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20th January 2002

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A Chara

Re: A Proposal to Study the Effects on Public Health and the Environment of Landfill and Thermal Treatment of Waste in Ireland

I'd be most grateful if you could include this submission for review by the Team assessing the above issue. I'm a mother of 2 children under the age of three, I'm a concerned parent, citizen and consumer. The reasons for my concerns are mani-fold, but relate primarily to the threat of incineration on the food we eat, the air we breathe, the water we drink, our health, animal health and the legacy we leave to future generations. These fears may be described as 'perceived fears' by many parties, but I feel confident that if even a small amount of the negative reports I have read regarding incineration have any basis in truth, then the fears are very well founded.

I note the aims of your study are to review national and international literature on the various waste management practices available and to do a comparison between the ill effects of incineration/thermal treatment and landfill. I am a firm believer in the Zero Waste policy which is being adopted by many communities worldwide. This has been scoffed at and ridiculed by many, but is already making huge in-roads in Canberra, Australia, New Zealand, parts of Canada and other communities. The Renmore community in Galway have successfully diverted over 56% of their household waste from landfill by adopting practices advocated by the Zero Waste proponents. I hope therefore, that an assessment of technologies which embrace minimisation, reduction, re-use, recycling, composting, common sense and community involvement are also being assessed, along with the compare and contrast between thermal treatment and landfill. I do realise that there will be some residual waste from the Zero Waste programme, until we have a system where we have facilities for separated waste streams, and industries which do a cradle to grave assessment of the products they put out on the market, ensuring that all components can be re-used, recycled, biodegrade or can be disposed of safely in dry landfills. For reference, please see enclosed document on a Citizen's Agenda for Zero Waste (1), this is a strategy that avoids incinerators and eventually eliminates landfills. It is specific to an US/Canadian perspective, but the strategy could easily be extrapolated to Ireland.

Even though there is incineration in Europe, doesn't necessarily mean that it is right. They also have nuclear power, which we thankfully don't have, with all it's associated complications, though there was serious pressure put on Ireland to have a nuclear powerstation, people power won out in the end, we hope that the same will apply in respect of incineration.

Ludwig Kraemer, Head of EU Waste Management in June 26th 2000 stated 'an incinerator needs to be fed for up about 20 to 30 years and in order to be economic needs an enormous input from quite a region, so for 20 to 30 years you stifle innovation, you stifle alternatives, just in order to feed that monster which you build' (BBC 1 Panorama Documentary 'Rubbish'). Mr Kraemer on May 19th 2000 stated in the Guardian that 'In France, Belgium, Holland, Italy, Germany and Portugal, no more new incinerators are being built because the public will not stand for them.

They are treated in the same way as nuclear power stations – people no longer want them'. This is a very strong indication of the way public opinion is changing on the issue of incineration. The health, environmental, economic and sustainability lessons on incineration are being learnt, and communities are now trying to draw a line under this practice. Mr Kraemer stated on 7.6.1999 at the European Conference on Waste Management Planning that 'I would like to set the record straight: the Commission does not promote incineration. We do not consider this technique is favourable to the environment or that it is necessary to ensure a stable supply of waste for combustion over the long term. Such a strategy would only slow innovation. We should be promoting prevention and recycling above all. Those countries which are in the process of drafting their planning should not base it upon incineration.'

Please find attached a copy of a letter from Margot Wallstrom (2), EU Environment Commissioner, to Mr Crowley, TD, in which she states 'my opinion is that in most cases incinerators are not the answer to waste management. The incineration of waste helps reduce the volume of waste to be disposed of. However, the environmental impact of incineration is significant, given that, by incinerating waste, pollutants are only transformed – for example, they are concentrated in the incineration ashes, which must be disposed of.'

In the 2001 report on Sustainable Waste Management by the House of Commons Select Committee on the Environment, Transport and the Regions, it states that 'incineration will never play a major role in truly sustainable waste management, the health effects which result from an incinerator's emissions, are not yet fully known.' (Greenpeace Press Release 21.03.2001 attached) (3).

I am averse to incineration on many levels – from a community point of view, I look at the issue of proximity – large incinerators which take in the waste for up to 4 counties, with quite disparate communities, besides the economics and logistics of transport, the environmental effects of having diesel burning trucks driving round trips of 140 miles or more to dispose of waste is non-sensical. This is in contravention to the Proximity Principle. Each community should look after their own waste. The little and often principle. This issue could also be raised under the 'equity' argument, i.e. is it moral for one community to have to bear the brunt of the waste for 4 counties. Is it moral that we should allow incinerators in our midst to do untold damage to not only what is here and present now, but also to the next generation, as an incinerator has a life-span of 20 – 30 years. The emissions it creates may be fat soluble, bioaccumulate in body tissues, accumulate in the soil, water, their effects may be felt immediately, or over generations. This is in contravention to the Precautionary Principle. The polluters would be the incinerator companies, but the polluter payers would be the communities where these companies operate – this is in contravention to the Polluter Pays Principle.

Indaver Ireland, the company who have lodged a planning application for a 150,000 tonne municipal waste incinerator at Carranstown, Duleek, Co Meath list in their EIS the below as being some of the pollutants emitted 24 hours a day 365 days a year. All of these have negative effect either through direct contact, or through their manifestations, eg. greenhouse gases, acid gases, etc.

Oxides of nitrogen (NOx)
Carbon Monoxide
Hydrocarbons
Hydrogen Fluoride (HF)
Poly-chloro dibenzo furans (PCDF)
Thallium (Tl)
Antimony (Sb)
Lead (Pb)
Cobalt (Co)
Manganese (Mn)
Vanadium (V)

Sulphur Dioxide ($S0_2$)
Particulates (Dust)
Hydrogen Chloride (HCl)
Poly-chloro dibenzo dioxins (PCDD)
Cadmium (Cd)
Mercury (Hg)
Arsenic (As)
Chromium (Cr)
Copper (Cu)
Nickel (Ni)

Below some examples of damage to plants exposed to varying exposures to some of these pollutants

Plant type	Symptom	Possible causes
Conifers	Tip burn	NH_3; HCl; CL_2; N0x; $S0_2$; F; O_3;
	Red/Yellow/Brown discolouration	NH_3; HCl; N0x; $S0_2$; F
	Dull Grey / Green discolouration	CL_2; N0x; F
	Black discolouration	NH_3
	Chlorosis of young needles	Cl_2; N0x; $S0_2$; F; PAN; K
Broad leaved trees & shrubs	Chlorotic mottle	HCl
	Necrotic stipple	HCl; CL_2; $S0_2$, F
	Necrotic margins	NH_3; HCl; Cl_2; N0x; F
	Interveinal necrosis	NH_3; Cl_2; N0x; $S0_2$; F;
	Interveinal bleaching	HCl; N0x;
	Red discolouration	$S0_2$; F
	Bronzing / glazing	NH_3; Cl_2
	Leaf distortion	$S0_2$; F
	Yellow discolouration	NH_3
	Black discolouration	NH_3, Br_2
	Dieback	NH_3
	Premature necrosis of fruit	F
Narrow leaved plants	Chlorotic mottle	F
	Necrotic stipple	NH_3; F; O_3
	Tip necrosis	NH_3; HCl; Cl_2; $S0_2$; F; Br_2; Ca; Mg
	Interveinal necrotic streaks	NH_3; HCl; Cl_2; N0x; $S0_2$; F; O_3
	Bleached tips	NH_3; $C1_2$; $S0_2$; F
	Red discolouration	NH_3; $S0_2$
	Bronzing / glazing	NH_3; Cl_2; NOx; F
	Necrotic bracts	F
	Necrotic sepals	$S0_2$; F
	Necrotic awns	N0x; $S0_2$, F
Broad leaved plants	Marginal chlorosis	CL_2; F
	Necrotic stipple	HCl; $Cl_2$2; N0x; 0_3
	Tip necrosis	N0x; $S0_2$
	Marginal necrosis	HCl; Cl_2; N0x; $S0_2$; F; H_2S; B
	Interveinal necrosis	NH_3; Cl_2; N0x; $S0_2$; 0_3; PAN; B- H_2S; Hg; Br_2; F
	Bleaching	HCl; Cl_2; $S0_2$
	Red discolouration	Cl_2; $S0_2$; F; Ethylene; NH_3
	Yellow discolouration	NH_3
	Black discolouration	NH_3; HCl; Cl_2
	Dark stipple	NH_3; CL_2; N0x; $S0_2$; 0_3
	Bronzing / glazing	HCl; Cl_2; N0x; $S0_2$; PAN; NH_3
	Leaf distortion	HCl; Cl_2; $S0_2$; F; Ethylene; B; H_2S
	Shot holing	HCl; N0x; $S0_2$
	Necrotic sepals	HCl; N0x; $S0_2$; F; Ethylene
	Necrotic bracts	HCl; N0x; H_2S
	Necrotic petals	NH_3; HCl; F; Hg

Many of the known emissions from incinerators are environmentally detrimental – the table below outlines some of the effects on animal and plant life. What is also very worrying is that there are many compounds, which have as yet not been identified, or studied to any great extent. These are forming as man made products are burnt at very high temperatures, causing chemical and compositional reactions within mixtures of compounds, creating who-know's-what. The toxicity of dioxin has only come to light in the recent past, this is a by-product of the combustion of chlorine containing materials in the presence of carbon. As we don't know what else lurks in the incinerator grate waiting to be released I urge the precautionary principle.

