 June 1999, Durham, North Carolina, Irvine, CA: Air Pollution Effects Laboratory, University of California,1999, 11/23.
69) Pope CA 3rd. Respiratory disease associated with community air pollution and a steel mill, Utah Valley. Am J Public Health, 1989, 79(5): 623-8.
70) Costa DL, Dreher KL. Bioavailable transition metals in particulate matter mediate cardiopulmonary injury in healthy and compromised animal models. Environ Health Perspect 1997;105 (suppl 5): 1053-60
71) Dye JA, Lehmann JR, McGee JK et al. Acute pulmonary toxicity of particulate matter filter extracts in rats: Coherence with epidemiologic studies in Utah Valley. Environ Health Perspect 2001; 109 Suppl 3: 395-403.
72) Hutzinger O, Fielder H. Formation of Dioxins and Related Compounds in Industrial Processes. Pilot Study of International Information Exchange on Dioxins and Related Componds. No 173. Brussels: NATO Committee on Challenges to Modern Science, 1998
73) Rowat SC. Incinerator toxic emissions: a brief summary of human health effects with a note on regulatory control. Med Hypotheses 1999; 52(5): 389-96.
74) Casdorph R, Walker M. Toxic Metal Syndrome, New York: Avery Publishing Group 1995.
75) Ehmann WD, Markesbery WR, Alauddin M et al. Brain trace elements in Alzheimer's disease. Neurotoxicology 1986; 7 (1): 195-206.
76) Thompson CM, Markesbery WR, Ehmann WD et al. Regional trace-element studies in Alzheimer's disease. Neurotoxicology 1988; 9(1): 1-7.
77) Wenstrup D, Ehmann WD, Markesbery WR. Trace element imbalances in isolated subcellular fractions of Alzheimer's disease brains. Brain Res 1990; 533(1): 125-31.
78) Schettler T. Toxic threats to neurological development of children. Environ Health Perspect 2001; 109 (Suppl 6): 813-6.
79) Grandjean P, Weihe P, White RF et al. Cognitive deficit in 7-year old children with prenatal exposure to methyl mercury. Neurotoxicol Teratol 1997; 19(6): 417-28.
80) Palmer RF, Blanchard S, Stein Z et al. Environmental mercury release, special education rates, and autism disorder: an ecological study of Texas. Health Place, 2006;12(2): 203-9
81) Palmer RF, Blanchard S, Wood R. Proximity to point sources of environmental mercury release as a predictor of autism prevalence. Health Place, 2008; doi:10.1016/j.healthplace.2008.02.001
82) Thun MJ, Schnorr TM, Smith AB, et al. Mortality among a cohort of US cadmium production workers – an update. J Natl Cancer Inst 1985; 74(2): 325-33.
83) Blot WJ, Fraumeni JF Jnr. Arsenical air pollution and lung cancer. Lancet 1975; 2 (7926):142-4.
84) Severs R, Whitehead L, Lane R. Air quality correlates of chronic disease mortality: Harris County, Texas 1969-71. Tex Rep Biol Med 1978; 36: 169-84.

85) Wecker L, Miller SB, Cochran SR et al. Trace element concentration in hair from Autistic Children. J Ment Defic Res 1985: 29 (pt 1): 15-22.
86) Capel ID, Pinnock MH, Dorrell HM, et al. Comparison of concentrations of some trace, bulk, and toxic metals in the hair of normal and dyslexic children. Clinic Chem 1981: 27(6): 879-81.
87) Brockel BJ, Cory-Slechta DA. Lead, attention, and impulsive behaviour: changes in a fixed waiting-for-reward paradigm. Pharmacol Biochem Behav 1998: 60(2): 545-52.
88) David OJ, Hoffman SP, Sverd J, et al. Lead and hyperactivity: Behavioural response to chelation. Am J Psych 1976; 133(10): 1155-8.
89) Masters RD. Biology and politics: linking nature with nurture. Ann Rev Polit Sci 2001; 4: 345-65.
90) Leviton A, Bellinger D, Allred EN et al. Pre and postnatal low-level lead exposure and children's dysfunction in school. Environ Res 1993: 60(1): 30-43.
91) Eppright TD, Sanfacon JA, Horwitz FA. Attention deficit hyperactivity disorder, infantile autism and elevated blood lead: a possible relationship. Mol Med 1996: 93(3): 136-8.
92) Needleman HL, Gunnoe C, Leviton A et al. Deficits in psychologic and classroom performance in children with elevated dentine lead levels. N Eng J Med 1994; 331(13): 689-95.
93) Bellinger D, Leviton A, Waternaux C, et al. Longitudinal analyses of prenatal and postnatal lead exposure and early cognitive development. N Eng J Med 1987; 316 (17): 1037-43.
94) Needleman HL, Riess JA, Tobin MJ, et al. Bone lead levels and delinquent behaviour. JAMA 1996; 275 (5); 363-9.
95) Mispelstraat: Living under the smoke of a waste incinerator. Report on the health impact of the MIWA waste incinerator in Sint Niklaas, Belgium. www.milieugezondheid.
96) Schauss AG. Comparative hair-mineral analysis results of 21 elements in a random selected behaviourally "normal" 19-59 year old population and violent adult criminal offenders. Int J Biosoc Res 1981; 1: 21-41.
97) Bowdler NC, Beasley DS, Fritze EC et al. Behavioural effects of aluminium ingestion on animal and human subjects. Pharmacol Biochem Behav 1979: 10(4): 502-12.
98) Trapp GA, Miner GH, Zimmerman RL et al. Aluminium levels in the brain in Alzheimer's disease. Biol Pyschiatry 1978; 13(6): 709-18.
99) Multhaup G. Amyloid precursor protein, copper and Alzheimer's disease. Biomed Pharmocother 1997: 51(3): 105-11.
100) Zapatero MD, Garcia de Jalon A, Pascual F, et al. Serum aluminium levels in Alzheimer's disease and other senile dementias. Biol Trace Elem Res 1995; 47 (1-3): 235-40.
101) Martyn CN, Barker DJ, Osmond C et al. Geographical relationship between Alzheimer's disease and aluminium in drinking water. Lancet 1989; 1(8763): 59-62.
102) Crapper DR, Krishnan SS, Dalton AJ et al. Brain aluminium distribution in Alzheimer's disease and experimental neurofibrillary degeneration. Science 1973: 180(85): 511-3.
103) Neri LC, Hewitt D. Alzheimer's disease and drinking water. Lancet 1991; 338 (8763): 390.
104) Zayed J, Ducic S, Campanella G, et al. Environmental factors in the etiology of Parkinson's disease. Can J Neurol Sci 1990: 17(3): 286-91.

3.3 Nitrous Oxides

105) Richters A, Richters V. A new relationship between air pollutant inhalation and cancer. Arch Environ Health 1983; 38(2): 69-75.
106) Ruaslahti E. How cancer spreads. Scientific American Sept 1996: 72-77.
107) Andersen HR, Spix C, Medina S, et al. Air pollution and daily admissions for chronic obstructive pulmonary disease in 6 European cities: results from the APHEA project. Eur Resp J 1997; 10(5): 1064-71.
108) Sunyer J, Spix C, Quenel P, et al. Urban air pollution and emergency admissions for asthma in four European cities: the APHEA Project. Thorax 1997; 52(9): 760-5.
109) Ostro BD, Broadwin R, Lipsett MJ. Coarse and fine particles and daily mortality in the Coachella Valley, California: a follow-up study. J Exp Anal Environ Epidemiol 2000; 10(5): 412-9.

110) Breslin K. The impact of ozone. Env Health Perspectives 1995; 103(7-8): 660-4.
111) Hoek G, Schwartz JD, Groot B, Eilers P. Effects of ambient particulate matter and ozone on daily mortality in Rotterdam, The Netherlands. Arch Environ Health 1997; 52(6): 455-63.

3.4 Organic Toxicants

112) Den Hond E, Roels HA, Hoppenbrouwers K et al. Sexual maturation in relationship to polychlorinated aromatic hydrocarbons: Shape and Skakkebaek's hypothesis revisited. Environ Health Perspect 2002; 110(8): 771-6.
113) Eskenazi B, Mocarelli P, Warner M et al. Serum dioxin concentrations and endometriosis: a cohort study in Sevenso, Italy. Environ Health Perspect 2002; 110(7): 629-34.
114) Wolff MS, Weston A. Breast cancer risk and environmental exposures. Environ Health Perspect 1997; 105(Suppl 4): 891-6.
115) Hoyer AP, Granjean P, Jorgensen T et al. Organochlorine exposure and the risk of breast cancer. Lancet 1998; 352 (9143): 1816-20.
116) Oliva A, Spira A, Multigner L et al. Contribution of environmental factors to the risk of male infertility. Hum Reprod 2001; 16(8): 1768-76.
117) Sultan C, Balaguer P, Terouanne B et al. Environmental xenoestogens, antiandrogens and disorders of male sexual differentiation. Mol Cell Endocrinol 2001; 178 (1-2): 99-105.
118) Hardell L, van Bavel B, Lindstrom G et al. Increased concentrations of polychlorinated biphenyls, hexachlorobenzene and chlordanes in mothers of men with testicular cancer. Environ Health Perspect 2003; 111 (7): 930-4.

a) Organochlorines

119) Jay K, Stieglitz L. Identification and quantification of volatile organic components in emissions of waste incineration plants. Chemosphere 1995; 30: 1249-1260
120) Ecocyle Commission of the Government of Sweden. PVC: A Plan to Prevent Environmental Impact. Stockholm: Ecocycle Commission 1994
121) American Public Health Association. Resolution 9304: Recognizing and addressing the environmental and occupational health problems posed by chlorinated organic chemicals. Am J Public Health 1994; 84:514-5
122) Thornton J, Pandora's Poison, 2000, MIT Press, Cambridge, Massachusetts & London
123) US Enironmental Protection Agency. Estimating exposure to 2,3,7,8 TCDD. Nal Review Draft. Washington DC: US EPA, Office of Research and Development (EPA/600-6-99-007A), 1988
124) Tatsukawa R, Tanabe S. Fate and bioaccumulation of persistent organochlorine compounds in the marine environment. In: Baumgartner DJ, Dudall IM, eds. Oceanic Processes in Marine Pollution, Volume 6, Malabar FL: Kreiger, 1990:39-55
125) Frank H, Norokorpi Y SchollH et al. Trichloroacetate levels in the atmosphere and in conifer needles in Central and Northern Europe. Organohalogen Compounds 1993,14:307-8
126) Norstom R, Muir DCG. Chlorinated hydrocarbon contaminants in arctic marine mammals. Science of the Total Environment, 1994; 154:107-128
127) Arctic Monitoring and Assessment Programme. Arctic Pollution Issues: A state of the Arctic Environment Report. Oslo:AMAP Directorate, 1997

b) Dioxins

128) BrzuzyLP, Hites RA. Global mass balance of polychlorinated dibenzo-p-dioxins and dibenzofurans. Environmental Science and Technology, 1996, 30:1797-1804
129) US Environmental Protection Agency. The Inventory of sources of dioxin in the United States (Review Draft). Washington DC: US EPA Office of Research and Development (EPA/ 600/p-98-002a), 1998
130) Thomas V, Shapiro C. An estimation of dioxin emissions in the United States. Toxicology and Environmental Chemistrty, 1995; 50:1-37
131) Davis DL, Dinse GE, Hoel DG. Decreasing cardiovascular disease and increasing cancer among whites in the United States from 1973 through 1987. JAMA, 1994; 271:431-437
132) Tritscher AM, Clark GS, Lucier GW. Dose-response effects of dioxins:Species comparison and implications for risk assessment. In: Schecter A, Dioxins and Health. New York:plenum, 1994:227-248130)

133) Neubert R, Jacob-Muller U, Helge H et al. Polyhalogenated dibenzo-p-dioxins and dibenzofurans and the immune system: In vitro effects of 2,3,7,8 tetrachlorodibenzo-p-dioxin (TCDD) on lymphocytes of venous blood from a man and a non-human primate. Archives of Toxicology 1991;65:213-9
134) US Environmental Protection Agency. Health Assessment Document for 2,3,7,8 – tetrachlorodibenzo-p-dioxin and Related Compounds, Volumes 1-3, Review Draft. Washington DC:US EPA Office of Research and Development (EPA/600/BP-92-001),1994
135) Becher H, Steindorf K, Flesch-Janys D. Quantitative cancer risk assessment of dioxins using an occupational cohort. Env Health Perspect 1998; 106(Suppl 2): 663-670
136) Papke O. PCDD/F:Human background data from Germany, a 10 year experience. Env Health Perspect, 1998; 106 (Suppl 2): 723-31
137) Schecter A, Startin J, Wright C et al. Congener-specific levels of dioxins and dibenzofurans in US food and estimated daily dioxin toxic equivalent intake. Env Health Perspect 1994; 102: 962-966
138) Schecter A, Gasiewicz T. Health hazard assessment of chlorinated dioxins and dibenzofurans contained in human milk. Chemosphere 1987; 16:2147-54
139) Gray LE, Ostby JS, Kelce WR. A dose-response analysis of the reproductive effects of a single gestational dose of 2,3,7,8 tetrachlorodibenzo-p-dioxin in male Long Evens hooded rat offspring. Toxicology and Applied pharmacology, 1997; 146: 11-20
140) Theobald HM, Peterson RE. Developmental and reproductive toxicity of dioxins and other Ah receptor agonists. In: Schecter A, ed. Dioxins and Health. New York: Plenum. 1994:309-46
141) Seegal RF, Schantz SL. Neurochemical and behavioural sequelae of exposure to doxins and PCBs. In: Schecter A, ed. Dioxins and Health. New York: Plenum. 1994:409-448
142) Quass U, Fermann M, Broker G, European Dioxin Inventary Volume 3, Assessment of dioxin emissions until 2005,. Prepared by North Rhine Westphaliam State Environmental Agency on behalf of the European Commission, Directorate General for Environment (DG ENV)

3.5 Effects on genetic Material

143) Tomatis L. Transplacental Carcinogenesis. Lyon, International Agency for Research on Cancer, IARC Scientific Publications No 4 pp100-111.
144) Tomatis L, Goodall CM. The occurrence of tumours in F1, F2 and F3 descendants of pregnant mice injected with 7,12 dimethylbenz(a)anthracene. Int J Cancer 1969; 4(2): 219-25.

3.6 Effects on Immune System

145) Ross P, de Swart, Visser I, et al. Relative immunocompetence of the newborn harbor seal, Phoca vitulina. Veterinary Immunology and Immunopathology 1994; 42(3-4): 331-48.
146) Ross P, de Swart R, Reijnders P, et al. Contaminant-related suppression of delayed-type hypersensitivity and antibody responses in harbor seals fed herring from the Baltic Sea. Env Health Perspect 1995; 103 (2): 162-7.
147) De Swart R. Impaired immunity in seals exposed to bioaccumulated environmental contaminants, PhD Thesis, Erasmus University, Rotterdam, Netherlands, 1995.
148) Lahvis G, Wells RS, Kuehl DW et al. Decreased lymphocyte response in free-ranging bottle-nosed dolphins (Tursiops truncatus) are associated with increased concentration of PCBs and DDT in peripheral blood. Env Health Perspect 1995; 103(4): 67-72.
149) Cone JE,Harrison R, Reiter R. Patients with multiple chemical sensitivities: clinical diagnostic subsets among an occupational health clinic population. *In* Cullen M (ed) Workers with Multiple Chemical Sensitivities, Occupational Medicine: State of the Art Review 1987; 2(4):721-738 .
150) Sharma R. Immunological Considerations in Toxicology, Vols 1 and 2 (1981), CRC Press, Boca Raton, FL.
151) Ashford N, Miller C. Chemical Exposures: Low Levels and High Stakes. John Wiley & Sons 1998.

152) Massolo L, Muller A, Tueros M, et al. Assessment of mutagenicity and toxicity of different-size fractions of air particles from La Plata, Argentina, and Leipzig, Germany. Environ Toxicol 2002; 17(3): 219-31.
153) Kerkvliet NI. Immunotoxicity of dioxins and related chemicals. In: Schecter A, ed. Dioxins and Health. New York: Plenum1994:199-217
154) Tryphonas H. Immunotoxicity of PCBs (Aroclors) in relation to Great Lakes. Environ Health Perspect, 1995; 103 (Suppl 9):35-46
155) Hillam RP, Bice DE, Hahn FF, Scnizelein CT. Effects of acute nitrogen dioxide exposure on cellular immunity after lung immunization. Environ Res 1983; 31(1): 201-11.

3.7 Synergistic Effects

156) Carroll Wilson. Man's Global Impact on the Environment: A Study of Critical Environmental Problems, MIT Press, Cambridge, Mass 1971.
157) Mokhiber R. The Ecologist 1998; 28(2): 57-8.
158) Harrison PT, Heath JC. Apparent synergy in lung carcinogenesis: interactions between N-nitrosheptamethyleneimine, particulate cadmium and crocidolite asbestos fibres in rats. Carcinogenesis 1986; 7(11): 1903-8.
159) Wade MG, Foster WG, Younglai EV, et al. Effects of subchronic exposure to a complex mixture of persistent contaminants in male rats: systemic, immune and reproductive effects. Toxicol Sci 2002; 67(1): 131-43.
160) Soto AM, Chung KL, Sonnenschein C. The pesticides endosulphan, toxaphene and dieldrin have estrogenic effects on human estrogen-sensitive cells. Environ Health Perspect 1994; 102(4): 380-3.
161) Abou-Donia MB, Wilmarth KR, Jensen KF et al. Neurotoxicity resulting from co-exposure to pyridostigmine bromide, DEET and permethrin: Implications of Gulf War chemical exposures. J Toxicol Env Health 1996; 48(1): 35-56.
162) Ershoff BH. Synergistic toxicity of food additives in rats fed a diet low in dietary fibre. J Food Sci 1976; 41: 949-51.
163) Wade MG, Parent S, Finnson KW, et al. Thyroid Toxicity due to a subchronic exposure to a complex mixture of 16 organochlorines, lead, and cadmium. Toxicol Sci 2002; 67(2): 207-18.
164) Arnold SF, Klotz DM, Collins BM, et al. Synergistic activation of estrogen receptors with combinations of environmental chemicals. Science 1996; 272 (5267): 1489-92.
165) Germolec DR, Yang RSH, Ackermann MP et al. Toxicology studies of a chemical mixture of 25 groundwater contaminants: Immunosuppression in B6C3F mice. Fundamental and Applied Toxicology 1991;13: 377-387
166) Li MH, Hansen LG. Enzyme induction and acute endocrine effects in prepubertal female rats receiving environmental PCB/PCDF/PCDD mixtures. Environ Health Perspect 1996; 104(7): 712-22.