Pollutant	**Effects**	
Carbon monoxide (CO)	Affects humans. Combines with haemoglobin in blood, forming carboxyhaemoglobin, which does not dissociate. Deprives brain of $0_{2,}$ Causes faintness, slow reactions and ultimately death.	From incineration
Carbon particles	Blackens buildings, contributes to lung disease.	From incineration
Carbon dioxide (CO_2)	Greenhouse gas, global warming,	From incineration
Methane (CH_4)	Greenhouse gas, global warming	From the organic matter in the waste bunkers
Sulphur dioxide (SO_2)	Forms acid rain, kills lichen,etches buildings, exacerbates asthma, bronchitis and other respiratory conditions	From incineration
Oxides of nitrogen (N_2O, NO, NO_2)	Poisonous. May deplete ozone layer. Contribute to 'acid rain'.	From incineration
Peroxyacyl nitrates (PAN)	Choking 'photochemical smog'	Incineration process
Flourine (F_2), hydrogen fluoride (HF)	Fluorosis (bone disease) in cattle. Reduces plant growth; damages leaves.	Incineration process
Chlorofluorocarbons (CFC's)	Deplete ozone layer, allowing damaging UF rays to reach organisms, contribute to 'greenhouse effects'.	From incineration of un-separated waste
Lead (Pb)	Cumulative poison in brain, enters bloodstream through lungs (particulate lead) or gut. Mental retardation and death at high concentrations in humans. Lead from spoil heaps kills wildlife in streams and rivers.	Incineration process
Mercury (Hg)	Accumulates up food chains. Top predators particularly at risk. Cumulative brain poison, causing nervous discoordination, mental retardation and death. Prevents nitrification in some seas.	Incineration process
Cadmium (Cd)	Causes disappearance of cartilaginous part of bone. Aching joints, brittle bones. Kidney damage. Accumulated in shellfish in estuaries.	Incineration process
Dioxins	Very poisonous, carcinogen, developmental abnormalities in children.	Incineration process

With regard to human health, I enclose an executive summary from the Greenpeace Report 'Incineration and Human Health' (Allsopp M, Costner P & Johnston P, 2001) (**4**). I'm sure you've come across this full report in your research, but I'd just like to highlight the fact that even though it assesses studies, big and small from incinerators old and new, there is a large body of evidence which concludes that incinerators release toxic substances and that humans are exposed as a consequence. Studies on workers at incinerator plants, and populations residing near to incinerators have identified a wide range of associated health impacts. These studies give rise to great concerns about possible health impacts from incinerators even though the

number of studies (particularly those that have been conducted to appropriately rigorous scientific standards) is highly limited. These should be seen, however as strongly indicative that incinerators are potentially very damaging to human health. Lois Marie Gibbs & CHEJ's book 'Dying from Dioxin – A Citizen's Guide to Reclaiming Our Health and Rebuilding Democracy' purports that exposure to dioxin, even at doses 100 times lower than those associated with cancer, can cause infertility, hormonal imbalance, and immune system dysfunction.

Further along this theme I enclose just a few re-prints/extracts which raise some very worrying questions and state some very clear evidence of health will be compromised by incinerator emissions. For example, with regard to assessing whether biomarkers in adolescents can show exposure to, and health effects of, common environmental pollutants. This study had 2 groups, rural and urban (close to a lead smelter and 2 incinerators). The findings were that internal exposure was mostly within current standards. Concentrations of lead and cadmium in blood, PCBs (polychlorinated biphenyls) and dioxin-like compounds in serum samples, and metabolites of VOCs (volatile organic compounds; in urine were higher in one or both suburbs than in the rural control area. Children who lived near the waste incinerators matured sexually at an older age than others, and testicular volume was smaller in boys from the suburbs than in controls.' (Lancet, Vol 357, No. 9269 pg 1660-1670) (5).

Harking back to dioxin again, in a 20 year mortality study related to the health effects of dioxin exposure (Bertazzi *et al*, Am. J. Epid. vol 153, No. 11, 1.6.2001, pg 1031-44) (6) subsequent to the Seveso accident in 1976, an increase in many kinds of cancer and other illnesses are documented. 'Results support evaluation of dioxin as carcinogenic to humans and corroborate the hypotheses of its association with other health outcomes, including cardiovascular- and endocrine-related effects.'

Incineration does not dispose of the requirement for landfill. One-third by weight of all that goes into an incinerator comes out as ash, with varying levels of toxicity. This ash doesn't bio-degrade, therefore needs to be landfilled. It is a worry that the very fine particulates could become airborne and lodge in respiratory tracts or lungs, cover plant life, thus preventing the exchanges that occur in the leaves, the power-houses of plants. We also worry that if it's buried, how long before it comes out of solution in leachate and makes it's way into our water courses. Landfills are lined with a PVC liner, this is prone to rips, wear and tear, damage from sunlight and eventual old age. Elliott (BMJ 2001;323:363-368) (7) in his assessment of the 'Risk of Adverse Birth Outcomes in Populations Living near Landfill' concludes the fact that there was an excess risk of congenital anomalies and low birth weight near landfill sites in Great Britain, but acknowledges that further work is needed to assess this issue further. Again I go back to the Zero Waste concept, where we don't have mixed media in landfill.

Please find enclosed

- WHO Press Release (WHO/45, 3.6.1998) – WHO Experts Re-evaluate Health Risks from Dioxins (8)
- WHO factsheet (No. 225, June 1999) entitled 'Dioxins and their Effects on Human Health' (9)
- Incinerator cancer threat revealed – Guardian 18.05.2000 which states that dioxins from waste burning and industry far more dangerous than was thought' (10)
- Government figures show Edmonton incinerator responsible for fifteen deaths per year' – UK Dept of Health, where Environment Minister Michael Meacher has stated that there is 'no safe threshold' for incinerator emissions (Greenpeace Report – 10.12.2000) (11)
- Dioxin & Incinerators – letter by Julianne Byrne PhD – local epidemiologist in our town (12)
- Rachel's Hazardous Waste News No. 353 – 2.9.1993 – EPA: Dioxin does cause cancer in humans. (13)
- Rachel's Environment & Health Weekly, No. 270. 29.01.1992. EPA's Dioxin Reassessment – Part 2. Dioxin Damages Human Immune System. (14)
- Rachel's Environment & Health Weekly, No. 457. 31.08.1995. Dioxin Inquisition. (15)

- Rachel's Environment & Health Weekly. No. 508. 22.08.1996. How to Eliminate Dioxin (16)
- Pollution and Health Impacts of Waste Incineration – Greenpeace (17)

With regard to integrity of food produce, especially meat and dairy, please find attached a letter from the Agricultural Department of the Belgian Embassy (18) where proximity to incinerators is stated as a reason why fat/meat tissue of cattle may have an increased level of dioxin. Please also find attached a letter from the Co-Chariman of the Wexford Irish Farmers Association (19) where he states that 'farmers have responsibility for their produce and are governed by a number of laws surrounding food production; emissions from an incinerator, whether real or imaginary could cause food quality concerns with retailers or consumers.' From an economic point of view, Ireland is heavily dependent on its agriculture, this was evident during the foot and mouth threat earlier this year. To allow such a potential threat, whether real or perceived, into our communities could both be a threat to our own food source and also detrimental to our farming industry.

Please see attached a copy of a report from the Ministry for the Environment in New Zealand, entitled 'Valuing New Zealand's Clean Green Image'(20) . This is a very interesting paper which may go some way to quantitatively evaluating the intangible of perception.

The New Zealand Ministry for the Environment commissioned a study to assess this theory with the aim of quantifying the extent to which particular New Zealand exports benefit from positive perceptions about their environment. The project focused on three export sectors; dairy, inbound tourism and organic produce. The former 2 of these being of great importance to Ireland, with the latter growing in importance. The study assessed the potential consumer reaction to an illustrative decline in New Zealand's cleanness and greenness.

It concluded that the empirical work done in this study reinforces the qualitative evidence that a clean green image is valuable, and provides some useful insights into the size and nature of that value. The results are of course not definitive – no contingent valuation study can ever be so – but they do strongly indicate a significant vulnerability of export value (through reduction in product quantities likely to be purchased by consumers) in the event of a (hypothetical) degradation of New Zealand's environment. It concludes that New Zealand's clean green image does have a value. Environmental image is a substantial driver of the value New Zealand can derive for goods and services in the international market place. The study suggests this is worth at least hundreds of millions, possibly billions, of dollars – aggregating value elements from dairy, tourism and organic produce, and extrapolating to other sectors such as meat.

To highlight further my concerns regarding incineration and dioxin production, no matter how minimal – the POPS Treaty, ratified in 2001 by 122 countries which is a legally binding instrument to phase out a group of 12 persistent organic pollutants, lists dioxin as one of the dirty dozen. By endorsing a technology which is proven to produce dioxin is in contravention to the POPS treaty. Please find enclosed Greenpeace Toxics Press Release 10.12.2000 – Beginning of the end of toxic pollution: world's most dangerous chemicals to be banned. (21)

We have also signed up to the Kyoto protocol which is aimed at reducing greenhouse gases, incinerators create these by the cubic tonne, therefore to endorse this polluting technology would also be in contravention to the Kyoto protocol.

I attach a document from Greenpeace entitled 'Why Ban Incineration' (22), it lists the impacts of incineration, their emissions such as heavy metals, unburned toxic chemicals and new pollutants. It also outlines the issue of hazardous ash. Incinerators create 3 different forms of ash, two of which are known to be highly toxic then the bottom ash, which incinerator companies state is non-toxic, but which is still under debate. To date the EPA in Ireland haven't finished concluding trials to confirm guidelines for assessing whether this ash is toxic or non. It needs to be assessed on an ongoing basis to monitor the levels of heavy metals and other contaminants in it. This ash has been proven toxic in many countries, eg. in Byker in Newcastle, 2000, where it was used in a mix for covering footpaths in allotment paths and children's playgrounds by Local Council. When the substrate was investigated, it was found to have Mercury levels @ 2,406% above normal, cadmium at 785% above normal and lead at 136% above normal, along with an substantial dioxin load.

Greenpeace and Friends of the Earth globally have undertaken much research into the economics, safety, morality, sustainability and sense of thermal treatment of household waste. Please find attached documents covering these topics. (Friends of the Earth – Up in Smoke, Greenpeace – Five Popular Myths about Incineration) (23, 23a).

The EU endorsed waste hierarchy is supposed to underpin our country's waste management strategy. Please find enclosed an ENFO document on Public Action for Sustainability (24). This outlines the hierarchy as being; prevention, minimisation, re-use, recycling, energy recovery, disposal in the order of most favoured, down to least favoured option. To date, in our town we have no recycling centre, the town I live in has 26,000 people, we have 3 can and bottle banks run by charities which are emptied on a very ad hoc basis. The nearest recycling centre for batteries, paper, plastics, white goods, clothes, etc. is a round trip of over 40 miles. In the capacity of your study in assessing best practices in waste management this shows that our government's proposals for waste management jump straight to the least favoured options. This is wrong.

Also, with regard to energy recovery – incineration has been deemed as a non-renewable energy source by the EU (16th November 2000). It is one of the least efficient methods of energy generation. There are, however, methods of energy generation from composting which can generate large amounts of renewable energy, eg. the city of Freiburg in Germany has a municipal composter/digester which, through the harnessing of methane from the organics collected from a city the size of Cork, creates enough energy to heat 16,000 households, with a great organic by-product of compost. There are many other such sustainable examples which I don't feel have been investigated or considered.

The Waste Management Plan for the North East was compiled by MCO'Sullivan, Consultants. This plan covers the counties Louth, Meath, Cavan and Monaghan, the Region where I and my family live. This plan relies heavily on incineration as a method of waste disposal and has quite depressing *potential* recycling figures. Please find attached a critique of this document (25) by Anne-Marie Cunningham of The Waste Working Group (a coalition of environmental NGO's including VOICE of Irish Concern for the Environment and Earthwatch – Friends of the Earth Ireland.

Please also find attached some observations (26) on this Plan by Dr Maurice O'Reilly, a Mathematician from St Patrick's, Dublin. He contends that there are many anomalies and flaws in the figures contained within the document, which form the backbone of the hypotheses they propose.

Incineration in the context of sustainability and sustainable development. A paper by Anne-Marie Cunningham. (27)

Incineration as an outdated technology. A paper by Anne-Marie Cunningham. (28)

Economics of Incineration. A paper by Anne-Marie Cunningham. (29)

Much of this letter has been very negative, highlighting what could be wrong. I'd like to therefore finish on a positive note, promoting waste management practices which are environmentally friendly and have minimal effects on both health and environment. I have seen examples of waste management strategies for various areas in Ireland which have been compiled by community groups, but as I have not secured their authorisation to use their documents, I will leave them out of this section. Please be aware however, that many communities are so insensed by the threat of having incinerators foisted on them, that they came together, gave up their valuable time and energy to becoming informed on the issue and, without any funding, put together waste management strategies which adhere to the Zero Waste strategies, which are underpinned by strong leadership, community involvement at all levels and industrial responsibility.

Paper on Quinte, Ontario, Canada (30)– over a seven year period, household waste was reduced by an average of 68%. At the same time the total cost of the waste management and recycling program, including disposal fell by 39%.

Comparative Report of Three International Sustainable Waste Management Programmes, Including Feasibility in the Irish Situation (31). Anne-Marie Cunningham MSc, BSc, Waste Working Group

Alternatives to Incineration. (32) Greenpeace Ban the Burn Campaign – Toxics Homepage

Zero Waste Update (33) – this is just one example of a Newsletter from Zero Waste New Zealand Trust. Issue 17 – August 2001.

Don't burn it or bury it (34)– Alternatives to Landfill and Incineration – Friends of the Earth Briefing Sheet.

Thank you very much for the time and energy it will take to read this and the many attachments. Please know that I, my family, and many others in our community, locally and nationally, would welcome the chance to practice good waste management by adopting the 3 R's and composting. For our authorities to even entertain the idea of incineration is not only pre-mature, but highly irresponsible and morally wrong.

Please do not hesitate to contact me if there are any issues with this submission. I would also be very grateful if you could keep me abreast of developments with your Report, if possible, advising when it is complete and possibly forwarding a copy to the above address.

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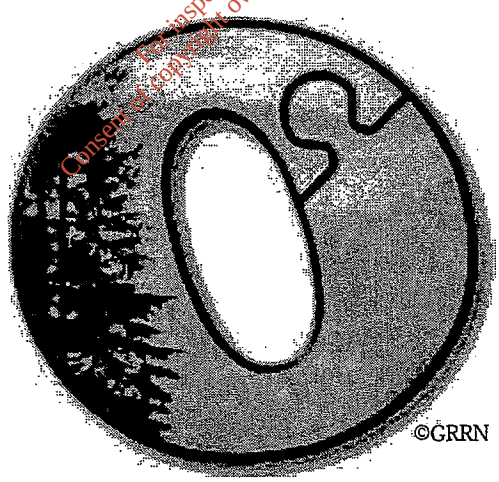
Aideen Doyle

Enclosures: 1 - 34

A Citizen's Agenda for Zero Waste

A UNITED STATES / CANADIAN PERSPECTIVE[1]

A strategy that avoids incinerators and eventually eliminates landfills



By Paul Connett and Bill Sheehan

1. INTRODUCTION

This essay is an updated and expanded version of one Paul Connett wrote in 1998, entitled Alternatives to Trash Incineration. That paper was based on Paul's 14-year experience of helping communities in over 40 countries fighting unwanted incinerators and landfills, and on his co-producing videotapes of alternative solutions mostly initiated by citizens. Several key events and developments have triggered this update.

First and foremost, Paul Connett met Bill Sheehan, director of the GrassRoots Recycling Network[2]. Bill is as avidly opposed to landfills as Paul is to incinerators. It was Bill who encouraged Paul to attend the meeting of the California Resource Recovery Association (one of the oldest and largest recycling organizations in the US) in June 1999. It was there that we - Paul and Bill - met with some of the key theorists and practitioners of zero waste and captured many of their ideas and activities in the videotape, *Zero Waste: Idealistic Dream or Realistic Goal?* (see Resources section at end).

Unfortunately, community groups with single-minded determination to stop an incinerator at all costs have frequently ended up supporting a landfill (often somewhere else!), and similarly, those single-mindedly resisting a landfill have often ended up with an incinerator (also somewhere else!). It was with the strategy of Zero Waste that Bill and Paul have found common ground. We believe it can offer common ground to community groups as well. Zero Waste offers a solution to trash that neither involves incineration nor a large reliance on landfill, and certainly not the huge mega-raw-waste landfills so popular with the solid waste industry. Zero Waste also allows citizens a positive agenda rather than simply opposing something. Hopefully, it will encourage citizen activists, such as those who have helped to stop the building of over 300 trash incinerators in the United States, and many others in other countries, to integrate their efforts in the larger goal of moving towards a sustainable economy.

A message that the Washington DC-based Institute for Local Self-Reliance has been delivering for over 25 years is that stopping incinerators makes recycling possible, and recycling makes economic development possible. As they argued in the 1989 report, *Salvaging the Future: Waste-Based Production*[3], the most important economic benefit occurs when the recovered materials are manufactured into finished products within the local economy.

In short, the movement for zero waste has grown out of decades of grassroots efforts to promote community-based recycling and defeat incinerators and landfills[4]. Zero Waste is a guiding principle that says that waste is not natural and can be eliminated with the proper design, policy and advocacy efforts.

The second key development is that as of 2001, 40% of the municipal authorities in New Zealand have adopted Zero Waste goals[5]. Most are shooting for Zero Waste by the year 2015 and some by 2020. They have thus shattered the notion that Zero Waste is a hopelessly 'idealistic' cause. Their adoption of a Zero Waste strategy confirms that it is a very practical approach for both local authorities and local activists.

A third important event occurred in 1999 with the publication of the book *Creating Wealth from Waste* by Dr. Robin Murray, an economist from the London School of Economics[6]. About a third of this book is devoted to the concept of Zero Waste. Murray's analysis underlines the sound economic basis for a Zero Waste approach.

A fourth event was Paul's participation in a press conference in Toronto in November 2000, at which Earth Day Canada launched the Target Zero Canada campaign[7]. At this conference Paul met several exciting people including Lucio Di Clemente, chief executive officer of the Beer Store in Ontario, which captures and reuses 97% of its glass beer bottles; Trish Johnson, who has masterminded the successful Take it Back to Retail program in Ottawa, which involves over 300 retailers; Rahumathulla Marikkar from Interface Canada, the multinational carpet manufacturer that is pledged to become a truly sustainable corporation; and Barry Friesen, solid waste-resource director for the Ministry of Environment and Labor in Nova Scotia (see Resources section), a province that under his leadership has achieved a 50% diversion of municipal solid waste in just five years. All of them are making significant strides on the Road to Zero Waste. Paul and his son Michael have since visited and videotaped each program.

The fifth key development was a trip organized by Arne Schoevers, director of the Dutch environmental group, Waste & Environment[8], to the European headquarters of the Xerox Corporation in Venray, Netherlands. Xerox is one of a number of leading corporations that have announced a commitment to Zero Waste. Using a massive 'reverse distribution' system, the Xerox Corporation is recovering its old copying machines from throughout Europe, repairing them, reusing parts, or recycling their constituent materials. Ninety-five percent of the returned material is either being reused or recycled. In the process they have saved $76 million in production and avoided waste disposal costs. Xerox candidly admits that they went into this program for economic rather than environmental reasons, which clearly underlines the fact that Zero Waste is a win-win solution for both the environment and the economy.

All five events for us have reinforced the fact that the move towards Zero Waste is not pie-in-the-sky. That does not mean, however, that it is going to happen without a tremendous effort from citizens, more vision in industry, and enlightened leadership from government officials.

To aid this effort, Grass Roots and Global Video[9], with the help of the GrassRoots Recycling Network and Waste & Environment, is producing a series of videotapes with the running title, *On the Road to Zero Waste.* We completed Part 1, *Nova Scotia, Community Responsibility in Action* in October 2001. This Guide is designed to accompany this series. In it we will look more closely at three key elements of a Zero Waste strategy: Community Responsibility, Industrial Responsibility and Political Leadership. But first we will look more closely at the Zero Waste vision.

2. ZERO WASTE VISION:

Ending the Age of Wasting

The grassroots recycling movement has been tremendously successful over the past 30 years in encouraging communities to handle their discarded materials responsibly. Recycling advocates realized that dealing with waste at the back end is not enough to stem the vast over-exploitation of virgin resources (including fossil fuels) that is the fundamental cause of global environmental degradation. Thus, while the Zero Waste vision recognizes the importance of recycling, it also recognizes its limitations. Communities cannot solve the trash problem alone and should not be

forced to clean up after irresponsible industries.

Zero Waste requires a mind shift. We have to change the task from getting rid of waste, to one of ensuring sustainable material practices at the front end of the manufacturing process. Communities faced with discarded materials and objects they cannot reuse, recycle or compost have to demand that industry stops producing them. Total recycling is not approachable without industry's help.

Thus, Zero Waste consciously links 'community responsibility' to 'industrial responsibility.'

Zero Waste combines community practices such as reuse, repair, recycling, toxic removal and composting, with industrial practices such as eliminating toxics and re-designing packaging and products for the key demands of the twenty first century: the need to develop sustainable communities and sustainable companies.