Section 4. Increased Morbidity and Mortality near Incinerators

4.1 Cancer

167) Elliot P, Shaddick G, Kleinschmidt I etal, Cancer incidence near municipal solid waste incinerators in Great Britain. Brit J Cancer 1996; 73(5): 702-10.
168) Elliot P, Eaton N, Shaddick G et al. Cancer incidence near municipal waste incinerators in Great Britain. Part 2: Histopathological and case note review of primary liver cancer cases. British J Cancer 2000; 82(5): 1103-6.
169) Knox EG, Gilman EA. Migration patterns of children with cancer in Britain. J Epidemiology & Community Health 1998; 52(11): 716-26.
170) Knox EG. Childhood cancers, birthplaces, incinerators and landfill sites. Int J Epidemiology 2000; 29 (3): 391-7.
171): Biggeri A, Barbone F, Lagazio C, et al. Air pollution and lung cancer in Trieste, Italy: Spatial analysis of risk as a function of distance from sources. Environ Health Perspect 1996; 104 (7): 750-4.

172) Viel JF, Arveux P, Baverel J, et al. Soft tissue sarcoma and non Hodgkin's lymphoma clusters around municipal solid waste incinerators with high dioxin emission levels. Am J Epidemiology 2000; 152(1): 13-19.
173) Ohta S, Kuriyama S, Nakao et al. Levels of PCDDs, PCDFs and non-ortho coplanar PCBs in soil collected from high cancer-causing area close to batch-type municipal solid waste incinerator in Japan. Organohalogen Compounds 1997; 32: 155-60.
174) Comba P, Ascoll V, Belli S et al Risk of soft tissue sarcomas and residence in the neighbourhood of an incinerator of industrial wastes. Occup Environ Med 2003; 60(9): 680-3
175) Zambon P, Ricci P, Bovo E et al. Sarcoma risk and dioxin emissions from incinerators and industrial plants: a population-based case-control study (Italy). Environ Health 2007; 6:
176) Gustavsson P. Mortality among workers at a municipal waste incinerator. Am J Ind Med 1989; 15(3): 245-53.
177) Gustavsson P, Evanoff B, Hogstedt C. Increased risk of esophageal cancer among workers exposed to combustion products. Archives Environ Med 1993; 48(4): 243-5.

4.2 Birth Defects

178) ten Tusscher GW, Stam GA, Koppe JG. Open chemical combusting resulting in a localised increased incidence of orofacial clefts. Chemosphere 2000; 40(9-11): 1263-70.
179) Van Lorebeke N. Health effects of a household waste incinerator near Wilrijk, Belgium. *In* Health Impacts of Waste Management Policies, Hippocrates Foundation, Kos, Greece, 2000.
180) Cordier S, Chevrier C, Robert-Gnansia E et al. Risk of congenital anomalies in the vicinity of municipal solid waste incinerators. Occup Environ Med 2004: 61(1): 8-15.
181) Dummer TJ, Dickinson HO, Parker L. Adverse pregnancy outcomes around incinerators and crematoriums in Cumbria, North-west England, 1956-93. J Epidemiol Community Health 2003: 57(6): 456-61.
182) Dolk H, Vrijheld M, Armstrong B et al. Risk of congenital anomalies near hazardous-waste landfill sites in Europe: the EUROHAZCON study. Lancet, 1998; 352(9126): 423-7.
183)Elliot P, Briggs D, Morris S et al. Risk of adverse birth outcomes in populations living near landfill sites. BMJ, 2001; 323(7309): 363-8.
184) Croen LA, Shaw GM, Sanbonmatsu L et al. Maternal residential proximity to hazardous waste sites and risk for selected congenital malformations. Epidemiology 1997; 8(4): 347-54.
185) Orr M, Bove F, Kaye W et al. Elevated birth defects in racial or ethnic minority children of women living near hazardous waste sites. Int J Hyg Environ Health, 2002; 205(1-2): 19-27.
186) Johnson BL. A review of the effects of the effects of hazardous waste on reproductive health. Am J of Obstetrics and Gynecology 1999; 181: S12-S16.

Section 5. Disease Incidence and Pollution

5.1 Cancer

187) NCI, 1991: "Cancer Statistics Review 1973-88", NIH Publications No 91-2789.
188) Tomatis L, Cancer, Causes, Occurrence and Control, IARC Scientific publications 100, (Lyon, France, IARC 1996) 21.
189) Graphs of chemical production: From International Trade Commission, Washington DC.
190) Davies DL, Hoel D, Foxj, Lopez A. International trends in cancer mortality in France, West Germany, Italy, Japan, England and Wales and the USA. Lancet 1990; 336 (8713): 474-81.
191) Pickle LW, Mason TJ, Fraumeni JF Jr. The new United States Cancer Atlas. Recent Results Cancer Res, 1989; 114: 196-207.
192) Najem GR, Louria DB, Lavenhar MA et al. Clusters of cancer mortality in New Jersey municipalities, with special reference to chemical toxic waste disposal sites and *per capita* income. Int J Epidemiol 1985; 14(4): 528-37.
193) Najem GR, Greer W. Female reproductive organs and breast cancer mortality in New Jersey Counties and the relationship with certain environmental variables. Prev Med 1985: 14(5): 620-35.
194) Hoover R, Fraumeni JF Jr. Cancer mortality in US counties with chemical industries. Environ Res 1975; 9(2): 196-207.

195) Goldman BA. The Truth About Where You Live: An Atlas for Action on Toxins and Mortality. New York: Random House 1991.
196) Zahm SH, Blair A. Cancer among migrant and seasonal farmers: an epidemiologic review and research agenda. Am J of Ind Med 1993; 24(6): 753-66.
197) Tornling G, Gustavsson P, Hogstedt C. Mortality and cancer incidence among Stockholm fire fighters. Amer J Industrial Med 1994: 25(2): 219-28.
198) Zahm SH, Weisenburger DD, Babbitt PA et al. A case control study of non-Hodgkin's Lymphoma and the Herbicide 2,4 Dichlorophenoxyacetic acid (2,4-D) in Eastern Nebraska. Epidemiology 1990; 1(5): 349-56.
199) Hardell L, Eriksson M, Lenner P et al. Malignant lymphoma and exposure to chemicals, especially organic solvents, chlorophenols and phenoxy acids: a case control study. Brit J Cancer 1981; 43(2): 169-76.
200) Harshbarger JC and Clark JB. Epizootiology of neoplasms in bony fish of North America. Sci Total Environ 1990; 94(1-2): 1-32.
201) Hayes HM Jr, Hoover R, Tarone RE. Bladder cancer in pet dogs: a sentinel for environmental cancer. Am J Epidemiol 1981; 114(2): 229-33.
202) Baumann PC, HarshbargerJC. Decline in liver neoplasms in wild brown bullhead catfish after coking plant closes and environmental PAHs plummet. Environ health Perspect 1995; 103: 168-70.
203) Perera F.P, Hemminki K, Gryzbowska E et al. Molecular and Genetic Damage in Humans from Environmental Pollution in Poland. Nature 1992; 360 (6401): 256-58.
204) Perera FP, Mooney LA, Stamfer M et al. Associations between carcinogen-DNA damage, glutathione S transferase genotypes, and risk of lung cancer in the prospective Physician's Health Cohort Study. Carcinogenesis 2002; 23(10): 1641-6.
205) Lewis-Michl EL, Melius JM, Kallenbach LR et al. Breast cancer risk and residence near industry or traffic in Nassau and Suffolk Counties, Long Island, New York. Arch Environ Health 1996; 51(4): 255-65.
206) The Long island Breast Cancer Study Reports 1-3 (1988-90), New York State Department of Health, Department of Community and Preventative Medicine, Nassau County Department of Health and Suffolk County Department of Health Services.
207) Aschengrou A, Ozonoff DM. Upper Cape Cancer Incidence Study: Final Report. Boston: Mass. Depts of Public Health and Environment Protection 1991.
208) Aschengrau A, Ozonoff D, Paulu C et al. Cancer risk and tetrachloroethylene-containing drinking water in Massachusetts. Arch Environ Health 1995; 48(5): 284-92.
209) McKelvey W, Brody JG, Aschengrau A et al. Association between residence on Cape Cod, Massachusetts, and breast cancer. Ann Epidemiol 2004; 14(2): 89-94.
210) Fagliano J, Berry M, Boye F et al. Drinking water contamination and the incidence of leukaemia:an ecologic study. Am J Public Health 1990; 80 (10): 1209-12.
211) Cantor KP et al., Water Pollution *In* Schottenfeld D and Fraumeni JF Jr (eds.), Cancer Epidemiology and Prevention, 2nd ed. Oxford: Oxford Univ Press 1996.
212) Lagakos S.W et al. An analysis of contaminated well water and health effects in Woburn, Massachusetts. J Amer Stat Assoc 1986: 395: 583-96.
213) Osborne J.S, Shy CM, Kaplan BH. Epidemiologic analysis of a reported cancer case cluster in a small rural population. Am J Epidemiol 1990; 132 (Supp 1): S87-95.
214) Lampi P, Hakulinen T, Luostarinen et al. Cancer incidence following chlorophenol exposure in a community in Southern Finland. Arch Environ Health 1992; 47(3): 167-75.
215) IARC Monographs on Evaluation of Carcinogenic Risks to Humans Suppl 7 (Lyon, France: IARC 1987).
216) US.DHHS Seventh Annual Report on Carcinogens, Research Triangle Park, NC:us. Department of Health and Human Services, 1990.
217) Holzman D. Banking on tissues. Environ Health Perspect 1996; 104(6): 606-10.
218) Moses M, Johnson ES, Anger WK et al. Environmental equity and pesticide exposure. Toxicol Ind Health 1993; 9(5): 913-59.

219) Onstot J, Ayling R, Stanley J. Characterization of HRGC/MS Unidentified Peaks from the Analysis of Human Adipose tissue. Volume 1: Technical approach. Washington DC: US Environmental Protection Agency Office of Toxic Substances (560/6-87-002a), 1987
220) Body Burden: Executive Summary, 2003, Environmental Working Group, Mount Sinai School of Medicine and Commonweal. www.ewg.org/reports/bodyburden/
221) Foster W, Chan S, Platt L, Hughes C. Detection of endocrine disrupting chemicals in samples of second trimester human amniotic fluid. J Clinic Endocrinol Metabol 2000; 85(8): 2954-7.
222) Zieger M. Biomarkers: The clues to genetic susceptibility. Environ Health Perspectives 1994; 102(1): 50-7.

5.2 Neurological Disease

223) Rodier PM. Developing brain as a target of toxicity. Environ Health Perspect 1995: 103 Suppl 6: 73-6.
224) Hattis D, Glowa J Tilson H et al. Risk assessment for neurobehavioural toxicity: SGOMSEC joint report. Env Health Perspect 1996;104 (Suppl 2): 249-71
225) Landrign PJ, Graham DG, Anger WK et al. Environmental toxicology. Washington DC: National Academy Press,1992.
226) WeissB.Risk assessment: The insidious nature of neurotoxicity and the aging brain. Neurotoxicology 1990;11:305-14
227) Knapp et al. Dementia UK: Report to the Alzheimer's Society, King's College, London and London School of Economics and Political Science, 2007
228) Pritchard C, Baldwin D, Mayers A. Changing patterns of adult neurological deaths (45-74 years) in the major western world countries (1979-1997). Public Health 2004; 118(4): 268-83.
229) Taylor B, Miller E, Farrington CP et al. Autism and measles, mumps and rubella vaccine: no epidemiological evidence for a causal association. Lancet 1999; 353(9169): 2026-9.
230) Rybicki RA, Johnson CC, Uman J, Gorrell JM. Parkinson's disease mortality and the industrial use of heavy metals in Michigan. Mov Disord 1993; 8(1): 87-92.

5.3 Mental Disease

231) Strahilevitz M, Strahilevitz A, Miller JE. Air pollutants and the admission rate of psychiatric patients. Am J Psychiatry 1979; 136(2): 205-7.
232) Briere J, Downes A, Spensley J. A. summer in the city: urban weather conditions and psychiatric emergency room visits. J Abnorm Pyschol 1983; 92(1): 77-80.
233) Morrow LA, Kamis H, Hodgson MJ. Psychiatric symptomatology in persons with organic solvent exposure. J Consult Clinic Pyschol 1993; 61(1): 171-4.
234) Morrow LA, Stein L, Scott A et al. Neuropsychological assessment, depression and past exposure to organic solvents. Applied Neuropyschol 2001; 8(2): 65-73.

5.4 Violence and Crime

235) Hall RW. A study of mass murder: evidence of underlying cadmium and lead poisoning and brain-involved immunoreactivity. Int J Bioscoc Med Res 1989; 11: 144-52.
236) Marlowe M, Schneider HG, Bliss LB. Hair mineral analysis in emotionally disturbed and violence prone children. Int J Biosoc Med Res 1991; 13: 169-79.
237) Pihl RO, Ervin F. Lead and cadmium levels in violent criminals. Pyschol Rep 1990; 66(3Pt 1): 839-44.
238) Denno DW. Gender, crime and the criminal law defences. J Crim Law Criminol 1994; 85: 80-180.
239) Deborah Denno. Biology and Violence: From Birth to Adulthood. Cambridge University Press, 1990.
240) Stretesky PB, Lynch MJ. The relationship between lead exposure and homicide. Arch Ped Adolesc Med 2001; 155(5): 579-82.
241) Stretesky PB, Lynch MJ. The relationship between lead and crime. J Health & Soc Behav 2004; 45(2): 214-29.

6. High Risk Groups

6.1 The Foetus

242) Centers for Disease Control. Blood and hair mercury levels in young children and women of childbearing age. United States 1999 Morbidity and Mortality Report, 2001; 50: 140-43.
243) Anderson LM, Diwan BA, Fear NT, Roman E. Critical windows of exposure for children's health: cancer in human epidemiological studies and neoplasms in experimental animal models. Environ Health Perspect 2000; 108 suppl 3: 573-94.
244) US Environmental Protection Agency, Office of Pollution Protection and Toxic Substances, Chemical Hazard Data Availability Study: What do we really know about high production volume chemicals? USEPA: Washington DC,1998.
245) Sonnenschein C, Soto AM. An Updated review of environmental estrogen and androgen mimics and antagonists. J Steroid Biochem Mol Biol 1998; 65 (1-6): 143-50.
246) Markey CM, Coombs MA, Sonnenschein C, Soto AM. Mammalian development in a changing environment: exposure to endocrine disruptors reveals the developmental plasticity of steroid-hormone target organs. Evol Dev 2003; 5(1): 67-75.
247) Tilson HA, Jacobson JL, Rogan WJ. Polychlorinated biphenyls and the developing nervous system: cross species comparisons. Neurotoxicol Teratol 1990; 12 (3): 239-48.
248) Weisgals-Kuperas N, Patandin S, Berbers GA, et al. Immunological effects of background exposure to polychlorinated biphenyls and dioxins in Dutch preschool children. Environ Health Perspect 2000; 108(12): 1203-7.
249) Rogan WJ, Gladen BC, McKinney JD, et al. Polychlorinated biphenyls (PCBs) and dichlorodiphenyldichloroethene (DDE) in human milk: effects of maternal factor and previous lactation. Am J Public Health 1986; 76(2): 172-7.
250) Body Burden: The Pollution in Newborns: Executive Summary, July 2005, Environmental Working Group, Mount Sinai School of Medicine and Commonweal.www.ewg.org/reports/bodyburden2/execsumm.php
251) A Present for Life: Hazardous chemicals in umbilical cord blood. WWF/Greenpeace, September 2005.

[redacted]

6.2 The Breast-fed Infant

252) Jensen AA, Slorach SA. Assessment of infant intake of chemicals via breast milk *in* Chemical Contaminants in Human Milk. Boca Raton: CRC Press 1991. pp215-22.
253) Koopman-Esseboom C, Huisman M, Weisglas-Kuperus N, et al. Dioxin and PCB levels in blood and human milk in relation to living in the Netherlands. Chemosphere 1994; 29 (9-11): 2327-38.
254) Patandin S, Dagnelie PC, Mulder PG, et al. Dietary exposure to polychlorinated biphenyls and dioxins from infancy until adulthood: a comparison between breast-feeding, toddler and long-term exposure. Environ Health Perspect 1999; 107(1): 45-51.
255) Rogan WJ, Bagniewska A, Damstra T. Pollutants in breast milk. N Engl J Med 1980; 302(26): 1450-3.
256) Jacobson JL, Jacobson SW. Prenatal exposure to polychlorinated biphenyls and attention at school age. J Paediatr 2003; 143(6): 780-8.
257) Jacobson JL, Jacobson SW. Association of prenatal exposure to an environmental contaminant with intellectual function in childhood. J Toxicol Clin Toxicol 2002; 40(4): 467-75.
258) Jacobson JL, Jacobson SW. Intellectual impairment in children exposed to polychlorinated biphenyls in utero. N Eng J Med 1996; 335(11): 783-9.
259) Kinbrough RD. Toxicological implications of human milk residues as indicated by toxicological and epidemiological studies *in* Jensen AA & Slorach SA: Chemical Contaminants in Human Milk, 1990 pp271-83.
260) Rice DC. Behavioural impairment produced by low-level postnatal PCB exposure in monkeys. Env Res 1999; 80(2 Pt 2): S113-S121.
261) Rice DC. Effects of postnatal exposure of monkeys to a PCB mixture on spatial discrimination reversal and DRL performance. Neurotoxicol Teratol 1998; 20(4): 391-400.

262) Rice DC, Hayward S. Effects of postnatal exposure to a PCB mixture in monkeys on non-spatial discrimination reversal and delayed alternation performance. Neurotoxicology 1997; 18(2): 479-94.
263) Hallgren S, Sinjari T, Hakansson H, Darnerud PO. Effects of polybrominated diphenyl ethers (PBDEs) and polychlorinated biphenyls (PCBs) on thyroid hormone and vitamin A levels in rats and mice. Arch Toxicol 2001; 75(4): 200-8.
264) Hooper K, McDonald TA. The PBDEs: an emerging environmental challenge and another reason for breast milk monitoring programs. Env Health Perspect 2000; 108(5): 387-92.