Zero Waste combines ethical practice with a solid economic vision, both for local communities and major corporations. On the one hand, it creates local jobs and businesses, which collect and process secondary materials into new products, and on the other, it offers major corporations a way of increasing their efficiency, thereby reducing their demands on virgin materials as well as their waste disposal costs.

Our current industrial system and throwaway society is based on the one-way flow of virgin resources to polluting dumps and incinerators. Extracting, processing, transporting and wasting resources is a primary cause of environmental destruction and global warming. We need to reconfigure our one-way industrial system into a circular, closed-loop system, recycling discarded resources from communities back to industries, both new and old.

Zero Waste recognizes the larger bookkeeping of nature. We never actually 'own' anything: we are simply borrowing its constituent materials for a short time. We are breaking this 'contract' when we simply throw things away. Nature makes no waste; waste is a human invention. Our task - both in the community and in industry - is to cycle these materials for future use. To do this, more than anything else, we need strong leadership at the community, industrial and political levels.

3. COMMUNITY RESPONSIBILITY

3.1 Zero Waste Policy and Legislation

Several communities have already introduced Zero Waste legislation or goals and they are listed at the end of this section. We have pulled out a number of policy steps that we believe are important for communities to take in order to a launch a Zero Waste program.

1) **Designate a target year.** When adopting a Zero Waste goal, it is important for communities to designate a year by which no waste will be delivered to the 'interim' landfill. Most communities have chosen a year some 15 or 20 years ahead. Doing this allows communities to approach an 'idealistic goal' in a realistic time frame. It allows the mind shift from managing waste to eliminating waste and managing resources time to develop.
2) **Design program with whole community.** During this first step and all subsequent ones it is critical, in our view, that the whole process be overseen and designed by a group of committed people drawn from the community, including people in local government, businesses and private citizens. Without this cooperative effort neither strong laws nor good intentions will

go very far.

3) **Ban key items from the landfill.** These should include ALL organic material (that is, compostables, or things that can be composted and safely returned to the Earth), any material that can be currently recycled, and any toxic material that can be dropped off at collection centers or retailers.
4) **Place a surcharge on material that is landfilled.** This is important for two reasons: a) to provide a disincentive for the generation of this fraction and b) to provide finance for other critical parts of the Zero Waste program.
5) **Provide incentives for recycling.** It is important to stimulate development of businesses, small or large, that can collect, process and reuse, repair or recycle materials in the community discard stream. Ideally, such businesses will provide jobs for the local community.
6) **Encourage waste audits.** It is critical to provide financial help or professional advice to businesses and institutions to embark on waste audits. Such audits identify where waste is being generated in both industrial processes and office operations, so that it can then be reduced or eliminated. The good news here is that almost invariably when such steps are taken they result in saving money.
7) **Stimulate take-back programs.** Provide incentives to local retailers and manufacturers to take back their products and packaging after use. Such incentives can range from deposits on such things as beverage and food containers; batteries and automobile tires, to the free publicity that surrounds a community sponsored 'Take It Back' program for hazardous materials like paint, fluorescent bulbs and electronic goods.
8) **Convert old landfill into industrial or ecopark.** Set in motion plans to convert the old landfill site into a completely different operation. As conceived and described by Dan Knapp and others, this site will look more like an industrial park. The local government can own and maintain the infrastructure but franchise out different parts of the site to local businesses involved with collecting, processing, recycling, reusing, repairing and remanufacturing source separated materials and objects in the community discard stream.

It is clear that many these policy changes impact community economics. Instead of paying waste companies to get rid of discards, we are suggesting that tax payers' money is better spent recovering resources. Thus the role of local government changes when discarded materials are treated as community enhancing assets rather than as liabilities (waste). Instead of managing liabilities, local government policies instead promote entrepreneurial innovation by maximizing delivery of clean resource streams to local enterprises.

As materials once considered waste gain value, Zero Waste principles will help local economies become more self-sufficient and create opportunities for increased civic participation and sustainable employment.

To the extent that communities and citizens can pressure industry to reduce the extraction and processing of virgin resources, they not only reduce the demands on local services but they also contribute to solving larger global problems.

Following are examples of communities that have passed Zero Waste legislation,

plans or resolutions:

- Canberra, Australia (population 300,000)[10]. Australia's capital adopted a No Waste by 2010 goal and plan in 1996. The plan envisions a waste-free city by 2010, with its two landfills replaced by 'Resource Recovery Estates.' Since 1995, recycling has increased 80%. This landfill design looks more like an industrial park than the typical landfill disposal site.
- Del Norte County, California, USA (population 32,000)[11]. Del Norte County is the first county in the United States to guide its solid waste strategy with a comprehensive Zero Waste plan, which it adopted in 2000. Officials expect the plan to ease the conversion from a timber-oriented economy to a new, sustainable economy using local resources currently being wasted.
- New Zealand Councils[12]. As of 2001, 40% of New Zealand's 74 local governments have adopted goals of Zero Waste to landfills by 2015, and an effort is underway to get the goal adopted nationally. Zero Waste New Zealand Trust provides a small amount of grant money to help councils get started but does not supply a blueprint -- that is being developed by local officials, managers and engineers. The trust predicts the creation of 40,000 jobs over 10 years through converting local transfer stations to resource recovery centers, and through the resulting proliferation of reuse and recycling businesses.
- Seattle, Washington, USA (population 534,700)[13]. Seattle adopted Zero Waste as a 'guiding principle' in 1998. The plan emphasizes managing resources instead of waste, and conserving natural resources through waste prevention and recycling.
- Santa Cruz County CA, USA (population 230,000) adopted Zero Waste as a long-term goal in 1999.

3.2 Practical Steps

The importance of passing legislation in support of a Zero Waste plan is that it puts a large conceptual umbrella over a whole series of practical steps, many of which are familiar to people who have already been involved in discard management. We will now consider those practical steps.

3.2.1 There are no magic machines. Frequently, after giving a blistering attack on the idea of burning trash or dumping it into a mega landfill, we are asked, "Well, if we can't burn it and we can't bury it, what can we do with it?" Such questioners are usually seeking an alternative technology, because they have become accustomed to salesmen that offer them 'turnkey' solutions. "Give us this much money and we will solve your trash problem with our state-of-the-art technology," is what they are used to hearing. At the outset, we have to stress that there are no magic machines that can solve the trash problem. Trash is a not a high tech problem. Technology has a role to play but only when judiciously applied to carefully selected components of the discard stream. Zero Waste is not a technology; it is a strategy and that strategy begins with better industrial design and ends with source separation of discarded products.

3.2.2 Trash is made by mixing. From the citizens' perspective, trash is made by the ten things at the end of our hands, and if we want a solution that we and the planet can live with, it is those ten things that have to be co-opted from the very beginning. In short, trash is made by mixing, and it is prevented by keeping discards

separated at source.

3.2.3 Source separation. Avoiding expensive and potentially dangerous incinerators and huge regional landfills requires keeping our discarded items in several well defined categories (both mentally and physically). These are:

- avoidables
- reusables
- compostables
- recyclables
- toxic materials, and
- residuals (re-designables)

These separated materials will be discussed under the following headings:

3.2.4 Collection systems.
3.2.5 Avoidables and waste reduction strategies.
3.2.6 Reusables and reuse & repair centers.
3.2.7 Compostables and composting facilities.
3.2.8 Recyclables and recycling economics.
3.2.9 Resource recovery parks and ecoparks.
3.2.10 Toxics, household hazardous waste collection, and take-back programs.
3.2.11 Residuals screening facilities.
3.2.12 Better industrial design.

3.2.4 Collection systems. In our view the most successful public collection scheme for the urban setting is a three container curbside system. This has been used in pilot projects in San Francisco and throughout Nova Scotia. There are many variations on such scenarios. A key point to remember when a community is embarking on a source separation system is to organize separation around the existing collection system. If the community is used to curbside collection of trash, then it is best to organize the collection of recyclables and compostables at curbside. If, on the other hand, the community is used to taking discards to the landfill (this is often the case in small rural communities) or a transfer station (sometimes the case in suburbia), then it is best to organize collection at these facilities.

As far as the number of containers used at curbside is concerned, if communities opt for only two, then it is critical to put the emphasis on collecting source-separated organic discards. This is critical for two reasons: a) it is the organic material that causes so many of the problems at landfills and b) it is very difficult, if not impossible, to pick out clean compostables from the residual fraction. Unfortunately, most communities that use a blue box system put the emphasis on collecting recyclables and thus dramatically reduce the amount of material that they can divert from landfill and eliminate the chance of getting good clean organic material for composting.

With these problems in mind, Guelph, Ontario, departed from the blue box approach (containers and paper in one bin and everything else in another) and developed a two-container system that put the emphasis on getting clean organics. They use a green bag for source separated organics, and the residuals and recyclables go into a blue bag. This is called a wet/dry system. The green and blue bags go into two different sections of light weight trucks and are delivered to a facility that has two

sections: a separation line for recyclables and a screening line for compostables. The recyclables are further processed (crushed or baled) to meet market specifications and the compostables are put through a composting operation enclosed in a large building. This two-way division is very simple for the citizen and they have a 98% participation rate. Within a few years the city was achieving a 58% diversion rate from landfill. The city also operates a household hazardous collection depot and a separate collection for bulky yard trimmings[14].

If communities are able to increase the number of containers to four, then its best to have two containers for the recyclables, allowing the separate collection of paper products. This minimizes the contamination of paper with glass shards from the other recyclable fraction (bottles, cans, etc).

Garbage lottery. Some communities have come up with novel ideas to encourage people to separate their discards carefully. Rockford, Illinois, increased its recycling rate fourfold by introducing a garbage lottery. Each week one household is selected at random to have its garbage picked up and examined. If no designated recyclables are found in the trash, they win $1,000! If that is not the case, a householder the following week stands to win $2,000, and so on. The participation rate in this community increased by 400% in a few months. This system is illustrated on two videotapes produced by Videoactive Productions entitled Joe Garbario and the Marin Resource Recovery Plant and Millie Zantow: Recycling Pioneer (see Resources section).

3.2.5 Avoidables and waste reduction strategies. In recent years two key activities have produced astonishing results with respect to waste reduction.

Waste audits. When local manufacturers and businesses are required to find out at what points in their processes that they generate waste, they typically find many places where they can make less waste and save money in the process. For example, Quaker Oats of Canada, after a waste audit, was able to reduce its waste stream by over 90% and save an enormous amount of money in the process. That's truly, a win-win solution.

Volume-based trash charging systems for households and institutions. Simply put, the more waste you generate, the more you have pay. There are a number of different ways of applying this kind of system. The city of Seattle has a monthly garbage fee that is based upon the size of container used for the residual fraction of the discard stream. Households that opt for a large container for their residuals pay a larger monthly fee than household that opts for a small one. Other communities require a pre-paid coupon to be used on every bag of residuals put out at the curb. These are often referred to as 'Pay-by-bag' or 'Pay-as-You Throw' systems. In some communities in the Netherlands there is an electronic microchip in the residuals container and when the can is picked up it is weighed and the household is automatically charged according to how much residual material they have put out.

3.2.6 Reusables and reuse & repair centers. Many householders and communities around the world have developed both formal and informal means of getting reusable objects moving from one owner to the next. These include garage sales, yard sales, jumble sales, flea markets, and thrift shops run by charities like the Salvation Army and Goodwill Industries. Some of these are run for profit and others as a community service.

While reusables represent a small fraction of the discard stream, it is the most

valuable one. Some reuse and repair programs not only recover materials but they also recover people (through job training etc). A municipal official given the responsibility of diverting material from the local landfill needs to investigate how comprehensive the existing services are in his or her community. Such an official should support them in any way possible, including finding ways to bring different reuse and repair functions together in a Community Reuse and Repair Center (the last thing you want to happen is to introduce a facility that puts existing operations out of business). Many models exist.

WasteWise, Georgetown, Ontario. One early example of a community non-profit center is the WasteWise operation. This facility came about because local activists were tired of defending themselves from 'back-end' solutions proposed for their community. They had fought to prevent a large quarry from being used to accept 40 million tons of Toronto's trash and then a 1,500 ton-per-day trash incinerator, again for part of Toronto's waste (Georgetown is about 30 miles from Toronto). They set up WasteWise to show that an alternative approach was possible. With the help of a grant from the Ontario government, they rented a large warehouse and set it up (1) to repair many items like furniture, appliances and bicycles (2) sell these and other ready-to-use items (3) collect, process and sell recyclables not covered by the local blue box (recycling) program, and (4) provide educational services for waste and toxics use reduction. Largely run by volunteers, the operation became self-sufficient after five years and now has two full time staff. A videotape of this operation is available (see Resources section).

The important thing about the reuse and repair center is that it can be the springboard for many other community activities. It can be used for education, especially youngsters, who can be taught how to repair things at an early age. It can provide a venue for senior citizens, many of whom have important repair skills that they are eager to share with the community. It can act as an incubator for small repair businesses by providing affordable overhead. It can be used to teach people how to compost in their backyards and even to build their own composting units out of materials collected at the center. It can also be used to collect potentially hazardous materials like paints, varnishes and cleaners. Paint can either be used in renovation of items for resale or be made available to the public in a 'paint exchange.' The center may also become a meeting place for the community.

Recycle North, Burlington, Vermont. One of the best examples we have seen of a community non-profit operation that includes extensive repair and job training is Recycle North. In addition to a large area devoted to the resale of reusable items, there are four areas devoted to repair. The items that are repaired are (1) large household appliances like stoves and refrigerators, (2) small electrical appliances, (3) electronic equipment and (4) computers. In each section people are trained. After six months they receive a training certificate as well as training in skills needed to get a job (e.g. writing application letters and practicing job interviews). They also attempt to service the local community in other ways. In addition to offering the reusable items at very reasonable prices, they provide these goods in exchange for vouchers provided by the local department of Social Services. In 2000 they generated a gross income of $750,000 and employed over 20 full time staff. They have since added a building deconstruction and salvage service to their operation. A videotape of Recycle

North is in preparation (2001).

Urban Ore, Inc. Berkeley, California. Urban Ore is another excellent example of a reuse and repair center run for profit. It is owned and directed by Dan Knapp[15]. This operation grosses over $1.5 million and has created many permanent and well paid jobs. Urban Ore, Inc. has pioneered the resource recovery park concept (see Resource Recovery Parks section below)

Hobo Hardware, Guelph, Ontario. This large warehouse handles only reused building materials, fittings and do-it-yourself items. Even though the products are all second-hand, it is run as if the items were new, with tidy arrangements and things easy to find. Paul has visited the store and videotaped the operation and hopes to include in a forthcoming video which examines the business opportunities in the community discard stream.

3.2.7 Compostables and composting facilities. Composting can be run on almost any scale. It can be done in the backyard, in the basement with worm bins (vermiculture), in the community or in a centralized facility. However, a key principle is to maintain tight control over what materials enter the composting operation, because the ability to use the material can easily be compromised if unsuitable materials are composted.

In our view, after source separation, composting is the most important step in the community part of the Zero Waste strategy, because it is the organic material in landfills that cause so many problems. When organic material rots underground it generates (1) methane, which contributes to global warming (molecule for molecule methane traps over 20 times more heat than carbon dioxide), (2) organic acids, which are capable of dissolving the metals in the waste load and getting them into surface and ground water, and (3) awful odors, which make landfills so unpopular with the public. Thus a key objective of composting is to keep organic materials out of the landfill.

The key step in Nova Scotia's program was the passing of legislation banning organic material from landfill. Such a regulation forced both source separation at the household and institutional levels, as well as creation of a back-up screening facility at the landfill (see Section 3.2.8).

Backyard composting is the single most cost effective treatment of a large fraction of the domestic discard stream. Seattle has subsidized backyard composting kits and a Master Composters' program, in which citizens are taught all the ins and outs of composting and are then make themselves available to help other citizens troubleshoot their backyard composting problems. The program is run by the Seattle Tilth Association. A video, Zoo Doo and You Can Too! (see Resources section), was made at the association's demonstration site and illustrates many home made and commercially available composting units. In our view, the composting of yard trimmings and food scraps in one's backyard is one of the biggest contributions a citizen can make to solving the trash problem.

Community composting. Composting conducted at the community level is well illustrated by the program in Zurich, Switzerland. A 1991 videotape of this program, Community Composting in Zurich (see Resources section), describes the city's 480 community composting plots involving 3 to 200 households. In August of 2001, Paul revisited the program. The number of community composting operations has risen to

about 1,000 and approximately half the householders of Zurich are now served. Paul also videotaped this and it, too, will be included in a forthcoming video focussing on the full range of methods of handling organic discards.

Mulching lawnmowers. A simple and cost effective way of reducing one type of organic waste is to encourage both householders and institutions to use mulching lawnmowers. This one step saved the New York City's Parks Department over $1 million in avoided disposal costs.

Community gardens. Many citizens who might not be interested in community composting may become excited about a community garden. The latter would be ideally supported with a community composting operation. It makes economic sense for municipalities to support such operations, because every pound of organic material composted means one pound of waste that does not have to be picked up, transported and disposed. It is also a very positive way of integrating discard management with the local community. Such gardens have become havens of delight in New York City and other large cities.

Centralized composting facilities. In the United States there are now over 3,000 yard trimmings composting operations[16]. When handling leaves and brush, the technology does not need to be very sophisticated. Composting yard trimmings usually involves a static pile or windrow system. Such windrows are long rows that have a triangular cross section. They need to be turned regularly to make sure that they get a plentiful supply of air and thus maintain aerobic conditions. They can be turned in one long sweep using mobile turning devices like the Wildcat system manufactured in North Dakota and the Scarab in Texas.

In Nova Scotia centralized composting facilities handle all source separated organic material. Seventy-two percent of the citizens in the province are currently provided with curbside collection of organics (see Nova Scotia video listed in Resources section).

Around the world, many facilities are composting special organic materials, such as food scraps, agricultural waste, fishery waste, sewage sludge and mixtures of these products. To serve these ends, a variety of in-vessel and indoor systems are designed to speed up the composting process and minimize odors. Such systems are either aerobic (plentiful supply of air) or anaerobic (starved of air). The latter are used to generate methane to be used as a fuel or chemical feedstock. Many of these systems are described in articles that appear in the bible for composting: the monthly journal, BioCycle[17]. This journal is an essential resource for any official who wants to include an aggressive composting component in a Zero Waste program.

Vermiculture is the use of worms to degrade organic material. These remarkable creatures provide yeoman service for those prepared to put them to work. One woman, who has worked with worms practically her whole life, is Mary Appelhof, who lives near Kalmazoo, Michigan. Her book, *Worms Eat My Garbage*,[18] is a delight. Her enthusiasm for these industrious worms has no bounds!

The place where vermiculture has received its largest municipal support is in the area around Bombay, India. There they have a variety of vermiculture sites located in backyards, hospital grounds and near local food markets.

3.2.8 Recyclables and recycling economics. According to professional recyclers, the three golden rules to secure markets for recyclables are 'quantity, quality and

regularity.' The industries that will use these materials must be confident that they will get a regular supply of material free from contaminants that can ruin their process, e.g. ceramics in glass, plastics in paper, PVC plastic co-mingled with polyethylene or PET. Source separation schemes have helped to meet some of these demands. The materials recovery facility with human picking lines and along with some mechanical equipment, which can separate steel (magnets), aluminum cans (eddy currents) and plastic cans, helps to complete the process. Hundreds of such facilities are operating around the world. A facility operated by the Miller Corporation in Halifax, Nova Scotia is illustrated in the video, On the Road to Zero Waste, Part I. Nova Scotia, Community Responsibility in Action (see Resources section).

The economics of recycling. Today, the driving force underpinning the economics of recycling is 'avoided disposal costs.' It costs money to recycle, but it is economically viable when the overall cost of collecting and recycling a ton of recyclables is less than disposing a ton of waste. Yard trimmings composting is particularly favorable when making this comparison.

The enemy of recycling is cheap landfills. Those in favor of recycling need to argue that cheap landfilling is artificially cheap because the long term costs of future damage to the environment, both locally (toxic emissions to air and ground water) and globally (waste of finite resources), are being ignored. The web page of the GrassRoots Recycling Network provides more details of the artificial economics of landfilling[19].