6.3 Children

265) Moolgavkar SH, Venzon DJ. Two-event model for carcinogenesis: incidence of curves for childhood and adult tumours. Maths Biosci 1979; 47: 55-77.
266) Rodier PM. Chronology of neuron development: animal studies and their clinical implications. Dev Med Child Neurol 1980; 22(4): 525-45.
267) Ekbom A, Hsieh CC, Lipworth L, et al. Intrauterine environment and breast cancer risk in women: a population-based study. J Natl Cancer Inst 1997; 89(1): 71-6.
268) Knox EG. Childhood cancers and atmospheric carcinogens. J Epidemiol Community Health 2005; 59(2): 101-5.
269) Tomatis L, Overview of perinatal and multigeneration carcinogenesis. ARC Sci Publ 1989; 96: 1-15.
270) Anderson LM, Donovan PJ, Rice JM, Risk assessment for transplacental carcinogenesis. *In* New Approaches in Toxicity Testing and their Application in Human Risk Assessment (ed Li AP). 1985 pp179-202.
271) Landrigan PJ, Garg A. Chronic effects of toxic environmental exposures in children's health. J Toxicol Clinical Toxicol 2002; 40(4): 449-56.
272) Calabrese E.J. Age and Susceptibility to Toxic Substances. New York, John Wiley & Sons 1986.
273) National Academy of Sciences. Scientific Frontiers in Developmental Toxicology and Risk Assessment. National Academy Press, Washington DC 2000.
274) Windham GC, Zhang L, Gunier R et al. Autism spectrum disorders in relation to distribution of hazardous air pollutants in the San Francisco bay area. Environ Health Perspect 2006;114(9): 1438-44
275) Kenet T et al. Perinatal exposure to a noncoplanar polychlorinated biphenyl alters tonotopy, receptive fields and plasticity in rat primary auditory cortex. Proc Natl Acad Sci USA 2007; 104 (18):7646-51
276) Jacobson JL, Jacobson SW, Humphrey HE. Effects of *in utero* exposure to polychlorinated biphenyls and related contaminants on cognitive functioning in young children. J Paediatr 1990; 116(1): 38-45.
277) Needleman HL, Leviton A, Bellinger D. Lead-associated intellectual deficit. N Eng J Med 1982; 306(6): 367.

6.4 The Chemically Sensitive

278) Rinsky RA et al. Benzene and leukemia: an epidemiologic risk assessment. N Eng J Med 1987; 316(17): 1044-50.
279) Pelkonenn O. Comparison of activities of drug-metabolizing enzymes in human fetal and adult livers. Clinic Pharmacol Ther 1973; 14(5): 840-6.
280) Hattis D, Russ A, Goble R, et al. Human interindividual variability in susceptibility to airborne particles. Risk Anal 2001; 21(4): 585-99.
281) Friedman R. Sensitive Populations and Environmental Standards. The Conservative Foundation, Washington DC (1981).

Section 7. Past Mistakes and the Precautionary Principle

7.1 The Precautionary Principle

282) European Commission 2000. Communications from the Commission on the Precautionary Principle (COM (2000) 1) Brussels. URL: http://europa.eu.int/comm./dgs/health_consumer/library/pub/pub07_en.pdf (accessed 30 November 2003).
283) Grandjean P, Bailar JC, Gee D, et al. Implications of the precautionary principle in research and policy-making. Am J Ind Med 2004; 45(4): 382-5.
284) Franchini M, Rial M, Buiatti E, Bianchi F. Health effects of exposure to waste incinerator emissions: a review of the epidemiological studies. Ann Ist Super Sanita, 2004; 40(1): 101-15.

7.2 Learning from Past Mistakes

285) Rice DC, Evangelista de Duffard AM, Duffard R et al. Lessons for neurotoxicology from selected model compounds SGOMSEC joint report. Env Health Perspect 1996; 104 (Supp 2): 205-15.

Section 8. Alternative Waste Technologies

8.2 Producing Less Waste

286) Department of Food, Environment and Rural Affairs (2006); Review of England's Waste Strategy: A Consultation Document, London, Defra.
287) Energy Green Paper, Towards a European Strategy for the Security of Energy, European Commission,2001. http://europa.eu.int/comm/energy_transport/doc-principal/pubfinal_en.pdf
288) World Energy Outlook 2001, 2001 Insights, International Energy Agency(IEA) and the Organisation for Economic Co-operation and Development ()ECD), 2001.
[redacted]
289) Mobbs P, Energy beyond Oil, Cronwell Press Ltd, 2005,Trowbridge, UK
290) Cambridge Econometrics, UK Energy and the Environment. March 2008

8.7 Adavanced Thermal Technologies (ATT) and Plasma Gasification

291) Gomez E, Amutha Rani D, Cheesman CR et al. Theral plasma technology for the treatment of wastes: A critical review, 2008, Journal of hazardous Materials, doi:10.1016/j.hazmat.2008.4.017:1-13

8.8 Greenhouse Gases

292) White P, Franke M, Hindle P. Integrated Solid Waste Management: A Lifestyle Inventory, 1994, Blackie Academic And Professional

Section 9. Costs of Incineration

9.1 Direct and Indirect Costs

293) Public Consultation on Waste management Options, Scottish Environmental Protection Agency, Western Isles Strategy Area, Nov 2001
294) Russell M, Colglazier W, Tonn BE. The US hazardous waste legacy. Environment, 1992; 34(6): 13-39

9.2 Health costs of Incineration

295) Energy Technology Support Unit (ETSU), 1996, Economic Evaluation of the draft incinerator Directive, European Commission
296) Final report for DEFRA by Enviros Consulting Ltd in association with ENTEC: Valuation of the External Costs and Benefits to Health and Environment of Waste Management Options. Dec 2004
297) Eshet T, Ayalon O, Schecter M. A Critical Review of Economic Evaluation Studies of Externalities from Incineration and landfilling. Waste management Res, 2005; 23:487-504
298) Miranda ML, Hale B. Waste not, want not, the private and social cost of energy production. Energy Policy, 1997; 25:587-600

9.3 Financial Gains from reducing Pollution

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

300) Pianin E. Study finds Net Gain from Pollution rules. Washington Post, Sept 27th, 2003.

9.4 Other Studies of the Health Costs of Pollution

301) Muir T, Zegarac M. Societal Costs of Exposure to Toxic Substances: Economic and health Costs of Four Case studies that are Candinates for Environmental Causation. Env Health Perspect 2001; 109 (Suppl 6): 885903
302) World Wildlife Fund Report: Compromising Our Children: Chemical Impacts on Children's intelligence and Behaviour, June 2004. www.wwf.org.uk/chemicals

Section 10. Other Considerations of Importance

10.1 The Problem of Ash

303) Markus T, Behnisch P, Hagenmaier H et al. Dioxinlike components in incinerator fly ash: A comparison between chemical analysis data and results from a cell culture bioassay. Environ Health Perspect 1997; 105(12): 475-81

10.2 Radioactivity

304) Beral V, Rooney C,Maconochie N et al. Case control study of prostatic cancer in Employees of the United Kingdom Atomic EnergyAuthority. BMJ,1993; 307: 1391-7
305) Stokke T, Oftedal P, Pappas A. Effects of a small doses of radioactive strontium on the rat bone marrow. Acta Radiologica 1968: 7:321-9
306) Luning KG, Frolen H, Nelson A. Genetic effects of Strontium 90 injected into male mice. Nature 1963; 197:304-5
307) Busby C, Wings of Death: Nuclear Pollution and Human Health, 1995, Green Audit (Wales) Ltd, Aberystwyth
308) Berd V et al (eds.), Childhood Cancer and Nuclear Installations (London, BMJ Publishing Group 1993).
309) Gardner MJ. Childhood leukaemia around the Sellafield nuclear plant. *In* P Elliot et al (eds.) Geographical and Environmental Epidemiology: Methods for Small Area Studies. Oxford, Oxford University Press 1992, pp291-309.
310) Heasman MA, Kemp IW, Urquart JD, Black R. Childhood cancer in Northern Scotland. Lancet 1986; 1 (8475): 266.
311) Roman E, Watson A, Beral V, et al. Case control study of leukemia and Non-Hodgkin lymphoma among children aged 0-4 Years living in West Berkshire and North Hampshire health districts. BMJ 1993; 306(6878): 615-21.
312) Morris MS, Knorr RS. Adult leukemia and proximity-based surrogates for exposure to Pilgrim plant's nuclear emissions. Arch Environ Health 1996; 51(4): 266-74.
313) Clapp RW et al. Leukaemia near Massachusetts nuclear power plant. Lancet 1987; 2(8571): 1324-5.
314) Baker PJ, Hoel DG. Meta-analysis of standardized incidence and mortality rates of childhood leukaemia in proximity to nuclear facilities. Eur J Cancer Care, 2007;16: 355-63

10.3 The Spread of Pollutants

315) National Research Council (2000): Waste Incineration and Public Health ISBN: 0-309-06371-X,Washington DC, National Academy Press.
316) Mittal AK, Van Grieken R, Ravindra.. Health risk assessment of urban suspended particulate matter with special reference to polycyclic aromatic hydrocarbons: a review. Rev Environ Health 2001; 16 (Pt 3): 169-89.
317) Final report to the North American Commission for Environmental Cooperation (Flushing, N.Y.: Centre for the Biology of Natural systems, Queens College, CUNY, 2000).
319) Raloff FJ. Even Nunavut gets plenty of dioxin. Science News 2000; 158 : 230.

10.4 Cement Kilns

319) Personal Communication, Christine Hall, Emission-Watch, Chester
www.emission-watch.com

Section 11. Monitoring

320) Wang L, His H Chang J et al. Influence of start-up on PCDD/F emission of incinerators. Chemosphere, 2007; 67: 1346-53
321) De Fre and Wevers. Underestimation of Dioxin emission inventories.1998: Organohalogen Compounds 36:17-20
322) Farland W, Lorber M, Clevely D. WTI Screening level analysis. Washington DC: US Environmental Protection Agency Office of Research and Development, February 9, 1993.
323) Wates J. The Non-Governmental Organization and Management of Hazardous Waste in Ireland, 1994, Earthwatch, European Environmental Bureau

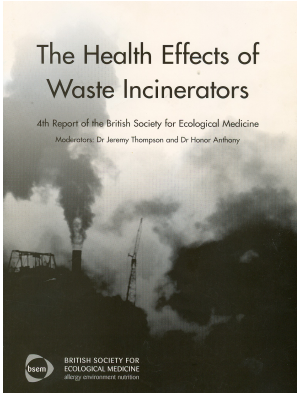
Section 12. Risk Assessment

324) Alleva E, Brock J, Brouwer A et al. Statement from the work session on environmental endocrine-disrupting chemicals: Neural, endocrine and behavioural effects. Erice, Italy: Ettore Majorana Centre for Scientific Culture, 1995.
325) National Research Council (NRC). Toxicity Testing: Strategies to Determine Needs and Priorities (1984), National Academy Press, Washington, D.C.
326) Moore CF. Silent Scourge: Children, Pollution and Why Scientists Disagree. Oxford University Press, 2003, Oxford
327) Bostock A. Waste Incineration and its Impact upon Health, the Environment and Sustainability, 2005, Acro Logic
328) Schettler T, Solomon G, Valenti M and Huddle A. Generations at Risk: Reproductive Health and the Environment, 1999, MIT Press, Cambridge, Massachusetts, & London.

Section 15. Recommendations

329) Friends of the Earth Briefing. Incinerators and Deprivation, Jan 2004.

(This article is also available as a booklet. For a copy please contact BSEM)



Health and environment

Study of the incidence of cancers close to municipal solid waste incinerators

Summary

Pascal Fabre, Côme Daniau, Sarah Goria
Perrine de Crouy-Chanel, Adela Paez
Pascal Empereur-Bissonnet

InVS
FRENCH INSTITUTE FOR PUBLIC HEALTH SURVEILLANCE

Contents

Study of the incidence of cancers close to municipal solid waste incinerators

Summary

Authors

Pascal Fabre, Côme Daniau, Sarah Goria, Perrine de Crouy-Chanel, Adela Paez, Pascal Empereur-Bissonnet

Scientific Coordinator

Pascal Empereur-Bissonnet

Acknowledgements

The team responsible for this study would like to thank all those who made this study possible and contributed to its quality.
We would particularly like to thank the members of the Scientific Committee for the relevance of their advice and for the major contribution they made during the four years of this work.

We would also like to thank:
- Mrs Mireille Chiron, of the French Institute for Research on Transportation and Safety (INRETS), for providing us with valuable information concerning pollution due to road traffic;
- Mr Nicolas Jeannée, of Géovariance, for being generally helpful and providing us with data concerning pollution due to road traffic;
- Mr Yann Martinet, of the Technical Centre of Studies on the Atmospheric Pollution (CITEPA), for providing us with useful information concerning air pollution;
- Mrs Danièle Blot, of the French Meteorological Bureau (Météo France), for the quality of her meteorological advice;
- Mr Frédéric Pradelle, of Numtech, for assistance with the modelling of atmospheric dispersion;
- Mrs Joëlle Le Moal, of the French Institute for Public Health Surveillance (InVS), for advice concerning the epidemiology of cancers;
- The staff of the cancer registries of the Isère, Tarn, Bas-Rhin and Haut-Rhin, for the quality of data concerning cancer cases and their scientific contribution to the study;
- The staff of the Regional Offices of Industry, Research and Environment (DRIRE) of the Alsace, Rhône-Alpes and Midi-Pyrénées regions for their support in the collection of data relating to incinerator function;
- Household waste incinerator operators, who were consulted and supplied the data required for modelling the dispersion of mixtures of pollutants;
- The members of the Expert Committee, which estimated the flow of emissions from incinerators: Patrick Poisseau (Industrial Management of Urban Waste – TIRU), Serge Collet (French Institute of Industrial Environment and Risks – INERIS), Hubert Dechefdebien (National Association of Waste Industry), José de Freitas (Novergie), Emmanuel Fiani, Agency for Environment and Control of Energy (ADEME) and Michel Mori (CGEA Onyx).

Foreword

This document is a summary of the study report, presenting an exhaustive description of the materials and methods used in this work and all the results obtained. The complete report is available online from the InVS website: www.invs.sante.fr/publications

Abbreviations

ADEME	*Agence de l'environnement et de la maîtrise de l'énergie* / Agency for Environment and Control of Energy
ADMS	Atmospheric dispersion modelling system
AFSSET	*Agence française de sécurité sanitaire de l'environnement et du travail* / French Agency for Environmental and Occupational Safety
AhR	Aryl hydrocarbon receptor
CHU	*Centre hospitalier universitaire* / University hospital
ICD-O-2	Version 2 of the international classification of diseases for oncology
DRIRE	*Direction régionale de l'industrie, de la recherche et de l'environnement* / Regional Office of Industry, Research and Environment
FRANCIM	*France-cancer-incidence et mortalité* / French network of cancer registries
GAM	General additive models
PAH	Polycyclic aromatic hydrocarbon
IFEN	*Institut français de l'environnement* / French Institute for Environment
IGN	*Institut géographique national* / National Geographic Institute
IRIS	*Îlots regroupés pour l'information statistique* / Clusters for statistical information
INSEE	*Institut national de la statistique et des études économiques* / French Institute of Statistics and Economic Studies
INSERM	*Institut national de la santé et de la recherche médicale* / French Institute for Health and Medical Research
MNHL	Malignant non-Hodgkin's lymphoma
PCB	Polychlorobiphenyl
PM_{10}	Fine particles with a diameter of no more than 10 microns
TCDD	Tetrachlorodibenzo-*p*-dioxin
MSWI	Municipal solid waste incinerator
INRETS	*Institut national de la recherche sur les transports et leur sécurité* / French Institute for Research on Transportation and Safety
GIS	Geographic Information System
InVS	*Institut de veille sanitaire* / French Institute for Public Health Surveillance
RR	Relative Risk

1. Introduction

1.1 The incineration of household waste

France has been using incineration to eliminate household and similar waste since 1970. According to Ademe, French households produced more than 26 million tonnes of waste (household waste and large objects) in 2003, 35% of which was incinerated [1]. The number of municipal solid waste incinerators (MSWIs) has decreased in recent years, from 292 in 1985, to 213 in 2000 and 135 in 2004 [2].

However, the impact of the rejects discharge from MSWIs on human health remains a subject of concern for French populations living in the vicinity of these industrial installations.

The deleterious effects on health of the pollution generated by MSWIs result from the quantity and type of chemical agents emitted into the air from the incinerator stack. These emissions consist of complex mixtures containing, essentially, sulphur dioxide, nitrogen oxides, hydrochloric acid, heavy metals, dioxins, particles and polycyclic aromatic hydrocarbons (PAHs) [3-8]. Most of these compounds are toxic, and some have demonstrated or suspected carcinogenic properties in humans or animals [9-17].

1.2 Epidemiological justification of the study

Nowadays, all the 135 French MSWI meet the European norms of atmospheric emission [18;19]. Nevertheless, oldest incinerators have contributed to increase the past overall environmental load of dioxins and other persisting pollutants in soils and local food. The atmospheric emissions from incinerators contain various substances individually known or suspected to be toxic for human in chronic exposure situations [5;20-22]. The complex mixtures emitted from MSWI include numerous metals such as cadmium, thallium, lead, arsenic, antimony, chromium, cobalt, copper, manganese, nickel, zinc and mercury [12;23-26]. Information on effects of environmental exposure to metals is limited but some of them are classified as certain or potential carcinogens for humans by the International Agency for Research on Cancer (IARC) [27;28] Airborne particles, nitrogen dioxide, sulfur dioxide and carbon monoxide are also emitted by municipal incinerators [5]. Polycyclic aromatic hydrocarbons (PAHs), released during the incomplete combustion or pyrolisis of organic matter, are associated with cancer occurrence, in particular with lung [29;30], breast and bladder cancers [31;32] and also with non-Hodgkin's lymphomas [17]. Moreover, poorly controlled combustion processes entail the production of dioxins, a class of compounds that includes two chemical families, polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzo-*p*-furans (PCDFs). In 1997, the IARC has classified the most toxic of these compounds, the 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), as a known human carcinogen [10;11]. This statement was primarily based on animal experiments [33], and then has been essentially supported by numerous occupational studies. Becher, in highly dioxin and furan exposed workers, showed an excess risk of mortality by respiratory cancer (standardized mortality ratio (SMR)=1.54; 95% confidence interval (CI),1.15-2.02) [34]. Kogevinas in a historical cohort study showed a higher mortality rate from all malignant neoplasms (SMR=1.12; 95% CI,1.04-1.21) of 21,863 male and female workers in 36 cohorts exposed to phenoxy herbicides, chlorophenols, and dioxins [35]. Hooeiveld in a retrospective cohort study of workers exposed to herbicides containing TCDD and other polychlorinated hydrocarbons showed increased relative risk (RR) for total mortality (RR=1.8; 95% CI,1.2-2.5), cancer mortality (RR=4.1; 95% CI,1.8-9.0), respiratory cancer mortality (RR=7.5; 95% CI,1.0-56.1) and non-Hodgkin's lymphoma mortality (RR=1.7; 95% CI,0.2-16.5) in male workers [36]. Steenland, in a mortality cohort involving 5,132 TCDD exposed workers at 12 US plants observed a SMR of 1.60; 95% CI,1.15-1.82 for all cancers combined in the highest exposure group (30).