Shortage of markets for recyclables is often offered as a reason to limit recycling. However, the markets for certain recyclables are a highly cyclic phenomenon, and certainly should not be used as an argument for building a trash incinerator or mega landfill, which represent a long term (at least a 20-years for an incinerator) capital investment. Communities can insulate themselves from the vagaries of commodities markets by developing local markets for their recyclables. For example, when Arcata, California, lost their market for glass they developed Fire and Light, an upscale tableware company that uses exclusively recycled glass from the Arcata Community Recycling Center. Similar business opportunities exist with wood, tires, plastics, and other materials. Communities are well served if they invest in and/or support business opportunities that use the materials they generate but for which markets are poor. This creates other economic benefits too, like jobs and sales taxes.

We argue that if we are forced to bury stuff, then this stuff shouldn't have been manufactured in the first place. Some activists advocate a 'return to sender' approach as a way of drawing attention to bad examples of industrial design such as the silly squeezable ketchup bottle. Paul has provided a great deal of amusement at the expense of this particularly bad form of packaging. A little thought would suggest that a simple spoon could deliver ketchup just as precisely from a recyclable or reusable jar, with a wider opening, as a non-recyclable plastic ketchup bottle.

A net profit. The way for recycling to generate a net income for the community is to find ways of utilizing the salvaged materials locally. Examples include: newspaper to make cattle bedding, or insulation material; glass to make fiber glass; tires to make crumb rubber; crab shell waste to make surgical sutures and dietary products; post consumer wood to make fiber board, furniture or flooring, old building materials used to make furniture and old carpets used to make new ones.

Dr. Robin Murray, in his book Creating Wealth from Waste (see Resources

section), provides a very persuasive strategy to encourage companies to move to cities in order to capture the flow of separated resources generated there. Such an approach means that local, rather than distant, economies can capture the 'value added' of local manufacture.

3.2.9 Resource recovery parks and ecoparks. Looking to the future, visionaries like Dr. Dan Knapp of Urban Ore, Inc. envisage Resource Recovery Parks and Ecoparks as the community replacement facilities for landfills and incinerators[20]. These facilities locate reuse, recycling and composting businesses close together and can be the core of a comprehensive strategy for local resource management. Local collection entrepreneurs and the public can deposit all recoverable materials at one processing facility, get paid for some of them and buy other items at bargain prices. Some designs place the recovery park together with a waste facility or transfer station, arranged so that traffic passes recovery businesses before coming to the waste facility. When combined with incentives for recycling, disincentives for wasting, and a commitment to gradually phase out the waste facility, such an arrangement can be the centerpiece of a Zero Waste community.

Resource recovery parks can be privately financed, or local government can create an authority whose role is to secure the land, build the core facility and lease space to private entrepreneurs, as is frequently done for airports. When located close to appropriate industries, resource recovery parks can provide feedstocks for Eco-industrial parks, where the byproducts of one industry become inputs for the next[21].

Serial resource recovery systems, are a variation of resource recovery parks where a critical mass of resource conservation businesses are located in a neighborhood, but not necessarily on the same property. Repair shops and secondhand shops are good examples of existing businesses that need only to bring their services into greater synergy and prominence in a Zero Waste system.

Urban Ore Ecopark, Berkeley, California, USA. Urban Ore, Inc. has pioneered the resource recovery park concept. In 2001, Urban Ore moved to a 2.2-acre former steel pipe manufacturing facility and established a building materials exchange, a hardware exchange, an arts and media exchange, a general store, and salvage and recycling activities. Two major lumberyards, a hardware store and two other reuse facilities, all in a three-block area, provide a stream of potential customers. Urban Ore Development Associates (UODA), a spin-off of Urban Ore, designs, builds and operate resource recovery parks[22].

Other Resource Recovery Parks are in development:

San Leandro Resource Recovery Park, San Leandro, Calif., USA. Waste Management, Inc. is developing a resource recovery park that recycles wood, greenwaste, curbside and other recyclables, operates a buy-back center, and sells recycled-content soil and landscape products. Tenants include a tire recycling and crumb rubber facility and a building materials exchange. The park is at a waste transfer site.

Monterey Regional Environmental Park, Marina, Calif., USA. This park includes public drop-off and commercial waste recycling stations, a Last Chance Mercantile reused goods resale operation, a landfill gas power project, a household hazardous waste collection facility, construction and demolition recycling operations, composting facilities, and a soils blending facility, at an existing regional landfill.

3.2.10 Toxics, household hazardous waste collection, and take-back

programs. While toxics only make up 1-2% of the household waste stream, if ignored, they threaten other aspects of the Zero Waste strategy. It is important to get these materials identified and made visible.

Curbside collection. Some communities have organized separate curbside collection of certain toxics like automobile oil (Hamburg, NY) and batteries (Neunkirken, Austria).

Household hazardous waste collection sites. Some communities have organized household hazardous waste collection days, on which citizens are requested to bring their hazardous materials to a central collection point. In Halifax, Nova Scotia, there is a very well organized and efficient drop-off facility operating most Saturdays from 9- 4 p.m. This facility is illustrated in the video, *On the Road to Zero Waste, Part I. Nova Scotia, Community Responsibility in Action* (see *Resources* section). Some communities have set aside buildings at the landfill to collect, store and even exchange potentially hazardous materials, like paint, with the community.

Use it up. Some paint manufacturers have offered to reblend recollected paint and donate it for community projects. In New Brunswick, Canada, there is a company specializing in collecting used paint and recycling it into new paint.

In the absence of a commercial operation we would advocate the use of a Community Reuse and Repair Centers (see above) to collect paint and use it for community projects. The principle is a simple one: if it is safe enough to use (and it may not be, but this is a different issue) then it is safe enough to use up. If the individual cannot use it up, the community should.

Producer Take-Back. Some toxic substances, like mercury, are so intractable that we should question their use altogether. If industries insist on mercury's continued use and governments allow them, then legislation should be introduced that would require these industries to take back the mercury-containing objects, such as household batteries, thermometers, and fluorescent lights. A citizen who has devoted more than a decade to getting governments and industries to eliminate the mercury problem, is Michael Bender in Vermont USA[23].

In a similar fashion to mercury, we should require the oil industry to take back used motor oil, and tire manufacturers (where communities don't have access to modern tire recycling facilities like the one in Nova Scotia) to take back used tires. These manufacturers should be challenged to find chemical ways of recovering these valuable feedstocks and put them back into their manufacturing process. They need to 'close the loop.' This is called *Extended Producer Responsibility* for waste or EPR (see Section 4.2).

Retailer Take Back. Ottawa, Canada, has a successful 'Take It Back!' (to retail) program in which over 350 retailers take back from customers 65 different toxic and difficult-to-recycle products that do not belong in curbside recycling bins[24]. These items include used motor oil, batteries, consumer electronics, and prescription drug containers, among others. Retailers are anxious to get involved because of the free publicity and the way being on the program attracts customers into their stores. Trish Johnson, who directs this award-winning program, described some of the details in the video *Target Zero Canada* (see *Resources* section). Inspired by the Ottawa example, Washington County MN, USA, has introduced a similar program.

While Retailer Take Back programs put the emphasis on retailer responsibility

for waste, the ultimate goal is to build a community coalition to increase pressure on the manufacturers, or Brand Owners, who profit from making products that become waste, and, more importantly, who make the design decisions on toxicity, durability and recyclability of products and packaging. And in the meantime, such programs educate citizens that there is no *a priori* reason that taxpayers have to continue to clean up after industry. We anticipate that as the program evolves and retailers question the expense of disposing brand name products, retailers will begin to put pressure on manufacturers to take financial or physical responsibility for their products at end-of-life.

3.2.11 Residuals screening facilities. After source separation has kicked in and materials like reusables, recyclables, compostables and hazardous materials have been sent to different facilities for processing, there will still be a fraction left over: *the residuals*. This fraction consists largely of the items that are deemed to be currently non-reusable, non-recyclable or non-compostable. To this we have to add materials that individuals or institutions have not bothered to put into the correct container.

Ultimately, in the Zero Waste strategy we have to develop creative and forceful ways of telling manufacturers that if the community cannot reuse, repair, recycle or compost these objects or this material, they should not be making them (see *Industrial Responsibility*, below).

In typical communities in North America, once the community has done what it can with recycling and composting, the residue is shipped off to landfills. Often these landfills are very distant and very large. The rationale for their development has been the need for expensive and complicated engineering systems to contain, collect and treat the leachate (garbage juice!) that emerges from them. This equipment, along with the lining systems, is so expensive that it is usually cost-prohibitive for the community to use this back end approach on a small scale for local needs; hence, the drive for regional facilities.

We have argued that, despite this equipment and these lining systems, all landfills eventually leak toxic materials into the ground water and emit other polluting gases and particulates into the air. We have further argued that if engineers cannot control what comes out of a landfill, the community's only rational choice is to control what goes in.

Controlling what goes into a landfill. There are two stages at which control can be exerted over what goes into the landfill. The first stage comes from source separation prior to curbside pick up, leading to all the measures discussed in the activities described above (e.g., reuse, repair, composting, recycling and toxic removal). The second level of control can be exerted immediately prior to landfill in a residual screening facility.

We further argue that, if the residual screening facility is properly overseen by the community, there will be little or no need to build huge regional landfills. With community controlled screening facilities we can return to the small, locally operated landfill.

One of the first such screening facilities is operating in Halifax, Nova Scotia and is illustrated in the videotape, *On the Road to Zero Waste, Part I. Nova Scotia, Community Responsibility in Action* (see *Resources* section). This screening facility, locally called a 'front-end processing facility,' starts with conveyor belts manned by well-protected

workers. These workers separate out more recyclables (which escaped the source separation net), bulky items, and toxic materials like batteries and paint cans (which escaped household hazardous waste drop off centers). They leave on the conveyor belts (i.e., using a negative sort) a dirty organic fraction as well as a variety of non-recyclable plastic items. This material is shredded and put through another composting process. The purpose of this operation is to stabilize the dirty organic fraction biologically for 21 days prior to landfilling. With more effective source separation and longer curing times this material might (after the plastics are removed) eventually be used for landfill cover. When Paul visited the landfill at the end of this operation he was struck by how odor-free the landfill was and the almost total absence of seagulls or other birds.

We would argue that, if the screening facility is properly overseen by the community, there will be less, or no, need to build huge regional landfills with elaborate lining systems. With community controlled screening facilities we can return to the small, locally operated landfill. In Halifax, however, they have backed up their 'residual screening facility' with a double lined, leachate collecting system at the landfill. While, it may be a good idea to have a back up, the danger is that this back end support might eventually undermine the care with which toxics are removed and organics are stabilized.

3.2.12 Better industrial design. This is not the end of the road to Zero Waste. Even though the material exiting a 'residual screening facility' may be biologically stable and safe to bury, it still represents a waste of resources, some of them in finite supply. We believe that the objects and materials that end up in this interim landfill should be studied, possibly by research students destined to work in manufacturing industries. They should be challenged to recommend design changes in manufacturing to avoid this fraction in the future. In short, we need *better industrial design* for the 21st Century. In our view, this is where community responsibility can help drive industrial responsibility.

3.3 Community Success Stories

In the late 1980's, Dr. Barry Commoner and co-workers performed an experiment in East Hampton, Long Island in the state of New York[25]. With the help of 100 volunteer families they measured how much diversion from landfill could be achieved with a four-container system and existing commercial recycling and composting facilities. They used one container for bottles, cans and other hard recyclables, a second container for all paper products, a third for the compostable fraction (they used a multiply kraft paper bag for this fraction), and a fourth container for the residuals. In this experiment they achieved a remarkable 84% diversion from landfill.

Critics have argued that this sample is not a representative of the American people and that the 100 families were highly committed to the success of the project. We would argue that this is precisely the point. This experiment showed how much diversion was physically possible when you had a very strong commitment from householders. From our point of view, it clearly underlines the need to spend sufficient money from the waste budget on the kind of education programs that might generate this kind of commitment.

USA recycling rate. Despite the pessimistic projections of waste experts in the early 80's, who suggested that the maximum recycling rate you could expect from a

typical American community would be about 15%, Americans have done far better than this. A survey financed by the US Environmental Protection Agency indicates that over the whole country, in 1996, Americans were recycling 27.3% of the municipal discard stream[26], with nearly 9,000 curbside recycling programs in operation[27]. But that is for the whole country. This includes states that are recycling a lot and others that are doing very little.

NJ recycling rate. Without including junked automobiles and construction and demolition debris (C & D), the state of New Jersey is diverting over 45% of its municipal discards from landfills. If we include the autos and C & D, they are diverting over 60%.

California recycling rate. California has a recycling law that required communities to divert 50% of their discards from landfill by the year 2000. Over 60 communities had reached that target by 1996, and as many as half of all communities may have actually reached the target on time (reports are not due until the end of 2001)[28].

Nova Scotia recycling rate. In 2000, the province of Nova Scotia became the first province in Canada to achieve a 50% diversion from landfills.

Recycling in Communities. While states and countries can stimulate recycling with appropriate legislation, incentives and government purchasing, it is not states or countries but communities that recycle. National statistics that combine data from both excellent programs with very poor ones give a misleading impression of what an individual community can achieve. Thus officials from a village, town or city who are wondering how much they can divert from a landfill should comb the world, and the Internet, to see how much a community of their size and demography has actually achieved and consider whether they can copy their example or improve upon it.

Nova Scotia communities. A good place to start would be the Canadian province of Nova Scotia. In the sections above many of the details of this program have been described. Their program includes: backyard composting, curbside collection of all other separated organic material, curbside collection of recyclables, drop off facilities for all beverage containers except milk cartons (there are 95 eco-centers scattered throughout the province that collect these deposit containers), deposits on tires and recycling of tires to crumb rubber, household toxic waste collection sites and a 'residual screening facility' to handle and process the residuals prior to landfilling. Only non-toxic , non-recyclable and non-biodegradable materials are accepted at the landfill. Remarkably in just five years, the program has achieved over 50% diversion from landfills and in the process has generated over 3000 jobs. If we exclude construction and demolition ('C&D') debris, the city of Halifax in the year 2000 had reduced the amount of discards (calculated per capita to allow for population growth) going to landfill by nearly 60% over 1989 figures.

Citizen driven. A very exciting element in the Nova Scotia program is that it has been largely driven and designed by citizens, particularly the 'It's Not Garbage Coalition.' It was the citizens who produced a report in which the word 'waste' was struck out every time it appeared and replaced with the word 'resources.' To their credit, the Nova Scotia authorities, after initially proposing a trash incinerator to get them out of their landfill woes, have worked with citizens to make this program possible. Indeed, following the citizens' cue, Barry Friesen's title at the Ministry of Environment and Labor is 'Solid Waste Resource Director'.

United States communities. From 1996 to 1998, the Institute for Local Self-Reliance identified 100 communities and nearly 200 businesses, institutions, and other organizations reporting waste reduction rates at 50 percent or higher. The results of that survey are summarized in a report, Cutting the Waste Stream In Half: Community Record-Setters Show How, much of which is posted on ILSR's website[29]. The next two communities are from that study.

San Jose, California, USA (population 849,363). 60% of materials from single-family households are recycled or reused; 47% of overall municipal solid waste is diverted from landfill; businesses receive financial incentives to reduce waste.

Loveland, Colorado, USA (population 37,352). This rural community recovers 56% of residential materials for reuse and recycling using dual-collection vehicles that pick up both recyclables and trash.

Guelph, Ontario, Canada (population, 100,000) 58% of materials diverted from landfill. Uses wet/dry collection system. 98% participation rate. No waste goes direct to landfill. 67% diversion of wet waste. 51% diversion of dry waste. Overall: 58% diversion[30].

Belleville, Ontario (population 37,000) 63% reduction to landfill.

Sidney, Ontario (population, 17,000) 69% reduction to landfill.

Trenton Ontario (population, 15,000) 75% reduction to landfill.

These three towns are part of a 15- municipality blue box-2000 program. 20 materials are collected at curbside. They use a 'pay-by-bag' system and provide incentives to residents to compost in their backyard (65% participation rate)[31].

Canberra, Australia (population 273,300). 51% diversion from landfill in 1996, 12% of this was construction and demolition debris[32].

Bellusco, Italy (population 6,000). This small town is in the Milan area. 73% of municipal discard stream is diverted from landfill. Curbside collection of paper and green waste. Drop-off containers plus a very smart drop-off center run by volunteers[33].

Gazzo, Italy (population 3,220). Community near Padua. 81% diversion from landfill. No details[34].

4. INDUSTRIAL RESPONSIBILITY [35]

4.1 Introduction

The two major reasons we have become a toxic, throwaway society are that (1) taxpayers subsidize the extraction of virgin materials that compete with recovered (or secondary) materials, and (2) taxpayers assume the burden of disposing whatever products and packaging industry chooses to market. Hitherto, however, taxpayers and local government have had little say in the production of things that become waste. The Zero Waste strategy requires that this connection be made.

4.2 Producer Take Back

Producer Take Back, or Extended Producer Responsibility (EPR) for waste, holds manufacturers, and specifically brand owners, responsible for managing their products and packaging at the end of their useful life. When brand owners have physical or financial responsibility for their products and packaging at end of life, they have a built-in incentive to use less toxics, make more durable and recyclable products, and reduce excessive packaging.

EPR was first mandated in Germany for packaging in 1991, and is now being applied to packaging and other product sectors in most of the world's industrialized countries. A notable exception is the United States[36]. EPR policies in Europe have led to company recycling rates close to 90% and high recycled content, as well as an emphasis on reusable and returnable packaging. The policy has spread to other countries as well, including Canada and nations in Asia and Latin America. Often, U.S.-based companies follow EPR requirements in other countries but do not replicate the programs in the United States.

Examples of EPR programs in the United States and Canada include:

Deposit Systems for Beverage Containers. Deposit systems transfer the costs of recycling from taxpayers to consumers and beverage manufacturers. Deposits are not only fair; they work. In the ten U.S. states with container deposits, recycling rates average 80% for containers covered by deposits, compared with far less in non-bottle bill states (for example, around 10% for plastic soda bottles in non bottle bill states). In Canada, where the beer industry invested in refillable glass bottles, 97% of bottles are returned to the producer for refilling[37].

Take-Back Programs for Toxics. British Columbia's Product Stewardship laws require producers to take back household chemicals such as paint, thinners, pesticides, fuels and medicines for recycling or safe disposal. Millions of gallons of these toxic chemicals are collected at industry-funded depots at no cost to local communities. The costs create incentives for producers to keep toxic leftovers to a minimum.

Local Take Back to Retail. Ottawa, Canada, and Washington County, Minnesota USA, have implemented successful programs targeting problematic wastes not covered by curbside programs, as an alternative to taxpayer funded Household Hazardous Waste programs. Retailers like the program for its free publicity and opportunity to get return customers. These are examples of voluntary Retailer Responsibility programs that can complement other Producer Responsibility programs.

4.3 Environmentally Preferable Purchasing

Any organization, business or individual can promote Zero Waste by altering buying habits. Many government agencies and companies have already adopted preferences for recycled content products. Many are now moving to broader, environmentally preferable purchasing programs seeking to reduce resource use, cut air and water emissions, or achieve other environmental goals. Purchasing practices can target:

- materials purchased for manufacturing products and packaging;
- products purchased for use within the organization;
- packaging for products and materials delivered to the organization; or
- products specified through contractors, such as direct mailers, billing agents, printers, copier companies, office products retailers, architecture and construction companies.

Examples:

a) U.S. Federal Agencies. As a result of Executive Orders in the 1990s, federal agencies are taking the lead in buying recycled paper and other recycled products, as well as products that include features such as reduced toxics and reduced energy needs[38].

b) King County, Washington USA is a national leader in buying environmentally preferable products and has an excellent website. Likewise, the Pacific Northwest Pollution Prevention Resource Center has excellent resources on its website[40].

4.4 Product and Packaging Design

Many companies have been innovative in redesigning products, whether to reduce costs or to meet government incentives or requirements. Some have redesigned packaging to minimize materials. Others have redesigned products for ease of reuse and recycling. Still more have transformed the concept of their products to eliminate waste. Extended Producer Responsibility encourages manufacturers to design products for easy disassembly, to minimize the cost of manufacturer responsibility for recycling. A few examples include:

Interface, Inc. (Dalton GA, USA) This maker of commercial carpets is changing its focus from providing a product to providing a service, leasing carpets to customers and taking back old carpet and tiles for refurbishing or recycling. Interface also pioneered the practice of installing carpet in tiles, so that only high wear places need to be replaced when worn out.