This IARC classification has also been supported by data issued from the follow-up of the cohort of the resident population accidentally exposed to nearly "pure" TCDD after the explosion of a plant in Seveso (Italy) which brought additional evidences after a sequential follow-up at 10, 15 and 20 years [37-40]. Fifteen years after the accident, mortality among men increased from all cancers (RR=1.3; 95% CI,1.0,1.7), rectal cancer (RR=2.4; 95% CI,1.2,4.6), and lung cancer (RR=1.3; 95% CI,1.0,1.7). An excess of non-Hodgkin's lymphoma (RR=2.8; 95% CI,1.1,7.0) and myeloid leukemia (RR=3.8; 95% CI,1.2,12.5) were also observed. In women only, lymphohemopoietic neoplasms (RR=1.8; 95% CI,1.8,3.2) and multiple myeloma incidence (RR=3.2; 95% CI,1,2-8.8), were also increased. Twenty years after this industrial accident, an excess of lymphohemopoietic neoplasms was revealed in both genders (RR=1.7; 95% CI,1.2,2.5). The 30 year follow-up of this cohort revealed that the hazard ratio for breast cancer doubled with elevated TCDD serum levels [41].

However, in animal models, as well as in occupational or accidental settings in human, whatever the pollutant, the exposure is characterized by high doses during a relatively short period of time. The question of a potential effect remains open in environmental situation where the humans are daily exposed to extremely low concentrations of pollutants but for long periods of time, often several decades, as is the case for population residing in the vicinity of MSWI. Thus, it is not yet clear whether environmental exposure to MSWI atmospheric release affects the general population. Few studies have been carried out to evaluate the health impact of a long-term exposure of population living close to that type of facilities and some of them were controversial. For instance, Michelozzi in 1998 did not observe, in a suburb of Rome, overall excess or a gradient in risk for liver, lung, and lymphohaematopoietic cancers in either sex, with distance to a waste disposal site, a waste incinerator plant, and an oil refinery plants [42]. On the other hand, Elliott reported in 2000 an excess risk of liver cancer (between 0.53 and 0.78 excess cases per 10^5 per year) for people living within 1 km of 72 municipal solid waste incinerators in Great Britain [43;44]. Biggeri, also in 1996, showed in a case-control population-based study carried out in Italy a positive relationship between distance of homes from an incinerator and lung cancer incidence: p-value=0.0098 with an excess risk of 6.7 [45].

Viel, in 2000, identified clusters of non-Hodgkin's lymphoma and soft tissue sarcoma (STS) from 1980 to 1995 around a MSWI in a French district by applying a spatial scan statistic to 26 electoral wards. The standardized incidence ratios were 1.44 (p-value=0.004)

and 1.27 (p-value=0.00003) for non-Hodgkin's lymphoma and STS, respectively [46]. To complete these results, the authors found in a nested case-control study a.3 times higher risk of non-Hodgkin's lymphomas (95% CI [1.4-3.8]) among individuals living in the area with the highest dioxin airborne concentration [47], but they didn't found any significantly increased risk for STS [48]. Zambon, in a case-control population-based study in Italy with complete residential history, estimated in 2007 that the risk of developing a sarcoma was increased for subjects with the longest and highest exposure to emissions from incinerators and other industrial plants (Odd ratio (OR)=3.3; 95% CI,1.24-8.76) [49]. After complete reconstruction of the residential history of 37 population-based cases and 171 controls of STS, Comba, in Italy, showed a significant increase in risk of STS associated with residence within 2 km of an industrial waste incinerator (OR=31.4; 95% CI,5.6-176.1) [50].

Faced with public awareness and the growing number of epidemiological evidences of the health impact of the environmental pollution due to waste incineration, a working group was set up by the French Institute for Public Health Surveillance (InVS) in 2002, at the request of the Ministry of Health. The aim was to identify epidemiological studies that might help to increase our understanding of the environmental causes of cancer, focusing particularly on the effect of atmospheric emissions from MSWIs on the frequency of cancers in the neighbouring populations. This group recommended, in particular, the implementation of a multicentric study of cancer incidence to ensure a high level of statistical power and to increase the likelihood of observing a wide range of exposure levels.

This was the objective of the study described here, which was funded as part of the 2003-2007 Cancer Plan.

2. Study aims

2.1 Principal objective

The main objective of this study was to investigate the relationship between cancer incidence in the general population and exposure to atmospheric emissions from MSWIs.

2.2 Specific objectives

- To evaluate the exposure of populations to substances released into the atmosphere by MSWIs and to the main risk factors for cancer, during the period extending from 1970 to 1980.
- To estimate, in these populations, the incidence of cancers between 1990 and 1999, for all cancer types and for localisations for which a link has been established or suspected between cancer incidence and MSWIs exposure.
- To quantify the risk of cancer as a function of exposure to the atmospheric emissions from MSWIs.

3. Methods

3.1 Type of study

This epidemiological work was a geographic, ecological study. It analysed, at a collective level, the incidence of cancers as a function of past exposure to the atmospheric discharge from MSWIs. We also tried to take into account other factors potentially contributing to the occurrence of cancers.

3.2 The statistical unit

We conducted this ecological study in four French *"départements"* (administrative district subdividing a Region): Haut-Rhin, Bas-Rhin, Isère, and Tarn, covered by a population-based cancer registry. These districts were chosen according to statistical power and feasibility criteria and to be roughly representative of the overall geographical and socio-economical French heterogeneity. All taken together they were large enough to permit a 10 year observation of 2.5 millions of adults. Given the *a priori* power calculation, this could allow us detecting a RR = 1.1 for leukemias with the power of 80%.

The four districts were divided into 2,270 sub-areas called *"Ilôt Regroupé pour l'Information Statistique"* (IRIS). It was the statistical unit of this ecological study. This entity is a geographical unit defined by the French Institute of Statistics and Economic Studies (INSEE) by dividing up communities of more than 10,000 inhabitants into homogeneous groups of about 2,000 people. For each of these units, various types of information, including socio-demographic data, are available.

3.3 Study periods

This study included three successive periods: a phase in which the populations were exposed to emissions from MSWIs, followed by a latency period compatible with the onset of cancer and, finally, a period of observation in which the incidence of cancers was determined.

- The exposure period was defined as the time between the year in which each incinerator began activity (1972 for the oldest one) and the year at the start of the latency period, according to the year of calculation of the mean cancer incidence (1995), that is 1985 for solid cancers and 1990 for leukaemias.
- The latency period is the minimum period between the start of exposure and the time of cancer diagnosis. Knowledge in this domain remains fragmented. However, based on the references consulted [40;51] the latency period applied for this study was five years for leukaemias and 10 years for other kinds of general cancers.
- The cancer case collection period used for the observation of cancer incidence extended over ten years, from 01/01/1990 to 31/12/1999 inclusive.

3.4 Study population

The incidence of cancers was calculated for the adult population of both sexes aged over 14 years at the time of diagnosis.

3.4.1 Estimation of the required sample size

The population sample size required was estimated from cancer incidence rates for the French population during the study period, using leukaemias as a reference, since these cancers had one of the lowest incidences among the types of cancer studied. We calculated that the observation of cancer incidence during 10 years of 446,700 exposed individuals would give a statistical power of 80%. A sample of this size could be attained by including two or three *départements*.

In practice, in view of data availability and the need to take into account adjustments for confounding factors, we decided to include the population of four *départements*, to maximise statistical power.

3.4.2 Selection of the study zone

In 1999, France had 21 cancer registries belonging to the FRANCIM (France-cancer-incidence and mortality). This network included 10 general cancer registries covering all tumours in 11 *départements* of mainland France: Bas-Rhin, Calvados, Doubs, Haut-Rhin, Hérault, Isère, Manche, Somme, Tarn, Loire-Atlantique and Vendée.

We developed a procedure for ranking *départements* on the basis of a score for each of the following feasibility criteria:

1) Existence of a general cancer registry with validated data for the period 1990-1999;
2) Number of cases of cancer observed during the study period 1990-1999;
3) Availability in digital format of the precise home address of the patients at the time of cancer diagnosis;
4) Number of communities split into IRIS units;
5) Minimal migration rates according to the 1990 census.

The four *départements* considered the most appropriate for study, based on this procedure, were Isere, Haut-Rhin, Bas-Rhin and Tarn (figure 1).

3.5 Types of cancer studied

Incidence rates were estimated for all cancers together and for pre selected subtypes for which a relationship with the exposure to pollutants emitted by MSWI was already suspected or demonstrated in the literature: lung, liver, breast, bladder cancers, soft-tissue sarcomas, myelomas, acute and chronic lymphoid leukemias. Non-Hodgkin's lymphomas

The definition of cancer cases used in this study was that established by Remontet *et al.* [52;53]. Cancer sites were classified as in version two of the international classification of diseases for oncology (ICD-O-2). Only soft-tissue sarcomas were defined using a specific algorithm proposed by E. Desandes from the childhood solid tumours registry.[1]

In this study, we recorded only primary, strictly invasive cancers.

› *All cancers*

The smoke released by MSWIs contains many chemical agents, several of which have been identified as carcinogenic in humans (2,3,7, 8-TCDD, PAHs, heavy metals etc.) and are likely to affect various organs. The carcinogenic potential of one such group of agents, dioxins, has been well documented, but remains a matter of debate [11;54]. The biological mechanisms of action of dioxins is thought to involve the aryl hydrocarbon receptor (AhR), also known as the "dioxin receptor", which is present in many of the cells in the body and plays a role in immune system function and the control of cell proliferation [55;56].

ICD-O-2 characteristics: C00.0 à C80.9
All morphologies
Behavior/3

› *Multiple myelomas*

Multiple myelomas are haematological cancers characterised by a malignant proliferation, of unknown origin, of plasmocytes or their precursors (immunoglobulin-producing B-cell lines). The multiple myeloma-promoting effect of dioxin has been demonstrated in several studies [57] and in the Seveso cohort [38].

ICD-O-2 characteristics: C00.0 à C80.9,
M9730-9732, M9760-9764, M9830
Behavior/3

› *Malignant non-Hodgkin's lymphomas (MNHL)*

This group of cancers includes MNHL, malignant lymphomas of undefined type, lymphosarcomas, reticulosarcomas, microgliomas, peripheral cell lymphomas, B-cell monocytoid lymphomas, angioendotheliomatoses, angiocentric T-cell lymphomas, malignant histiocytoses, Letter-Siwe disease and true histiocytic lymphomas. Epidemiological studies of the general population have provided evidence of a risk of MNHL associated with exposure to smoke from incinerators in France [46;47] Italy [58] and the US [59] and after 15 to 20 years of follow-up in a cohort of individuals exposed to 2,3,7,8-TCDD during an industrial accident at Seveso [40].

ICD-O-2 characteristics: C00.0 à C80.9
M9590-9595, M9670-9723, M9761
Behavior/3

› *Soft-tissue sarcomas*

Soft-tissue sarcomas include all rare tumours of non-bony supporting tissues. Preliminary studies suggesting a relationship between exposure to dioxin and soft-tissue sarcomas were carried out in a work environment in the 1990s [60;61]. Two general population studies were subsequently carried out in Italy, on a small number of cases and controls [49;50]. These studies raised the possibility of a relationship between exposure to emissions from incinerators and the incidence of soft-tissue sarcomas.

ICD-O-2 characteristics: C38.1, C38.2, C38.3, C47, C48.0, C49, C76
M8800, M8801, M8802, M8803, M8804, M8805, M8806, M8810, M8811, M8813, M8814, M8815, M8825, M8830, M8840, M8842, M8850, M8851, M8852, M8853, M8854, M8855, M8857, M8858, M8890, M8891, M8894, M8895, M8896, M8900, M8901, M8902, M8910, M8912, M8920, M8921, M8963, M8990, M8991, M9040, M9041, M9042, M9043, M9044, M9120, M9130, M9133, M9140, M9150, M9170, M9180, M9220, M9231, M9240, M9251, M9252, M9260, M9364, M9580, M9581
Behavior/3

› *Liver cancers*

Liver cancers were defined exclusively as hepatocellular carcinomas and carcinomas of the intrahepatic biliary canal. All other liver tumours were excluded for the purposes of this study. Together with the adipose tissue, the liver is one of the principal sites of storage of organochlorine compounds in the body. A relationship between the role of the AhR and oncogenic mutations in hepatic cells has been demonstrated in several experimental studies in animals [62-64]. P. Elliott showed, in a study of the general population in the United Kingdom, that there was a relationship between living near an incinerator and an excess risk of liver cancer [43;44].

ICD-O-2 characteristics: C22.0 à C22.1
All morphologies
Behavior/3

› *Lung cancers*

Lung cancers included malignant tumours of the trachea, bronchi and lung and contiguous sites to which cancers might extend via the bronchi or pulmonary tissues. Studies of various groups of workers have provided evidence of a relationship between exposure to 2,3,7, 8-TCDD and the risk of lung cancer [35;36;65] General population studies and follow-up studies of the Seveso cohort [40] have also provided evidence in favour of a relationship between exposure to the pollutants released from incinerators and the risk of lung cancer [43;45].

ICD-O-2 characteristics: C33.0 à C34.9
All morphologies
Behavior/3

› *Acute leukaemias*

The term "acute leukaemia" encompasses the acute and subacute forms of leukaemia, aleukaemic forms, acute and subacute myeloid leukaemia, acute lymphoid leukaemia and acute lymphoblastic leukaemia, Burkitt cell leukaemia, erythroleukaemia, acute promyelocytic leukaemia, myelomonocytic leukaemia, acute and subacute monocytic leukaemia, megakaryocytic leukaemia and myeloid sarcomas.

ICD-O-2 characteristics: C00.0 à C80.9,
M9801, M9802, M9804, M9865, M9861, M9862, M9821, M9822, M9826,M9840, M9866, M9867, M9891,M9892, M9910, M9930
Behavior/3

[1] *The national registry of childhood solid tumours – Université Henri Poincaré Nancy 1, Faculté de Médecine 9, Avenue de la Forêt de Haye BP 184, 54505 Vandœuvre-lès-Nancy cedex, France.*

› *Chronic lymphoid leukaemias*

The hypothesis of a relationship between malignant haemopathies and industrial emissions has been raised by several studies: a case-control study in the general population living close to a source of industrial pollution in North America [66], a follow-up study by Eliott in the United Kingdom [43] and studies involving 15 to 20 years of follow-up of individuals accidentally exposed to 2,3,7,8-TCDD in the Seveso cohort [40].

ICD-O-2 characteristics: C00.0 à C80.9, M9823
Behavior/3

› *Bladder cancers*

Several studies in occupational environments and in general populations have provided evidence for a link between the incidence of bladder cancers and exposure to various toxic compounds released by incinerators, including PAHs [67] and dioxins [68]. A similar link has also been reported for environmental exposure to dioxins [66] and for passive smoking [69].

ICD-O-2 characteristics: C67.0 à C67.9
All morphologies
Behavior/3

› *Breast cancers*

Breast cancers were defined as tumours of the connective tissue of the breast, nipple, areole, central area and the four quadrants, axillary extensions and contiguous sites. In a literature review, the hypothesis of a relationship between breast cancer and dioxin exposure was initially rejected [6]. However, an analysis of the women of the Seveso cohort in autumn 2005 [41] revealed for the first time the existence of a highly significant relationship.

ICD-O-2 characteristics: C50.0 à C50.9
Toutes morphologies
Behavior/3

3.6 Collection and processing of data for the observed cancers

The data for cases of cancer diagnosed between 01/01/1990 and 31/12/1999, in patients of both sexes over the age of 14 years, were collected from general cancer registries in the four *départements* participating in the study. The data collected concerned:

- year of birth;
- age at diagnosis;
- gender;
- year of diagnosis;
- topography, morphology and behaviour of the cancer according to the second edition of the International Classification (ICD-O-2);
- postal code and town of residence at the time of diagnosis;
- precise home address at the time of diagnosis (including number, and the name and type of road).

The geographical coding of each cancer case to its IRIS of residence was based on the postal address of the patient at the time of diagnosis: more than 99% of cancer cases were successfully assigned to their IRIS of residence.

All cancer cases were then identified in accordance to their topography, morphology and behaviour ICD-O-2 characteristics. Then, cases of cancer were aggregated by IRIS to obtain the observed cancer incidence at the statistical unit level.

3.7 Estimate of exposure to atmospheric release from incinerators

Several steps were required to estimate retrospectively the level of exposure of statistical units to atmospheric discharge from incinerators.

3.7.1 Identification of sources of emission in the four *départements* studied

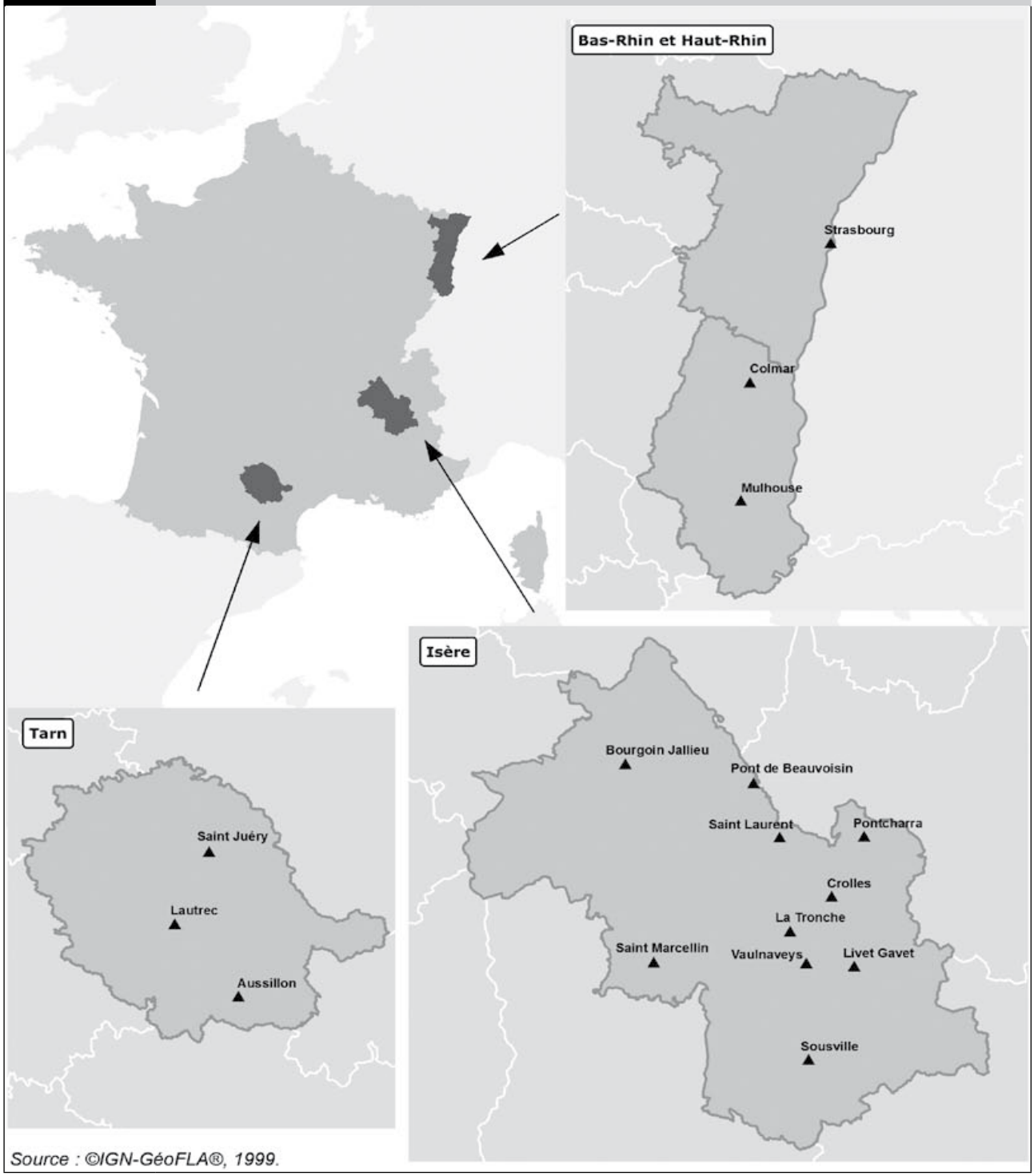
All incinerators operating between 1972 and 1990 in the four *départements* studied were considered. In total, 16 plants functioning during the study period were included (figure 1):

- **10 incinerators in Isère**: the MSWIs of La Tronche, Pontcharra, Bourgoin-Jailleu, Sousville, Livet-Gavet, Saint-Marcelin, Pont-de-Beauvoisin, Saint-Laurent, Crolles and Vaulnaveys;
- **1 incinerator in Bas-Rhin**: the MSWI of Strasbourg;
- **2 incinerators in Haut-Rhin**: the MSWIs of Mulhouse and Colmar;
- **3 incinerators in Tarn**: the MSWIs of Saint-Juéry, Lautrec and Aussillon (also known as Mazamet).

3.7.2 Determination of the technical characteristics of the incinerators

Visits were made to all the incinerators. We asked the operators of the MSWIs and Regional Offices of Industry, Research and Environment (DRIRE) for technical and historical information concerning each of the MSWIs studied, from its opening to the end of the 1990s. The principal data collected were administrative and operating data, information on environmental characteristics and, where available, emission data.

Figure 1 The four *départements* included in the study and their municipal solid waste incinerators (MSWIs)



3.7.3 Retrospective evaluation of the flow of pollutants emitted from incinerator stacks

In the absence of direct measurements of pollutant emission during the exposure of the study period, it was necessary to quantify retrospectively, by an alternative method, the emissions of three main groups of pollutants from each incinerator: particles, heavy metals and dioxins.

This retrospective evaluation of stack emissions was based on the consensus of a group of experts, representing operators, public authorities and a research institution. We used a simplified version of the Delphi method [70], that is an iterative process towards consensus, and took into account the incinerators technical characteristics and their evolution over time: capacity, type of combustion, clearance and filtration processes. This task was performed in three subsequent steps:

- classification of incinerators into eight homogeneous groups according to their technical characteristics, including the nominal capacity of the incinerator, the volume of waste incinerated, the continuous/discontinuous nature of the process, energy recovery, the existence of discharge treatment systems and the age of the installation;
- estimate of the emission flow (in µg/Nm^3) of pollutants released for each of the eight groups of MSWIs;
- the flow values estimated for each of the eight categories of incinerators were then multiplied by the annual tonnage of waste cremated by each incinerator: this gave the emission of each incinerator per µg/s.

The estimated emissions obtained for the three groups of pollutants were used as the input data for the model of atmospheric dispersion.

3.7.4 Modelling of atmospheric dispersion and surface deposition

A Gaussian model was used to model atmospheric dispersion and ground-level deposit within a square grid with unit cells of 200 m × 200 m, centred on the stack.

The extent of the modelling area was adapted to the plant characteristics and its environment, ranging from 20 km × 20 km to 40 km × 40 km. This work was done with the software ADMS version 3 (Numtech®) developed by CERC and UK Meteorological Office (www.cerc.co.uk). It is a second generation Gaussian model: it accounts for the changes in flow field and turbulence around complex terrain and uses them to compute concentrations. This was interesting as a few incinerators in the Isère *département* are located in valleys next to mountains as it can be seen in figure 2. The parameters considered in the modelling process are: estimated flow obtained from the experts, pollutant characteristics, stack height, meteorological data (wind speed and direction, temperature, atmospheric stability) and environmental characteristics such as surface topography and soil roughness. Figure 2 shows an example of cartographic representation of the modelled ground-level deposit of dioxins around one of the incinerators included in the study.

3.7.5 Choice of the indicator pollutant for the substances emitted

Three types of pollutant were initially identified as indicators of emissions from incinerators: a mixture of heavy metals; a mixture of dioxins, furanes and polychlorobiphenyls (PCBs); and a mixture of particles (PM_{10}).

A comparison of emission flow showed a strong statistical correlation between the emission flow of particles and that of heavy metals. Furthermore, during the modelling of atmospheric dispersion, it became clear that there was a strong correlation between the deposition on the soil of particles and that of dioxin, and between atmospheric concentrations of dioxins and dioxin deposits.

We therefore retained, as exposure indicator of emissions and exposure, surface deposits of a mixture of dioxins, furanes and PCBs – expressed in µg I-TEQ (international toxic equivalents; WHO)/m^2/year, which is referred to as "dioxins" hereafter.

3.7.6 Extent and route of exposure

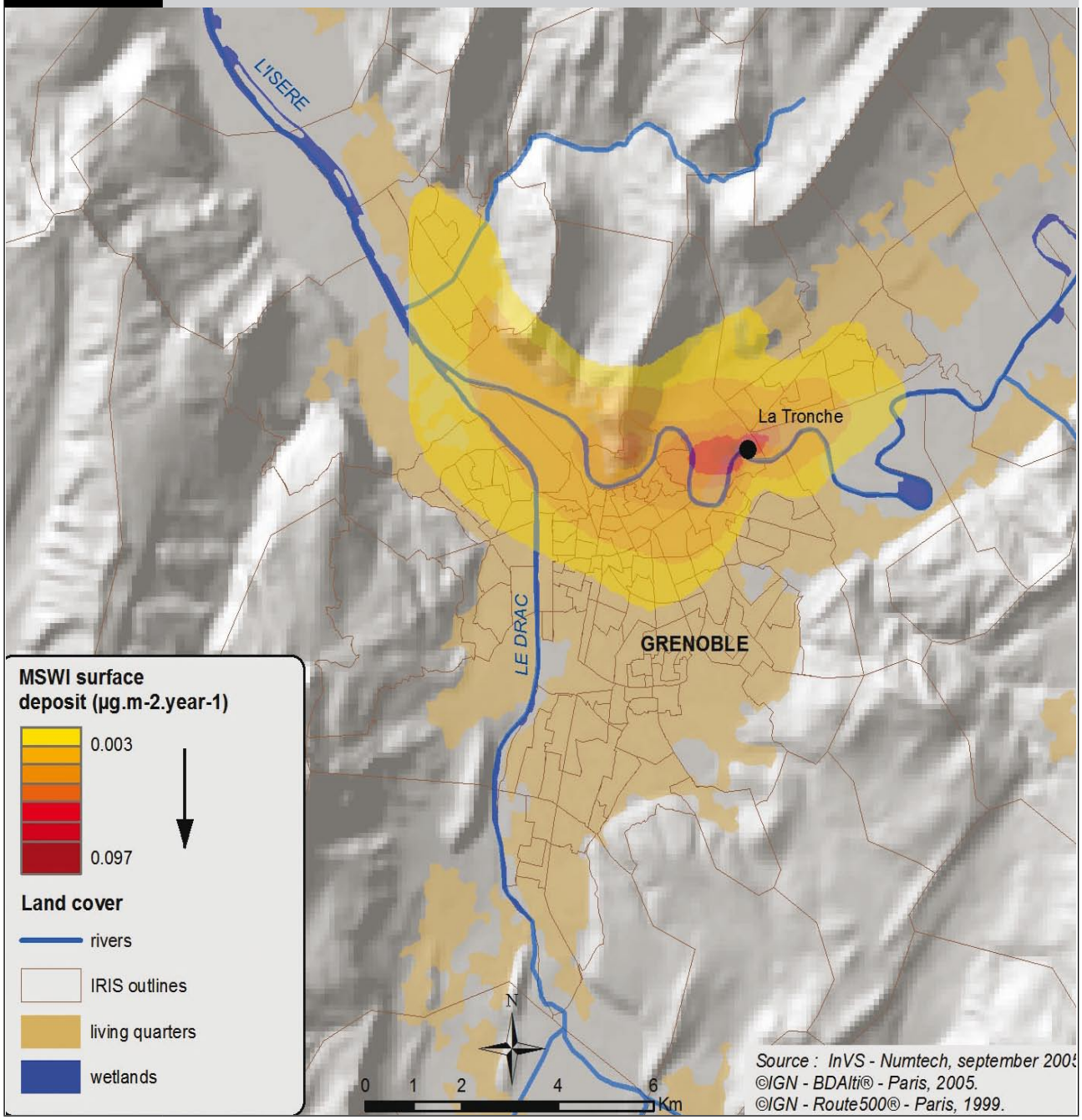
Dioxins persist in the environment and bioaccumulate. Thus the index of exposure was calculated to account for the number of years the plant had operated and the degradation speed in soils. It was defined as the mean of the cumulated ground-level deposits of dioxins since the start of the plant activity (µg I-TEQ/m^2/year). It corresponds to the annual average of the deposits accumulated on the ground surface over all the duration of the incinerators' activity. It was obtained applying an exponential decreasing function with a half-life of 10 years for dioxins in the environment [71].

This exposure variable was considered suitable for representing long-term exposure, and including exposure through the consumption of local products – the principal route of human exposure to dioxins [72-74].

We calculated, for all the IRIS with more than one value from the modelling grid, the median value for all values lying within the contours of the IRIS, with a view to obtaining a single exposure value for each IRIS. For that purpose, a Geographic Information System (GIS) was developped with the ESRI ArcGIS® software. If the area of the IRIS was too small to contain a modelling point, we attributed to the IRIS concerned the value of the point on the modelling grid closest to that IRIS.

The exposure of IRIS located outside the modelling areas was defined, by default, as equal to the smallest median value obtained for all the IRIS located in the modelled areas in the four *départements*. This value (1.85×10^{-5} µg I-TEQ/m^2/year) corresponds to the median value for an IRIS located in Isère.

Figure 2 Example of the modelling of surface deposits of dioxins between 1972 and 1984 around the incinerator of La Tronche (Isère)



3.8 Potential confounding factors

Our analysis took into account five factors known to affect the incidence of cancers that could be described at the level of the IRIS.

› *Socio-economic level*

The socio-economic status of individuals has been recognised as a confounding factor in studies on the effects of environmental exposure to a pollutant [75;76]. Using census information of 1990, the IRIS database (INSEE) and fiscal revenue data for 2001, a socio-economic indicator specific to the study was calculated for each IRIS by principal component analysis (PCA). The 6 socio-economic variables used to were selected according to several existing indices [76-78] and the composite poverty index defined for the Doubs [79;80]. This indicator was itself constructed using the following elements: the proportion of unemployed people, the proportion of low social class households, the proportion of households without a car, the proportion of households who are not owner-occupied, the proportion of public low-cost households and the mean number of people per room (for overcrowding).

› *Population density*

Several authors have suggested that this factor may be linked to the incidence of certain types of cancer [81-83]. The number of inhabitants per unit area of the IRIS was used to calculate population density (inhabitants/km^2).

› *The urban/rural status of the site of residence*

Each town was classified into one of the categories of a complex indicator, established by Insee: urban centre, monopolarised periurban community, multipolarised community, largely rural area.

› *Pollution from road traffic*

Many studies have addressed the possible carcinogenic effects of atmospheric pollutants from cars [84-86], justifying the consideration of this source of pollution as a putative confounding factor. However, the absence of precise and exhaustive measurements of road traffic pollution during the 1970s and 1980s, heterogeneity in vehicle counting data and the disparity of information concerning spatial changes in the road network in each *département* precluded the retrospective quantification of atmospheric pollution from road traffic at the scale of the IRIS. A proxy for exposure to road traffic pollution was defined by nitrogen dioxide (NO_2) concentrations (expressed in µg/m^3) that were taken to be a marker of road traffic emitted cancerigenic pollutants. The data were obtained from the WHO study [87]) supplied by The Agency for Environment and Control of Energy (ADEME): NO_2 concentrations were estimated on a grid of 4 km × 4 km unit cells covering the whole territory of France. These estimations were obtained by cokriging using observed NO_2 concentrations – year 2000 – and information about land use. These data were implemented in the GIS to be used and to define the variable at the IRIS level.

› *Industrial pollution*

Exposure to carcinogenic agents released into the atmosphere from industries classified for the protection of the environment was taken into account (as a function of the type of cancer because it determined the latency time), for the period 1972-1985 (solid tumors), or for the period 1972-1990 (leukaemias). An exposure index to industrial pollution, expressed per industry-years, was defined as the number of existing industries every year in each IRIS. For communities broken down into IRIS, we divided the number of industry-years by the area of the IRIS.

3.9 Statistical analysis

Cancers at all the sites considered were analysed for both sexes together and for the two genders separately. However, cancers for which a difference in incidence between the sexes was expected (all cancers considered together, lung cancer and bladder cancer) were analysed only for the two sexes separately. Cancers at rare sites (soft-tissue sarcomas) were analysed only for both sexes together.

3.9.1 Statistical models

The association between the number of cases of cancer in a given IRIS and the indicator of exposure to incinerators was estimated by a regression analysis, taking the potential confounding factors into account.

Because the observed number of cases are small, Poisson regressions Generalized additive models (GAMs) were used to assess the associations between the risk of cancer and the index of exposure to MSWIs. The models were fitted with an offset as the expected number of cancers [88]. These models are appropriate for exploring forms of associations between the risk of cancer and the exposure to MSWIs or the confounders without presupposing the shape, for example, linear. We used GAMs with penalized cubic regression splines; the degree of smoothness of model terms is estimated as part of fitting [89;90]. The covariates were selected through the Akaike criterion [91]. We proceeded in several steps. At first, residual variation was taken into account by fitting a Poisson regression model allowing for overdispersion. After fitting standard Poisson regressions, we modelled the overdispersion in a hierarchical Bayesian framework which is well adapted to the analysis of disease risk on a small geographical scale [92-94]. It allows integrating, in the estimation of the unknown relative risks, local information consisting of the observed and expected number of cases in each area, the value of the variable of interest and of the potential confounding factors and prior information on the overall variability of the relative risks.

The approach we followed, suggested by Besag *et al.* [95], splits the extra-Poisson variation in two components. The first component of variation is the spatially unstructured extra-Poisson variation, called heterogeneity. Modelling the heterogeneity variation allows for unmeasured variables that vary between areas in an unstructured way. The second component of variation, called clustering, varies smoothly across areas. Modelling the clustering variation allows for those unmeasured risk factors that vary smoothly with location.

The significance threshold was fixed at 5%.

These analyses were carried out using the R package mgcv [100] and WinBUGS [99].

3.9.2 Variables introduced into statistical models

- Number of observed cases per IRIS.
- Number of expected cases per IRIS (offset).
- Effect of *département*. It was included in all models (the reference *département* was Isère).
- Index of exposure to incinerators: square root of the mean cumulative annual deposits of dioxins (µg I-TEQ/m²/year), estimated at the scale of the IRIS.
- Population density, calculated at the scale of the IRIS (number of inhabitants per km²).
- Socio-economic indicator, estimated at the scale of the IRIS.
- Urban/rural indicator, available for IRIS level (four classes).
- Indicator of exposure to road traffic estimated at the level of the IRIS: concentration of NO_2 in the air (µg/m^3).
- Indicator of exposure to other industrial pollutants, calculated at the scale of the IRIS (number of industry-years).

The expected number of cases per IRIS was calculated in several steps:

1) population size per IRIS, per sex and per five-year age group was estimated for the year 1995 from data for the 1990 and 1999 censuses supplied by INSEE, by applying the "single diagonal" method to each age group; this estimate was used as denominator,
2) a reference incidence rate for each five-year age group and for each sex was then calculated from the cases of cancer recorded between 01/01/1990 and 31/12/1999 in the four cancer registries (Isère, Bas-Rhin, Haut-Rhin, Tarn), plus from those recorded in the registries of Doubs and Hérault. The 2 additional *départements* were taken to have more stable reference rates,
3) finally, the expected number of cases per IRIS was calculated from these reference incidence rates and population sizes per IRIS for 1995.

3.9.3 Expression of the results

The results of the study are expressed as relative risks (RR), comparing the risk of a cancer occurring in highly exposed zones with that for zones of low-level exposure.

High exposure is defined as the 90th percentile (P90) for the distribution of the 520 IRIS located within the modelled zones: only 4% of the total population of the four *départements* had a level of exposure equal or higher than this level.

Low-level exposure is defined as the 2.5th percentile (P2.5) for the distribution of IRIS within the modelled zones: 35% of the total study population was exposed to levels no higher than P2.5.

For each type of cancer, the coefficient of regression of the indicator of exposure to the MSWI obtained from the model was used to calculate the relative risk associated with an increase in the indicator of exposure from P2.5 to P90 for the distribution of the 520 IRIS located within the zones modelled.

4. Implementation of the study

4.1 Project team

This study was carried out by an interdisciplinary team composed of epidemiologists, risk assessors, a biostatistician specialized in spatial analysis, and modelling and geomatic engineers.

- Scientific coordination: Pascal Empereur-Bissonnet.
- Project leader: Adela Paez then Pascal Fabre.
- Retrospective quantification of exposure: Côme Daniau.
- Statistical analysis: Sarah Goria.
- Development of the GIS and mapping: Perrine de Crouy-Chanel and Lilias Louvet.
- Data collection: Jamel Daoudi and Béatrice Declercq.
- Secretary: Frédérique Suzanne and then Béatrice Jaillant.

4.2 Scientific Committee

This study had the support of a Scientific Committee. The principal missions of which were to evaluate the study protocol, to help resolve methodological difficulties encountered by the project team during the study, and to validate the results obtained. This committee consisted of the following individuals:

- Nathalie Bonvallot, followed by Sabrina Pontet and Cédric Duboudin, French Agency for Environmental and Occupational Safety (AFSSET);
- Pascal Brula, Polden-Insavalor;
- Marc Colonna, Isère cancer registry;
- Sylvaine Cordier, U625/French Institute for Health and Medical Research (INSERM);
- Hélène Desqueyroux, Agency for Environment and Control of Energy (ADEME);
- Pascal Empereur-Bissonnet, Department of Health and Environment/ InVS;
- Pascal Fabre, Department of Environmental Health/InVS;
- Guy Launoy, French network of cancer registries (FRANCIM);
- Martine Ledrans, Department of Environmental Health/InVS;
- Sylvia Richardson, Imperial College of London, United Kingdom;
- Florence Suzan, Department of Chronic Diseases and Injuries/InVS;
- Jean-François Viel, Faculty of Medicine, Besançon, France.

4.3 Communication Committee

This committee met twice to advise the project team on aspects relating to the communication of the results of the study to the scientific community and to the population.

In addition to those in charge of the study, this committee included members from the Communication Department of the InVS, representatives of the Ministry of Health and ADEME and members of the Scientific Committee.

4.4 Partnerships

Scientific collaboration or service contracts were established between the InVS and:

- CHU of Besançon;
- The cancer registries of Bas-Rhin, Haut-Rhin, Tarn and Isère;
- The French Meteorologial Bureau *(Météo France)*;
- The French Institute for Environment (IFEN);
- INSEE;
- The National Geographic Institute (IGN);
- Géocible;
- Numtech;
- The Polden-Insavalor Group.

5. Results

5.1 Estimate of the intercensus population in 1995

The total population of individuals over the age of 14 years in the four *départements* studied was estimated to be 2,487,274 for 1995. The observation of this population over a ten-year period therefore corresponds to approximately 25,000,000 person-years.

Table 1 shows the estimated population for 1995, for each *département*. The four *départements* studied include a total of 2,270 IRIS. The atmospheric release from 13 incinerators for the study of solid cancers between 1972 and 1984 covered 23% of these IRIS, 520 in total, corresponding to 35% of the total estimated study population in 1995.

5.2 Cancer cases observed during the study period

In total, just over 135,000 cases of cancer in adults were recorded in the four *départements* between 01/01/1990 and 31/12/1999.

Table 2 shows the number of observed cases for each type of cancer studied, for both sexes, with the exception of breast cancer, which affected only women.

Table 1 Total number of IRIS, and of exposed IRIS between 1972 and 1984, and the estimated population for each *département* in 1995

	Isère	Bas-Rhin	Haut-Rhin	Tarn	Total
Total number of IRIS	682	711	488	389	2,270
Number of exposed IRIS (%)	255 (37)	129 (18)	82 (17)	54 (14)	520 (23)
Population	844,366	802,082	554,373	286,453	2,487,274
Exposed population (%)	413,739 (49)	248,645 (31)	155,224 (28)	60,55 (21)	877,763 (35)

We note that the four *départements* included in this study are quite heterogeneous: Isère is a urban department, it is the most populated (around 850,000 inhabitants), the most exposed to MSWIs (50% of the exposed IRIS are in Isère) and with the highest values of exposure.

On the contrary, Tarn is a rural department, it is the least populated (around 290,000 inhabitants), the least exposed (10% of the exposed IRIS are in Tarn) and with the lowest values of exposure.

Table 2 Number of cancers observed (for both sexes, except for breast cancer) for 1990-1999

	Isère	Bas-Rhin	Haut-Rhin	Tarn	Total
All cancers	41,809	45,343	30,868	17,103	135,123
Breast cancer (women)	6,187	6,267	4,293	2,077	18,824
Lung cancer	4,169	4,694	2,918	1,565	13,346
MNHL	1,324	1,333	871	446	3,974
Liver cancer	975	929	700	180	2,784
Soft-tissue sarcoma	221	208	132	94	655
Acute leukaemia	443	350	309	136	1,238
Chronic lymphoid leukaemia	376	356	369	161	1,262
Multiple myeloma	578	454	435	233	1,700
Bladder cancer	1,456	1,744	1,141	770	5,111

5.3 IRIS exposure

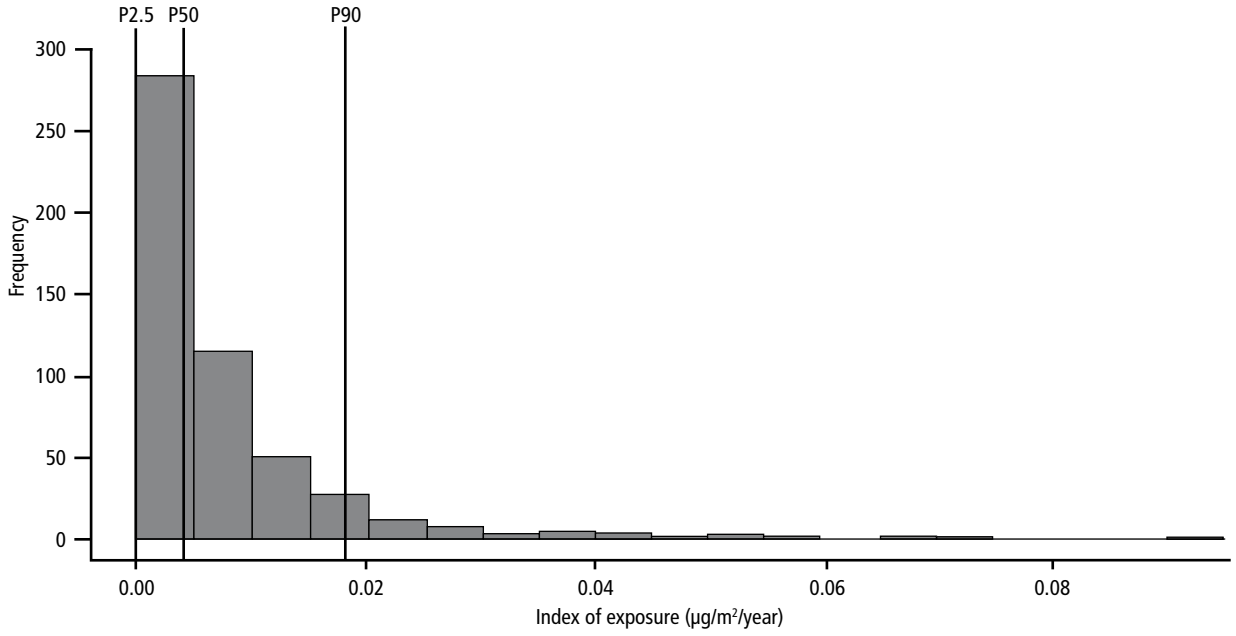
Table 3 shows the distribution of the exposure indicator "mean cumulative annual deposits" for dioxins (µg I-TEQ/m²/year) for the 1972-1984 period, for all the areas modelled.

Table 3 Indicator of IRIS exposure for the period 1972-1984

	Exposure indicator (µg/m²/an)	No. of IRIS (%) with an exposure value ≥ the percentile	% of the population with exposure values ≥ the percentile
Minimum	2.04×10^{-5}	520 (22.9)	35.5
Percentile 2.5	1.25×10^{-4}	507 (22.3)	35.0
Percentile 50	4.25×10^{-3}	260 (11.4)	19.8
Percentile 75	8.93×10^{-3}	130 (5.7)	9.8
Percentile 90	1.78×10^{-2}	52 (2.3)	3.9
Maximum	9.18×10^{-2}	1 (0.04)	0.1
Mean (standard deviation)	7.86×10^{-3} (1.09×10^{-2})	-	-

Figure 3 presents the distribution of mean cumulative annual dioxin deposits for the 520 IRIS for which exposure was modelled over the period 1972-1984. This distribution is highly asymmetric and shows that a large proportion of IRIS were subject to low levels of exposure. By contrast, only a few IRIS had high levels of exposure. We carried out a square root transformation of the exposure variable to prevent these few sites with high levels of exposure having too great a weighting in the statistical analysis.

Figure 3 Distribution of exposure to dioxins released from incinerators, for the IRIS for which exposure was modelled



5.4 Results of the statistical analysis

Table 4 shows the results for all the types of cancer studied, the regression coefficient for the exposure indicator and its standard deviation, statistical significance (p-value), and the number of cancers observed, for each type of cancer and for each sex.

In women, a significant and positive association was demonstrated for "all cancers" (p=0.01) and for breast cancer (p=0.03) and MNHL (p=0.03).

In men, a significant, positive association was observed only for multiple myeloma (p=0.05).

Taking both sexes together, there was a significant positive relationship for MNHL (p=0.04) and non-significant (at the 5% level, but close to this threshold) positive relationships for three other types of cancer: soft-tissue sarcoma (p=0.07), liver cancer (p=0.07) and multiple myeloma (p=0.10).

Table 4 Results of regression modelling by cancer type and by sex

	Regression coefficent	Standard deviation	p-value	No. of cases observed
All cancers, women	**0.502**	**0.223**	**0.01**	**59,076**
All cancers, men	0.237	0.224	0.30	76,047
Breast cancer, women	**0.680**	**0.320**	**0.03**	**18,824**
Lung cancer, women	0.867	0.736	0.24	1,983
Lung cancer, men	0.430	0.445	0.34	11,363
MNHL, women + men	**0.925**	**0.459**	**0.04**	**3,974**
MNHL, women	**1.340**	**0.628**	**0.03**	**1,827**
MNHL, men	0.106	0.625	0.86	2,147
Liver cancer, women + men	1.204	0.662	0.07	2,784
Liver cancer, women	1.342	1.022	0.19	511
Liver cancer, men	1.020	0.700	0.14	2,273
Soft-tissue sarcoma, women + men	1.594	0.887	0.07	655
Multiple myeloma, women + men	1.161	0.709	0.10	1,700
Multiple myeloma, women	0.347	0.984	0.72	811
Multiple myeloma, men	**1.597**	**0.823**	**0.05**	**889**
Acute leukaemia, women + men	0.269	0.731	0.71	1,238
Acute leukaemia, women	0.767	1.007	0.45	592
Acute leukaemia, men	-0.324	1.023	0.75	646
Chronic lymphoid leukaemia, women + men	0.928	0.817	0.26	1,262
Chronic lymphoid leukaemia, women	1.275	1.192	0.28	541
Chronic lymhoid leukaemia, men	0.597	1.097	0.59	721
Bladder cancer, women	-1.631	0.854	0.06	997
Bladder cancer, men	-0.446	0.477	0.35	4,114

The figures in bold correspond to relationships significant at the 5% level (p≤0.05).

5.5 Relative risks

The association between the risk of cancer and exposure to atmospheric emissions from incinerators is presented in table 5 in the form of relative risks of cancer for an increase in the exposure indicator from the 2.5th percentile to the 90th percentile in the distribution of exposed IRIS. For statistically significant relationships, these results correspond to an excess relative risk of between 6% for "all cancers" in women and 23% for multiple myeloma in men.

Table 5 Relative risk (RR) of cancer (and 95% confidence intervals [95% CI]) for an increase in exposure from the 2.5th percentile to the 90th percentile, by type of cancer and by sex

	RR	[95% CI]
All cancers, women	**1.06**	**[1.01-1.12]**
All cancers, men	1.03	[0.97-1.09]
Breast cancer, women	**1.09**	**[1.01-1.18]**
Lung cancer, women	1.11	[0.93-1.33]
Lung cancer, men	1.05	[0.95-1.18]
MNHL, women + men	**1.12**	**[1.00-1.25]**
MNHL, women	**1.18**	**[1.01-1.38]**
MNHL, men	1.01	[0.87-1.18]
Liver cancer, women + men	1.16	[0.99-1.37]
Liver cancer, women	1.18	[0.92-1.52]
Liver cancer, men	1.13	[0.96-1.35]
Soft-tissue sarcoma, women + men	1.22	[0.98-1.51]
Multiple myeloma, women + men	1.16	[0.97-1.40]
Multiple myeloma, women	1.05	[0.81-1.35]
Multiple myeloma, men	**1.23**	**[1.00-1.52]**
Acute leukaemia, women + men	1.04	[0.86-1.25]
Acute leukaemia, women	1.11	[0.85-1.43]
Acute leukaemia, men	0.96	[0.74-1.25]
Chronic lymphoid leukaemia, women + men	1.13	[0.91-1.39]
Chronic lymphoid leukaemia, women	1.18	[0.87-1.61]
Chronic lymphoid leukaemia, men	1.08	[0.82-1.43]
Bladder cancer, women	0.82	[0.66-1.00]
Bladder cancer, men	0.95	[0.84-1.06]

The figures in bold indicate relationships statistically significant at the 5% level (p≤0.05).

6. Discussion

This ecological geographic study concerned about 135,000 cases of cancer occurring in four *départements* of mainland France between 1990 and 1999. It demonstrated the existence of a significant positive relationship between exposure to the atmospheric emissions from MSWIs and the incidence of breast cancers, MNHL and "all cancers" in women. A significant positive relationship was also found for multiple myeloma in men, and for MNHL in both sexes. The study also showed, for both sexes, that there is a link close to the limits of statistical significance between environmental exposure to the emissions from MSWIs and soft-tissue sarcoma, liver cancer and multiple myeloma.

6.1 Internal validity of the results

6.1.1 Estimate of the incidence of cancers

The validity of the incidence rates for cancers calculated in this study is guaranteed by the quality and reliability of the data supplied by the registries. Grouped together into the Francim network, they apply the European guidelines for the standardisation and registering of cancers published in 2003 by the European Network of Cancer Registries [101]. The remarkable rate of geocoding of cancer cases to IRIS obtained, exceeding 99%, illustrates the high quality of the data provided by the registries, particularly as concerns home address.

The reference incidence rates for cancers used were calculated from the data from six registries, four of which corresponded to the four *départements* of the study. The populations in which these rates were measured included people exposed to emissions from incinerators. This may have decreased the difference between the numbers of expected and observed cases of cancer, leading to underestimate the exposure-risk relationships identified for cancer.

Scientific knowledge concerning latency times for cancers following environmental exposure remains limited. The values used here – five years for leukaemia and 10 years for solid cancers – were chosen on the basis of previous publications [51], as well as for operational reasons. However, the most recent publications providing information about latency times for cancers for environmental health, carried out in general populations exposed to urban traffic pollution [102], chronic industrial pollution [103] or accidental pollution [40], have reported lags of 15, or even 20 years. The latency periods used for the cancers considered in our study may therefore be too short. If this is the case, and if the MSWI exposure and cancer incidence relationship is thrue, then all cancers induced by exposure to the pollutants emitted by incinerators would not have had the time to form or to reach a detectable level. This potential bias may lead to the underestimate of the observed relationships.

6.1.2 Estimate of exposure to atmospheric release from incinerators

The flux of emissions from MSWIs stacks was evaluated by consensus between experts, obtained with a modified version of the Delphi method. The retrospective evaluation of dioxin emissions led to the greatest discussion. We compared these estimates with flow values for eight incinerators for which real measurements were taken over the period 1994-2000 as part of another study [104]. The flow values estimated for the most polluting incinerators seem to have been underestimated by the experts, potentially decreasing the difference in emission levels between the MSWIs studied here. However, the gradients of emissions and deposits were largely similar between incinerators, so this underestimate should not affect the exposure-risk relationships observed. Indeed, the impact of this potential error on the numerical value of relative risks is probably low, the estimate of these risks being based on a comparison between two percentiles after square root transformation of the exposure variable. Conversely, this limitation indicates that the exposure-risk relationships calculated cannot be transposed to data for current emissions.

We used dry and wet deposits on the soil of a mixture of dioxins, furanes and PCBs as an indicator of IRIS exposure to the pollutants discharged by MSWIs. Nonetheless, the relationships observed in this study between the incidence of cancers and exposure to emission from incinerators cannot be attributed either to these substances alone or to a particular route of exposure.

The median of all the points on the modelling grid corresponding to a given IRIS was used to describe the level of exposure of each statistical unit. However, we cannot rule out the possibility that this type of central indicator, by homogenising exposure over the whole IRIS, may have introduced a non-differential bias leading to underestimate of the observed relationships.

A default value for exposure was attributed to each IRIS located outside the zone modelled. This value corresponded to the lowest median deposition level obtained for the IRIS located in the zone modelled in the four *départements*. This arbitrary choice may have distorted the results obtained. It may have introduced a non-differential bias by diluting the observed effects.

6.1.3 Other factors taken into account

We used atmospheric NO_2 concentrations for 2000 as a proxy of exposure to carcinogenic agents released into the air by motor vehicles [105]. The use of these data is nonetheless based on the

assumption that atmospheric NO_2 concentrations changed little, if at all, between the exposure period (1970s and 1980s) and the year 2000. Although the construction of stretches of motorway, bypasses and ring roads affect local air quality, it is reasonable to consider that generally, relative changes in the atmospheric concentration of NO_2 have been homogeneous over the entire study zone.

The indicator of industrial pollution used in this study imperfectly reflects the true exposure of an IRIS located at some distance from a polluting installation in the same *département* or, conversely, of an IRIS located close to an industrial installation in a neighbouring *département*. Nonetheless, it is the only indicator we could find to take into account exposure to past industrial pollution at the level of our statistical unit.

6.1.4 Statistical analysis

We used GAMs and hierarchical Bayesian models. GAMs make it possible to take into account possible non-linear effects of variables. Hierarchical Bayesian models, with their heterogeneity and spatial components, can be used to take into account unknown or unmeasured risk factors. In particular, the modelling of a spatially structured source of variation made it possible to take into account the effect of variations in risk factor clustering over the geographical area. This was important, given the high level of extra-Poisson variability.

Differences were found between the four *départements* studied. The much larger contribution of Isère than of the other three *départements* to the results obtained should be stressed. This *département* is the most populous (850,000 inhabitants), contains the largest number of Irises exposed to incinerator emissions (50% of all the exposed IRIS in this study) and had the highest exposure values. Conversely, Tarn, which is mostly rural, has the lowest population (290,000 inhabitants), the lowest level of exposure (10% of the IRIS exposed in this study) and the lowest exposure values. This heterogeneity is partly taken into account by covariables. An effect of *département* was introduced into all models. Regression coefficients for the exposure indicator were calculated for each *département* (interaction between the effect of *département* and the exposure indicator), but did not differ significantly (α=0.05) from that for Isère.

6.1.5 Conclusion concerning the internal validity of the results

6.1.5.1 Limitations

This is an ecologic study, that does not deal with individual subjects or individual level traits or exposures, but rather with the characteristics of block groups. Indeed, it was not possible to take into account individual risk factors known to be strongly associated with the incidence of certain cancers: alcohol and tobacco consumption, occupational exposure, exposure associated with housing and leisure activities, medical treatments, eating habits and the origin of food. Similarly, we had no information concerning the recent residential history of the people concerned.

However, there is no reason to expect the distribution of these individual risk factors to be associated with a particular level of exposure. Furthermore, it is unlikely that residential mobility differed between those with and without cancers.

The various biases that may affect our study would probably result in an overall underestimate of the exposure-risk relationships observed.

6.1.5.2 Strong points

First, this study used a population-based design. Cases were actively identified through multiple sources within defined geographic areas and benefited from a very high georeferencing rate. Compared to other ecological studies on populations living close to incinerators [41-43;46;49], the statistical power obtained from the follow-up of approximately 25 million person-years is one of the strong points of this ecological-type study. Such power made it possible to enhance the several statistical relationships found.

The analyses carried out identified the associations classically found between lung cancer and low socio-economic level, and inversely, between breast cancer in women and high socio-economic level, or between liver cancer and living in a rural environment (data not shown). This consistency with established knowledge suggests that the quality of the means of observation and analysis was high.

Finally, an analysis of sensitivity after excluding extreme values for exposure was conducted, and showed that the exposure-risk relationships observed were stable.

These findings provide solid evidence to support the validity of the results of this epidemiological study.

6.2 Consistency with the literature and interpretation of the relationships observed

The statistical relationship between exposure to emissions from MSWIs, and the incidence of all cancers in women has not previously been reported in a general population. This overall carcinogenic effect may reflect the large number of chemicals emitted from incinerators. However, it remains unclear why this increase in cancer incidence affected essentially women. It can be assumed that women, particularly in the 1970s and 1980s, were more sedentary than men, and less exposed to occupational risks or to certain other risk factors, such as smoking and alcohol consumption, that may have concealed the effect of exposure to incinerator emissions in men in this study. There may also be a hormonal explanation, as the toxocological relationships between oestrogens and the intranuclear receptor AhR in the control of cell proliferation and hormonal balance seem to be well established [55;56;106-108].

This study showed, for the first time in the general population, that exposure to the agents emitted by MSWIs may be an environmental risk factor for breast cancer in women. Studies in occupational settings in Russia [109;110] and Germany [110] had already shown an excess risk of breast cancer in female pesticide industry workers exposed to dioxin and furane residues. Nonetheless, conflicting results have been obtained concerning the effects of exposure to dioxin on breast cancer. A deficit of breast cancers was initially reported at Seveso [39], after a 10 year follow-up of the cohort, whereas other studies have suggested that long-term exposure to 2,3,7,8-TCDD may be associated with high breast cancer rates [111;112].

Finally, it should be noted that, in our study, the exposure-risk relationship for all cancers in women persisted, even if breast cancers were excluded from the analysis (data not shown).

The significant positive relationship between exposure to atmospheric emissions from incinerators and the incidence of MNHL is consistent with the results of cluster and case-control studies carried out in the general population living around the incinerator of Besançon [46;47]. These observations should be compared with those made during the follow-up of the Seveso cohort, in which MNHL in men seemed to be exclusively linked to accidental exposure to 2,3,7,8-TCDD [40]. In our analysis by sex, the association between the risk of MNHL and exposure to incinerator emissions was statistically significant in women, but not in men. Is there an environmental or hormonal explanation or are women particularly susceptible due to a specific gene-environment interaction [113]? This study cannot provide any explanation as to the female nature of the relationship observed in this study.

The non-significant positive association (p=0.07) observed for the risk of soft-tissue sarcomas is consistent with the results of case-control studies carried out in the area around a MSWI in France [46] in an area around an industrial waste incinerator in Italy [50] and around industrial sources of dioxins, including incinerators [49].

Similarly, the positive relationship, close to the significance threshold (p=0.07) observed between liver cancer and exposure to incinerator emissions is consistent with the results of a study of incidence based on data from registries in the United Kingdom, in a general population living close to incinerators [43;44].

The positive association observed for both sexes between the risk of multiple myeloma and exposure to incinerator emissions, which was not significant at the 5% level (p=0.10), reflects an excess relative risk of 16%. Our analysis by sex suggested that this association resulted from a significant relationship for men (p=0.05). Our observations are consistent with the results obtained after 15 years of follow-up in the Seveso cohort [38]. They are also similar to those obtained in studies carried out in Sweden on cohorts of fishermen consuming large quantities of fish contaminated with organochlorine compounds, including dioxins [114;115].

We obtained no evidence for a significant association with lung cancer in either of the sexes. Our analysis shows that the covariates included in the models (economic score, road traffic and population density) played a key role in determining the incidence of lung cancer.

We found no relationship between acute or chronic leukaemia and exposure to emissions from incinerators, whereas a relationship of this kind was reported for the Seveso cohort [38;40].

Finally, this study showed a negative relationship between the risk of bladder cancer and exposure to incinerator emissions in women but this relationship is difficult to explain.

6.3 Implications of the study results

This ecological study provides new elements suggesting that past exposure to the pollutants emitted by incinerators has an effect on health, but it is not possible to presume a causal link from these observations. In addition, it should be noted that we used an exposure indicator identifying neither the substances involved, nor the route of exposure responsible for the relationships observed.

In terms of public health, excess risks observed should be interpreted depending on the number of people subject to the various situations of exposure.

Indeed, the relative risks for IRIS exposed to the 90th percentile (corresponding to 100 times background levels) concerned only 4% of the total population. The relative risks identified in IRIS exposed to the 50th percentile (4.25 x 10^{-3} µg I-TEQ of dioxins/m^2/year) were lower (results not presented here), but concerned 20% of the total population studied.

Thus, the relative risk of breast cancer in women, for an increase in exposure from the 2.5th to the 90th percentile, was 1.09, whereas the relative risk for an increase in exposure from P2.5 to P50 was 1.04. In similar conditions and for all cancers in women, the risk decreases from 1.06 to 1.03. For MNHL in women, relative risk was 1.18 for an increase in exposure from P2.5 to P90 and 1.07 for an increase in exposure from P2.5 to P50. For multiple myeloma in men, the corresponding relative risks were 1.23 and 1.08.

Overall, for all the types of cancer for which we found significant relationships to past exposure to incinerator emissions, the excess relative risk of cancer for an increase in exposure from P2.5 to P50 was two to three times lower than that for an increase from P2.5 to P90. Nonetheless, this lower risk concerned a population five times larger. There is therefore a clear public health risk due to the number of people potentially affected, rather than an individual risk.

It would be difficult to transpose the exposure-risk relationships identified in this work outside our study zones. Indeed, the four *départements* studied do not adequately reflect the heterogeneity of the French population and the exposure-risk relationships demonstrated include multiple interactions with demographic, economic and cultural factors that are difficult to identify and to control.

Similarly, the level of exposure to incinerator emissions, which was quantified retrospectively in our study by an expert panel consensus, has only a relative value. The exposure-risk relationships calculated based on these estimates cannot be exploited with data generated by other quantification methods. In addition, the relationships identified refer to particular exposure and latency periods between 1972 and 1990, with characteristics (environmental and professional exposure, demographics, socio-economic, cultural and health context) different from those of today.

7. Recommendations

7.1 Improvements in epidemiological knowledge

Work towards three objectives could be valuable to improve knowledge concerning the relationship between incinerator emissions and cancer:

1) **validation of the hypothesis generated by our study,** through an aetiological case-control study combined with the determination of biomarkers or other methods for determining individual exposure and including the collection of precise data on residential history and risk factors for each subject. Only this type of study would allow reliable confirmation that the relationships observed in our study persist after adjusting individual factors. It would also make it possible to obtain dose-response relationships and to develop predictive models. If positive, an analytical study could be used to confirm the excess risk of cancer associated with previous exposure. However, this would not provide information about the risk related to current emissions. The possible excess risk associated with current emissions could be evaluated only in 10 to 20 years, by carrying out another ecological study similar to this one;
2) **testing of longer latency periods to estimate more completely the strength of the exposure-risk relationships**, by extending the observation of these populations. Indeed, given our lack of knowledge on the real duration of the latency period for cancers, it is possible that the observation period of our study extends only to the start of the period in which excess cancers are likely to occur. The extension of this study should also contributed to evaluate more precisely the latency period of cancers;
3) **exploration of the relationship between cancers in women and exposure to incinerator emissions**, by completing the analysis of the study data, trying to find an explanation for the excess risk of "all cancers" in women. In particular, complementary studies of the incidence of uterine and ovarian cancers and particular aspects of breast cancer, such as age at diagnosis, comparing exposed and non-exposed women, would be informative.

7.2 Implementation of public health actions

First and foremost, we recommend to widely disseminate the results obtained in an accessible form to the general public. The implementation of preventive measures against cancers induced by incinerator emissions is no longer possible for people who were exposed during the period considered (1970s and 1980s) and until the application of new regulations limiting atmospheric emissions from MSWIs in 1997.

Provided expert advice is not contradictory in this field, given the low excess relative risks observed, and in the absence of a demonstration of causality, we do not recommend the establishment of particular secondary preventive measures (early screening, medical follow-up) for this group of population.

8. Conclusion

This ecological study demonstrates the existence of a link between the exposure of adult populations to the atmospheric emissions from MSWIs in activity between 1972 and 1990, and the incidence of cancers in the 1990s.

It has highlighted the statistically significant relationships between the exposure of populations to incinerator emissions and the risks of:
- breast cancer and "all cancers" in women;
- MNHL, for both sexes analysed together and for women;
- multiple myeloma in men.

These results also suggest, for both sexes, a possible link with liver cancer, soft-tissue sarcoma and multiple myeloma.

This study provides new evidence relating to the health risks of long-term environmental exposure to the emissions from MSWIs. Our findings are consistent with other studies in this field.

The large size of the population included in the analysis, the quality of the data supplied by the registries and the procedures used for the retrospective quantification of past exposure of the population contribute to the quality of this study.

The exploitation of the results obtained is subject to certain limitations, particularly as concerns their transposition to other times and places. This study dealt with a period of exposure in the past, and its results cannot be transposed to the current situation. Given the particular characteristics of ecological studies, the causality of the statistical link observed between exposure to incinerator emissions and the incidence of certain cancers cannot be demonstrated. Nonetheless, there are several lines of evidence to support the causality of this relationship. An aetiological study, with measurements of exposure and control for individual risk factors, could be carried out to evaluate the causality of the exposure-risk relationships observed.

This study, by demonstrating the health impact of MSWIs, confirms the usefulness of measures for reducing the emissions of pollutants imposed on these industrial installations, in France, at the end of the 1990s. We may therefore expect to see a decrease in the risk of cancer in populations exposed to current emission levels. However, given the uncertainty concerning the duration of the latency period to cancer onset, we cannot rule out the possibility that past exposure, from the 1970s onwards, may continue to favour the occurrence of cancers today.

Bibliographic References

[1] Ademe, Berthier F, Vanlaer H. Dioxines & Polluants Organiques Persistants. Journées Techniques Nationales, Chapitre : L'action des pouvoirs publics français. 2004 Mar.

[2] Comité de la Prévention et de la Précaution. *Les Incinérateurs d'ordures ménagères*: quels risques ? Quelles politiques ? Rapport du Ministère de l'Écologie et du Développement durable. Décembre 2004-page 10. 2007.

[3] Boudet C, Zmirou D, Laffond M, Balducci F, oit-Guyod JL. Health risk assessment of a modern municipal waste incinerator. Risk Anal 1999 Dec;19(6):1215-22.

[4] Gonzalez CA, Kogevinas M, Gadea E, Huici A, Bosch A, Bleda MJ, *et al.* Biomonitoring study of people living near or working at a municipal solid-waste incinerator before and after two years of operation. Arch Environ Health 2000 Jul;55(4):259-67.

[5] Franchini M, Rial M, Buiatti E, Bianchi F. Health effects of exposure to waste incinerator emissions: a review of epidemiological studies. Ann Ist Super Sanita 2004;40(1):101-15.

[6] Calle EE, Frumkin H, Henley SJ, Savitz DA, Thun MJ. Organochlorines and breast cancer risk. CA Cancer J Clin 2002 Sep;52(5):301-9.

[7] Hunter DJ, Hankinson SE, Laden F, Colditz GA, Manson JE, Willett WC, *et al.* Plasma organochlorine levels and the risk of breast cancer. N Engl J Med 1997 Oct 30;337(18):1253-8.

[8] Hond ED, Roels HA, Hoppenbrouwers K. Sexual maturation in relation to polychlorinated aromatic Hydrocarbons: Sharpe and Skakkebaek's hypothesis revisisted. Environ Health Perspect 2006;110(8):771-6.

[9] IARC. PAHs. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans 2006;92.

[10] IARC. PCDD PCDF. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans 1997;69.

[11] Steenland K, Bertazzi P, Baccarelli A, Kogevinas M. Dioxin revisited: developments since the 1997 IARC classification of dioxin as a human carcinogen. Environ Health Perspect 2004 Sep;112(13):1265-8.

[12] Elliott P, Arnold R, Cockings S, Eaton N, Jarup L, Jones J, *et al.* Risk of mortality, cancer incidence, and stroke in a population potentially exposed to cadmium. Occup Environ Med 2000 Feb;57(2):94-7.

[13] Dockery DW, Stone PH. Cardiovascular risks from fine particulate air pollution. N Engl J Med 2007;356(5):511-3.

[14] Sunyer J. Urban air pollution and chronic obstructive pulmonary disease: a review. Eur Respir J 2001 May;17(5):1024-33.

[15] Moulin JJ, Clavel T, Roy D, Dananche B, Marquis N, Fevotte J, *et al.* Risk of lung cancer in workers producing stainless steel and metallic alloys. Int Arch Occup Environ Health 2000 Apr;73(3):171-80.

[16] Zeegers MP, Swaen GM, Kant I, Goldbohm RA, van den Brandt PA. Occupational risk factors for male bladder cancer: results from a population based case cohort study in the Netherlands. Occup Environ Med 2001 Sep;58(9):590-6.

[17] Miligi L, Costantini AS, Benvenuti A, Kriebel D, Bolejack V, Tumino R, *et al.* Occupational exposure to solvents and the risk of lymphomas. Epidemiology 2006 Sep;17(5):552-61.

[18] European Communities. Council Directives on the prevention of air pollution from new municipal waste incineration plants, 89/369/EEC. Official Journal of the European Communities 1989;L163:32-6.

[19] European Communities. Council Directives on the prevention of air pollution from existing municipal waste incineration plants, 89/429/EEC. Official Journal of the European Communities 1989;L203:50-4.

[20] Sedman RM, Esparza JR. Evaluation of the public health risks associated with semivolatile metal and dioxin emissions from hazardous waste incinerators. Environ Health Perspect 1991 Aug;94:181-7.

[21] Maitre A, Collot-Fertey D, Anzivino L, Marques M, Hours M, Stoklov M. Municipal waste incinerators: air and biological monitoring of workers for exposure to particles, metals, and organic compounds. Occup Environ Med 2003 Aug;60(8):563-9.

[22] Bard D, Cabannes de Aymorce A, Cicolella A, Dab W, Desachy C, Dor F, *et al.* L'incinération des déchets et la santé publique : bilan des connaissances récentes et évaluation du risque *Société française de santé publique.* Collection Santé et Société 1999 Nov;7.

[23] Nawrot T, Plusquin M, Hogervorst J, Roels HA, Celis H, Thijs L, *et al.* Environmental exposure to cadmium and risk of cancer: a prospective population-based study. Lancet Oncol 2006 Feb;7(2):119-26.

[24] Knox EG, Gilman EA. Hazard proximities of childhood cancers in Great Britain from 1953-80. J Epidemiol Community Health 1997 Apr;51(2):151-9.

[25] Yoo JI, Kim KH, Jang HN, Seo YC, Seok KS, Hong JH, *et al.* The development of PM emission factor for small incinerators and boilers. Environ Technol 2002 Dec;23(12):1425-33.

[26] Balasubramanian R, Qian WB. Characterization and source identification of airborne trace metals in Singapore. J Environ Monit 2004 Oct;6(10):813-8.

[27] IARC. Chromium, nickel and welding. Lyon. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans 1990;49:49-445.

[28] IARC. Beryllium, cadmium, mercury, and exposures in the glass manufacturing industry. Lyon. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans 1993;58.

[29] Vrijheid M. Health effects of residence near hazardous waste landfill sites: a review of epidemiologic literature. Environ Health Perspect 2000 Mar;108 Suppl 1:101-12.

[30] Steenland K, Piacitelli L, Deddens J, Fingerhut M, Chang LI. Cancer, heart disease, and diabetes in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin. J Natl Cancer Inst 1999 May 5;91(9):779-86.

[31] Fingerhut MA, Halperin WE, Marlow DA, Piacitelli LA, Honchar PA, Sweeney MH, *et al.* Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin. N Engl J Med 1991 Jan 24;324(4):212-8.

[32] Boffetta P, Jourenkova N, Gustavsson P. Cancer risk from occupational and environmental exposure to polycyclic aromatic hydrocarbons. Cancer Causes Control 1997 May;8(3):444-72.

[33] Davis BJ, Mccurdy EA, Miller BD, Lucier GW, Tritscher AM. Ovarian tumors in rats induced by chronic 2,3,7,8-tetrachlorodibenzo-p-dioxin treatment. Cancer Res 2000 Oct 1;60(19):5414-9.

[34] Becher H, Flesch-Janys D, Kauppinen T, Kogevinas M, Steindorf K, Manz A, *et al.* Cancer mortality in German male workers exposed to phenoxy herbicides and dioxins. Cancer Causes Control 1996 May;7(3):312-21.

[35] Kogevinas M, Becher H, Benn T, Bertazzi PA, Boffetta P, Bueno-de-Mesquita HB, *et al.* Cancer mortality in workers exposed to phenoxy herbicides, chlorophenols, and dioxins. An expanded and updated international cohort study. Am J Epidemiol 1997 Jun 15;145(12):1061-75.

[36] Hooiveld M, Heederik DJ, Kogevinas M, Boffetta P, Needham LL, Patterson DG, Jr., *et al.* Second follow-up of a Dutch cohort occupationally exposed to phenoxy herbicides, chlorophenols, and contaminants. Am J Epidemiol 1998 May 1;147(9):891-901.

[37] Merlo F, Puntoni R. Soft-tissue sarcomas, malignant lymphomas, and 2,3,7,8-TCDD exposure in Seveso. Lancet 1986 Dec 20;2[8521-22]:1455.

[38] Bertazzi PA, Zocchetti C, Guercilena S, Consonni D, Tironi A, Landi MT, *et al.* Dioxin exposure and cancer risk: a 15-year mortality study after the "Seveso accident". Epidemiology 1997 Nov;8(6):646-52.

[39] Bertazzi PA, Bernucci I, Brambilla G, Consonni D, Pesatori AC. The Seveso studies on early and long-term effects of dioxin exposure: a review. Environ Health Perspect 1998 Apr;106 Suppl 2:625-33.

[40] Bertazzi PA, Consonni D, Bachetti S, Rubagotti M, Baccarelli A, Zocchetti C, *et al.* Health effects of dioxin exposure: a 20-year mortality study. Am J Epidemiol 2001;153(11):1031-44.

[41] Mocarelli P. Dioxin exposure and human health 30 years after the Seveso, Italy, accident. Isee-Isea International Conference on Environmental Epidemiology & Exposure Paris 2006 2006;361-2.

[42] Michelozzi P, Fusco D, Forastiere F, Ancona C, Dell'Orco V, Perucci CA. Small area study of mortality among people living near multiple sources of air pollution. Occup Environ Med 1998 Sep;55(9):611-5.

[43] Elliott P, Shaddick G, Kleinschmidt I, Jolley D, Walls P, Beresford J, *et al.* Cancer incidence near municipal solid waste incinerators in Great Britain. Br J Cancer 1996 Mar;73(5):702-10.

[44] Elliott P, Eaton N, Shaddick G, Carter R. Cancer incidence near municipal solid waste incinerators in Great Britain. Part 2: histopathological and case-note review of primary liver cancer cases. Br J Cancer 2000 Mar;82(5):1103-6.

[45] Biggeri A, Barbone F, Lagazio C, Bovenzi M, Stanta G. Air pollution and lung cancer in Trieste, Italy: spatial analysis of risk as a function of distance from sources. Environ Health Perspect 1996 Jul;104(7):750-4.

[46] Viel JF, Arveux P, Baverel J, Cahn JY. Soft-tissue sarcoma and non-Hodgkin's lymphoma clusters around a municipal solid waste incinerator with high dioxin emission levels. Am J Epidemiol 2000;152(1):13-9.

[47] Floret N, Mauny F, Challier B, Arveux P, Cahn JY, Viel JF. Dioxin emissions from a solid waste incinerator and risk of non-Hodgkin lymphoma. Epidemiology 2003;14(4):392-8.

[48] Floret N, Mauny F, Challier B, Cahn JY, Tourneux F, Viel JF. [Dioxin emissions and soft-tissue sarcoma: results of a population-based case-control study]. Rev Épidemiol Santé Publique 2004 Jun;52(3):213-20.

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

[50] Comba P, Ascoli V, Belli S, Benedetti M, Gatti L, Ricci P, *et al.* Risk of soft tissue sarcomas and residence in the neighbourhood of an incinerator of industrial wastes. Occup Environ Med 2003 Sep;60(9):680-3.

[51] Cancer Epidemiology and Prevention-Oxford University Press. Cancer Epidemiology and Prevention. New York: Oxford University Press. 1996.

[52] Remontet L, Esteve J, Bouvier AM, Grosclaude P, Launoy G, Menegoz F, *et al.* Cancer incidence and mortality in France over the period 1978-2000. Rev Épidemiol Santé Publique 2003 Feb;51(1 Pt 1):3-30.

[53] Remontet L, Buemi A, Velten M, Jougla E, Esteve J. Évolution de l'incidence et de la mortalité par cancer en France de 1978 à 2000. Francim, Inserm, InVS, 2003.

[54] Cole P, Trichopoulos D, Pastides H, Starr T, Mandel JS. Dioxin and cancer: a critical review. Regul Toxicol Pharmacol 2003 Dec;38(3):378-88.

[55] Mandal PK. Dioxin: a review of its environmental effects and its aryl hydrocarbon receptor biology. J Comp Physiol [B] 2005 May;175(4):221-30.

[56] Mulero-Navarro S, Pozo-Guisado E, Perez-Mancera PA, varez-Barrientos A, Catalina-Fernandez I, Hernandez-Nieto E, *et al.* Immortalized mouse mammary fibroblasts lacking dioxin receptor have impaired tumorigenicity in a subcutaneous mouse xenograft model. J Biol Chem 2005 Aug 5;280(31):28731-41.

[57] Schwartz GG. Multiple myeloma: clusters, clues, and dioxins. Cancer Epidemiol Biomarkers Prev 1997 Jan;6(1):49-56.

[58] Biggeri A, Catelan D. [Mortality for non-Hodgkin lymphoma and soft-tissue sarcoma in the surrounding area of an urban waste incinerator. Campi Bisenzio (Tuscany, Italy) 1981-2001]. Epidemiol Prev 2005 May;29[3-4]:156-9.

[59] De Roos AJ, Hartge P, Lubin JH, Colt JS, Davis S, Cerhan JR, *et al.* Persistent organochlorine chemicals in plasma and risk of non-Hodgkin's lymphoma. Cancer Res 2005 Dec 1;65(23):11214-26.

[60] Kogevinas M, Kauppinen T, Winkelmann R, Becher H, Bertazzi PA, Bueno-de-Mesquita HB, *et al.* Soft tissue sarcoma and non-Hodgkin's lymphoma in workers exposed to phenoxy herbicides, chlorophenols, and dioxins: two nested case-control studies. Epidemiology 1995 Jul;6(4):396-402.

[61] Saracci R, Kogevinas M, Bertazzi PA, Bueno de Mesquita BH, Coggon D, Green LM, *et al.* Cancer mortality in workers exposed to chlorophenoxy herbicides and chlorophenols. Lancet 1991 Oct 26;338(8774):1027-32.

[62] Elbi C, Misteli T, Hager GL. Recruitment of dioxin receptor to active transcription sites. Mol Biol Cell 2002 Jun;13(6):2001-15.

[63] Moennikes O, Loeppen S, Buchmann A, Andersson P, Ittrich C, Poellinger L, *et al.* A constitutively active dioxin/aryl hydrocarbon receptor promotes hepatocarcinogenesis in mice. Cancer Res 2004 Jul 15;64(14):4707-10.

[64] Dere E, Boverhof DR, Burgoon LD, Zacharewski TR. In vivo-in vitro toxicogenomic comparison of TCDD-elicited gene expression in Hepa1c1c7 mouse hepatoma cells and C57BL/6 hepatic tissue. BMC Genomics 2006 Apr 12;7.

[65] Becher H, Flesch-Janys D. Dioxins and furans: epidemiologic assessment of cancer risks and other human health effects. Environ Health Perspect 1998 Apr;106 Suppl 2:623-4.

[66] Verkasalo PK, Kokki E, Pukkala E, Vartiainen T, Kiviranta H, Penttinen A, *et al.* Cancer risk near a polluted river in Finland. Environ Health Perspect 2004 Jun;112(9):1026-31.

[67] Mastrangelo G, Fadda E, Marzia V. Polycyclic aromatic hydrocarbons and cancer in man. Environ Health Perspect 1996 Nov;104(11):1166-70.

[68] Prince MM, Hein MJ, Ruder AM, Waters MA, Laber PA, Whelan EA. Update: cohort mortality study of workers highly exposed to polychlorinated biphenyls (PCBs) during the manufacture of electrical capacitors, 1940-1998. Environ Health 2006 May 22;5:-13 pages.

[69] Baena AV, Allam MF, Del Castillo AS, az-Molina C, Requena Tapia MJ, bdel-Rahman AG, *et al.* Urinary bladder cancer risk factors in men: a Spanish case-control study. Eur J Cancer Prev 2006 Dec;15(6):498-503.

[70] Moulin JJ, Romazini S, Lasfargues G, Peltier A, Bozec C, Deguerry P, *et al.* [Development of a job-exposure matrix in the heavy-metal industry in France]. Rev Épidemiol Santé Publique 1997 Mar;45(1):41-51.

[71] Cordier S, Chevrier C, Robert-Gnansia E, Lorente C, Brula P, Hours M. Risk of congenital anomalies in the vicinity of municipal solid waste incinerators. Occup Environ Med 2004 Jan;61(1):8-15.

[72] Jylhä K. Empirical scavenging coefficients of radioactive substances released from Chernobyl. Atmospheric Environment 25A[2], 263-270. 1991. Ref Type: Journal (Full).

[73] Kaupp H, McLachlan MS. Distribution of polychlorinated dibenzo-P-dioxins and dibenzofurans (PCDD/Fs) and polycyclic aromatic hydrocarbons (PAHs) within the full size range of atmospheric particles. Atmospheric Environment 34[1],73-83.2000. Ref Type: Journal (Full).

[74] Nessel CS, Butler JP, Post GB, Held JL, Gochfeld M, Gallo MA. Evaluation of the relative contribution of exposure routes in a health risk assessment of dioxin emissions from a municipal waste incinerator. J Expo Anal Environ Epidemiol 1991 Jul;1(3):283-307.

[75] Ward E, Jemal A, Cokkinides V, Singh GK, Cardinez C, Ghafoor A, *et al.* Cancer disparities by race/ethnicity and socioeconomic status. CA Cancer J Clin 2004 Mar;54(2):78-93.

[76] Carstairs V. Deprivation indices: their interpretation and use in relation to health. J Epidemiol Community Health 1995 Dec;49 Suppl 2:S3-8.:S3-S8.

[77] SAHRU. A national deprivation index for health and health service research, Technical report. 1997.

[78] Townsend P. Deprivation. Journal of Social Policy 1987;16:125-46.

[79] Challier B, Viel JF. Pertinence et validité d'un nouvel indice composite français mesurant la pauvreté au niveau géographique. Rev Épidemiol Santé Publique 2001 Feb;49(1):41-50.

[80] Challier B, Baverel J, Arveux P, Mauny F, Pitard A, Viel JF. Validation of an area-based French deprivation index: a consistent link with lung cancer incidence. Questions en Santé publique : Chapter 4; 2001 Oct 17; 2001.

[81] Nasca PC, Burnett WS, Greenwald P, Brennan K, Wolfgang P, Carlton K. Population density as an indicator of urban-rural differences in cancer incidence, upstate New York, 1968-1972. Am J Epidemiol 1980 Sep;112(3):362-75.

[82] Howe HL, Keller JE, Lehnherr M. Relation between population density and cancer incidence, Illinois, 1986-1990. Am J Epidemiol 1993 Jul 1;138(1):29-36.

[83] Yang CY, Hsieh YL. The relationship between population density and cancer mortality in Taiwan. Jpn J Cancer Res 1998 Apr;89(4):355-60.

[84] Gauvin S, Zmirou D, Le MY, Cassadou S, Lauvergne N, Reungoat P, *et al.* [Air quality monitoring and personal exposure of children to NO(2) and fine particles]. Rev Épidemiol Santé Publique 2002 Jun;50(3):307-19.

[85] Pearson RL, Wachtel H, Ebi KL. Distance-weighted traffic density in proximity to a home is a risk factor for leukemia and other childhood cancers. J Air Waste Manag Assoc 2000 Feb;50(2):175-80.

[86] Feychting M, Svensson D, Ahlbom A. Exposure to motor vehicle exhaust and childhood cancer. Scand J Work Environ Health 1998 Feb;24(1):8-11.

[87] Lagache R, Sampic J., Rivat A. Cadastre des émissions des transports routiers en France Étude ADEME -OMS2-. CETE, editor. Réseau Scientifique et technique du ministère de l'Équipement. Référence documentation ISRN EQ-CT69-DV/RE--03.155_FR. 2005. Ref Type: Generic.

[88] Hastie T, Tibshiranin R. Generalized additive models. London, England: Chapman and Hall. 1990.

[89] Wood SN. Modelling and smoothing parameter estimation with multiple quadratic penalties. Journal of the Royal Statistical Society 2000;B(62):413-28.

[90] Wood SN, Augustin NH. GAMs with integrated model selection using penalized regression splines and applications to environmental modelling, Ecological Modelling. 2002.

[91] Akaike H. Information theory and an extension of the maximum likelihood principle, International Symposium on Information Theory. Budapest, Hungary: Akademiai Kiado. 1973.

[92] Best NG. Best NG: **Bayesian ecological modelling**. In *Disease mapping and risk assessment for public health*. Edited by Lawson A, Biggeri A, Bohning D, Lesaffre E, Viel JF, Bertollini R, Chichester: John Wiley & Sons 1999:193-201. In: Lawson A BABLEVJBRC, editor. *Disease mapping and risk assessment for public health*. 1999 ed. 1999. p. 193-201.

[93] Richardson S, Monfort C, Green M, Draper G, Muirhead C. Spatial variation of natural radiation and childhood leukaemia incidence in Great Britain. Stat Med 1995 Nov 15;14[21-22]:2487-501.

[94] Richardson S, Thomson A, Best N, Elliott P. Interpreting posterior relative risk estimates in disease-mapping studies. Environ Health Perspect 2004 Jun;112(9):1016-25.

[95] Besag J, York J, Mollié A. Bayesian image restoration, with two applications in spatial statistics, Annals of the Institute of Statistical Mathematics. 1991.

[96] Spiegelhalter DJ, Best NG, Carlin BP, Van der Linde A. Bayesian measures of model complexity and fit (with discussion). Journal of the Royal Statistical Society 2002;Series B(64):583-640.

[97] Best N, Richardson S, Thomson A. A comparison of Bayesian spatial models for disease mapping. Stat Methods Med Res 2005 Feb;14(1):35-59.

[98] Kelsall JE, Wakefield JC. Discussion of "Bayesian models for spatially correlated disease and exposure data" by Best *et al.* In Edited by Bernardo JM, Berger JO, Dawid AP, Smith AFM, Oxford: Oxford University Press 1999:151. In: BernardoJM BJDASA, editor. Bayesian Statistics 6. 1999 ed. Oxford University Press; 1999.

[99] Spiegelhalter DJ, Thoma A, Best N. WinBUGS, Version 1.4 User Manual. Cambridge, MA: MRC Biostatistics Unit. 2003.

[100] Wood SN. The mgcv package: GAMs with GCV smoothness estimation and GAMMs by REML/PQL. Cran r-project org 2007Available from: URL: cran.r-project.org/web/packages/mgcv/

[101] Tyczynski JE, Démaret E, Parkin DM. *"Standards and guidelines for cancer registration in Europe"* IARC technical publication N°40. IARC technical publication 2003;40.

[102] Nyberg F, Gustavsson P, Jarup L, Bellander T, Berglind N, Jakobsson R, *et al.* Urban air pollution and lung cancer in Stockholm. Epidemiology 2000 Sep;11(5):487-95.

[103] Read D, Wright C, Weinstein P, Borman B. Cancer incidence and mortality in a New Zealand community potentially exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin from 2,4,5-trichlorophenoxyacetic acid manufacture. Aust N Z J Public Health 2007 Feb;31(1):13-8.

[104] Zeger SL, Thomas D, Dominici F, Samet JM, Schwartz J, Dockery D, *et al.* Exposure measurement error in time-series studies of air pollution: concepts and consequences. Environ Health Perspect 2000 May;108(5):419-26.

[105] Correlation parameters of NO_2 to other traffic Pollutants near an Expressway Poster presentation, ISEE/ISEA International Conference Tucson 2006.

[106] Belpomme D, Irigaray P, Hardell L, Clapp R, Montagnier L, Epstein S, *et al.* The multitude and diversity of environmental carcinogens. Environ Res 2007 Aug 8.

[107] Schiestl RH, Aubrecht J, Yap WY, Kandikonda S, Sidhom S. Polychlorinated biphenyls and 2,3,7,8-tetrachlorodibenzo-p-dioxin induce intrachromosomal recombination in vitro and in vivo. Cancer Res 1997 Oct 1;57(19):4378-83.

[108] Wang SL, Chang YC, Chao HR, Li CM, Li LA, Lin LY, *et al.* Body burdens of polychlorinated dibenzo-p-dioxins, dibenzofurans, and biphenyls and their relations to estrogen metabolism in pregnant women. Environ Health Perspect 2006 May;114(5):740-5.

[109] Revich B, Aksel E, Ushakova T, Ivanova I, Zhuchenko N, Klyuev N, *et al.* Dioxin exposure and public health in Chapaevsk, Russia. Chemosphere 2001 May;43[4-7]:951-66.

[110] Manz A, Berger J, Dwyer JH, Flesch-Janys D, Nagel S, Waltsgott H. Cancer mortality among workers in chemical plant contaminated with dioxin. Lancet 1991 Oct;19;338(8773):959-64.

[111] Flesch-Janys D, Berger J, Gurn P, Manz A, Nagel S, Waltsgott H, *et al.* Exposure to polychlorinated dioxins and furans (PCDD/F) and mortality in a cohort of workers from a herbicide-producing plant in Hamburg, Federal Republic of Germany. Am J Epidemiol 1995 Dec 1;142(11):1165-75.

[112] Wolff MS, Weston A. Breast cancer risk and environmental exposures. Environ Health Perspect 1997 Jun;105 Suppl 4:891-6.

[113] Saintot M, Malaveille C, Hautefeuille A, Gerber M. Interaction between genetic polymorphism of cytochrome P450-1B1 and environmental pollutants in breast cancer risk. Eur J Cancer Prev 2004 Feb;13(1):83-6.

[114] Hagmar L, Linden K, Nilsson A, Norrving B, Akesson B, Schutz A, *et al.* Cancer incidence and mortality among Swedish Baltic Sea fishermen. Scand J Work Environ Health 1992 Aug;18(4):217-24.

[115] Svensson BG, Mikoczy Z, Stromberg U, Hagmar L. Mortality and cancer incidence among Swedish fishermen with a high dietary intake of persistent organochlorine compounds. Scand J Work Environ Health 1995 Apr;21(2):106-15.

Study of the incidence of cancers close to municipal solid waste incinerators

Summary

This ecological spatial study was performed in the context of the French Cancer Plan 2003-2007. It aimed to assess the relationship between the incidence of cancers in adults and exposure to emissions from municipal solid waste incinerators. It was based on cancers diagnosed in the Haut-Rhin, Bas-Rhin, Isère and Tarn districts between 1990 and 1999. Around 135,000 cancer cases were reported over almost 25 million person-years. The exposure of each statistical unit to 16 incinerators during the 1970s and 1980s was quantified by modelling atmospheric dispersion and cumulative surface dioxin deposition. Results are expressed as relative risks, comparing the risks of cancer occurrence in areas with high and low levels of exposure.

A statistically significant relationship was found between exposure to incinerator emissions and the incidence, in women only, of all types of cancer considered together, breast cancer and non-Hodgkin's lymphoma. A significant relationship was also found for malignant non-Hodgkin's lymphoma in both men and women, and for multiple myeloma in men only.

Although this study does not establish the causality of the observed relationships, it provides additional epidemiological evidence for a health impact of incinerator emissions. However, these findings concern a past period and cannot be applied to current emissions. They do, however, justify the implementation of regulatory measures for reducing the emissions of such industrial plants introduced in France at the end of the 1990s.

Key words: epidemiology, ecological study, incidence of cancers, municipal solid waste incinerator, environmental exposure, spatial analysis, modelling of atmospheric dispersion

Suggested citation:

Fabre P, Daniau C, Goria S, De Crouy-Chanel P, Paez A, Empereur-Bissonnet P. Study of the incidence of cancers close to municipal solid waste incinerators – Summary. Saint-Maurice (Fra): French Institute for Public Health Surveillance, July 2008, 29 p. Available from: [redacted]

FRENCH INSTITUTE FOR PUBLIC HEALTH SURVEILLANCE
12 rue du Val d'Osne
94 415 Saint-Maurice Cedex France
Tél. : 33 (0)1 41 79 67 00
Fax : 33 (0)1 41 79 67 67
[redacted]

ISSN : 1958-9719
ISBN : 978-2-11-098285-8
ISBN-NET : 978-2-11-098329-9
Printed in 50 copies
Printed by France Repro – Maisons-Alfort
Produced by DIADEIS-Paris
Legal deposition: July 2009