Herman Miller (Zeeland MI, USA) In manufacturing office furniture, Herman Miller used to receive molded plastic chair seats in single-use cartons containing shells in bags, separated by chipboard sheets, placed 56 to a double-sided corrugated box. After unpacking the seats and assembling the chairs, Herman Miller was left with 30 pounds of packaging for every 56 chairs. The company developed, with its vendor, a protective rack that stores 90 seats in the space that previously housed 56 and can be reused 80 to 100 times or more.

4.5 Comprehensive Zero Waste Business Approaches

Businesses pursue Zero Waste, in addition to redesigning products, by:

- Re-evaluating products and services to create the greatest consumer and environmental value within economic feasibility;
- Minimizing excess materials and maximizing recycled content in products and packaging;
- Finding productive uses for, reuse, recycling or composting over 90% of their solid waste;
- Reducing procurement needs, then specifying products that meet Zero Waste criteria;
- Establishing easily accessible repair systems, as well as recovery processes for packaging and products.

Examples:

Collins & Aikman, Dalton, Georgia, USA[41]. These makers of automotive fabric and trim sent zero manufacturing waste to landfill in 1998. Waste-minimization and energy-efficiency programs boosted production 300% and lowered corporate waste 80%.

Xerox Corporation, Rochester, NY, USA[42]. Xerox instituted an Asset Recycling Management program in 1990 as a cost saving rather than an environmental initiative. It is an example of a win-win voluntary EPR initiative. In 1997, it saved the company

$40 to $50 million and resulted in the remanufacture of 30,000 tons of returned machines. According to Bette Fishbein of INFORM, Inc.[43], it is an approach that can serve as a model for many companies, though it may only be profitable for high-value products. Even Xerox has found that for lower-value equipment such as fax machines, the ARM program generates net costs rather than savings.

Xerox corporation, Venray, Netherlands. Venray is the manufacturing headquarters of the Xerox corporation in Europe. There, Xerox operates a massive 'reverse distribution service' to recover old copying machines from 16 European countries. They reuse these machines or reuse their parts, or recycle their materials. They are only sending 5% of the returned materials for waste disposal. In 2000, this operation saved the company $76 million in reduced production costs and avoided disposal costs. This operation will be the subject of a future video: On the Road to Zero Waste. Models of Industrial Responsibility.

ZERI Breweries, Africa, Sweden, Canada and Japan[44]. The Zero Emissions Research and Initiative (ZERI) Foundation has helped design breweries that utilize 40 different biochemical processes to reuse everything, including heat, water and wastes. A digester transforms organic wastes into methane gas for steam for fermentation. Spent grain is used to grow mushrooms. Alkaline water supports a fish and algae farm.

Fetzer Vineyards, Hopland, California, USA[45]. Fetzer recycles paper, cardboard, cans, glass, metals, antifreeze, pallets and wine barrels, composts corks and grape seeds. Garbage was reduced by 93% in the past several years, with a goal of no waste by 2009.

5. THE NEED FOR GOOD LEADERSHIP

When we examine successful cases of Zero Waste, it is clear that leadership has come from all the areas of business, government and non-governmental organizations. We can anticipate even more leadership from the business community because reduction in waste here is indelibly linked to economic benefit.

When we look at communities that have achieved major breakthroughs, we find the key to their success is the fact that the government was prepared to work with community activists to design their programs. This was the case in Canberra, Australia, which first introduced the 'No Waste to Landfill' concept in the mid-nineties, and the province of Nova Scotia, in Canada, which has diverted 50% from landfill in just five years. The message is a simple one. As far as a genuine sustainable solutions are concerned, the future belongs to those in local government who put their faith in people, not 'magic machines'.

6. CONCLUSION

We would not wish to imply that achieving Zero Waste, or even getting close, is going to be easy. While simple in principle, the execution of these systems requires a lot of hard work, perseverance and creativity from the organizers in the community and in industry .We believe that adopting the Zero Waste goal as a local government or industry policy is the best way to get started. It forces the paradigm shift. It transforms the task from getting rid of waste to saving resources.

We should recognize that currently there is a considerable amount of tension between long-term goals and interim solutions. While the long term goal is to have

no landfills, in the interim we need some kind of landfill to handle the non-toxic and non-biodegradable residuals. The worry is that these 'interim' landfills may get fossilized unless citizens keep the pressure on local officials to live up to their Zero Waste commitment. Similarly, there are some commentators who are uneasy about how much money communities are putting into curbside collection of recyclables, when they believe that ultimately the collection (and re-design) of their packaging should be industry's responsibility.

For industrial officials, in addition to reducing toxic use and resource conservation, it means searching for ways of getting back objects and materials from their customers so that they can be used again. If the huge Xerox corporation can take on the daunting task of recovering its used copying machines (which contain over a 1,000 parts) from all over Europe, and clean, repair their parts or recycle their material components, any manufacturer *should be able to do it.* Moreover, when manufacturers hear that Xerox is saving $76 million a year doing this, they *should want to do it!* Moreover, once companies take on such a recovery task, it then feeds into the need to design new products with this ultimate goal in mind i.e. to make them easier to disassemble and reuse their constituent parts.

For the local official, the new Zero Waste paradigm, transforms the old 'waste disposal' task from the distressing one of looking for new landfill or incinerator sites, to a much more exciting one of searching for entrepreneurs who can create viable businesses that utilize discarded objects and materials. This task is better both for the planet and the bureaucratic 'psyche' than attempting to locate a hole in the ground or a non-existent 'magic machine' that will make the problem disappear.

The Zero Waste paradigm also offers another challenge and reward and that is working constructively with citizen activists rather than dreading their appearance at public meetings!

Our experience has convinced us of several things:

a) However daunting the task may appear, the Zero Waste approach is moving our society in the right direction.
b) It is certainly far superior to a reliance on raw waste landfilling or incineration.
c) It will improve as more and more manufacturers learn to combine selling to the present with sharing our limited resources with the future.
d) As far as community responsibility is concerned. People are not the problem. Once they recognize that source separation is easy, that it is in the best interests of their children and those in charge have organized effective systems to handle the materials they separate, they readily cooperate to make the system work.
e) As far as the local economy is concerned the pay off is far greater than the dead end of landfills and incinerators. With the latter a huge amount of money is put into complicated machinery and most of it leaves the community, and probably the country, in the pockets of multinational corporations. Whereas, with the low-tech components of the Zero Waste program most of the money stays in the community creating local businesses and local jobs.
f) Finally, we believe that the Zero Waste approach is the one that is most likely to lead to questions on how we should be living on a finite planet.

Today, with so much that we do, we are living on this planet as if we had another one to go to! The average person's most concrete connection to this important realization is our trash. The way we handle our discarded material is a microcosm of the way we handle our planet. If we care about the planet we have to care about the way we treat our discarded materials

While the economic and environmental benefits of a Zero Waste goal are very clear, ultimately the issue is an ethical one. Alan Durning brilliantly outlines the ethics in his book *How Much is Enough?*[46] He shows how a combination of slick advertising and too much time in front of the TV has trapped so many of us in a mindless binge of consumption. But the good news is that it is not making us very happy. Durning points out that while Americans are consuming in 2000 about five times more per capita than our ancestors in 1900, we are not five times happier. Meanwhile, the gap between our consumption patterns and the poorest fifth of the world's population steadily increases. As Mahatma Gandhi so succinctly and wisely put it, "The world has enough for everyone's need, but not for everyone's greed."

In short we have been seduced into believing that happiness lies in the series of objects we buy, rather than the relationships we nurture with our friends, our loved ones and our community. Thus in our view the antidote to over-consumption is community building.

If we are to succeed, the task of achieving, or moving towards, a Zero Waste society must be seen to be exciting, challenging and fun. If we approach it only with a sense of moral duty, and not with a sense of business opportunity, we will probably fail. If we approach reduced consumption with a sense of loss, rather than the opportunity to regain our 'sense of community' we will certainly fail. As far as having fun is concerned, We cannot think of anything quite as challenging, and as exciting, as having people in our communities, from businesses, from government and from activist circles, working together to create a community that is determined to share as much of their resources with the future as it can. Especially if we remember to celebrate often.

7. ZERO WASTE RESOURCES

VIDEOS

- Zero Waste: Idealistic Dream or Realistic Goal? (1999, 58 minutes; 2000, 28 minute version). This video was produced by Paul Connett, of Grass Roots and Global Video (GGvideo) with the help of the GrassRoots Recycling Network. The video conveys a sense of excitement, immediacy and practicality about recycling, reuse, deconstruction, sustainability and zero waste. It has been translated into two languages and distributed, by Essential Action, to activists in 20 countries.
- Target Zero Canada (2001, 51 minutes) covers the launch of a Zero Waste strategy for Canada and elaborates on principles and practicalities of the Zero Waste concept in both Canadian communities and industries. (See description in Section 1, above.)
- On the Road to Zero Waste. This new series of videotapes will spotlight successful initiatives in communities and businesses that illustrate community

responsibility, industrial responsibility and political leadership needed to get to Zero Waste. The series is being produced by GG Video and co-sponsored by Waste and Environment (Netherlands) and the GrassRoots Recycling Network (USA).

- Part 1. Nova Scotia: Community Responsibility in Action (32 minutes, 2001). This videotape covers many aspects of a Zero Waste program as described in this paper.

Videos by Paul Connett and GG Video can be purchased from the GrassRoots Recycling Network, by check to GRRN, P.O. Box 49283, Athens GA 30604-9283 (Tel: 706-613-7121), also described at www.grrn.org. All videos are $12 (postage included) for grassroots activists (add $6.00 to cover international postage), and $25 for libraries, local governments and all others. Check the status of new videos on www.grrn.org/order.

Earlier videos by Paul Connett referred to in the text were produced by Video-Active Productions and are available from GG Video, 82 Judson Street, Canton, NY 13617. Phone 315-379-9200. Fax: 315-379-0448. Email ggvideo@northnet. All videos are $12.00 (postage included. Add $6.00 for international postage).

- WasteWise: A Community Resource Center (1991)
- Community Composting in Zurich (1991)
- Zoo Doo and You Can Too (1988)
- Joe Garbarino and the Marin Resource Recovery Plant (1987)
- Millie Zantow: Recycling Pioneer and the Trashman (1987)

RECENT BOOKS & REPORTS

- Creating Wealth from Waste, by Robin Murray (London: Demos, 1999).
- Zero Waste Briefing Kit, by GrassRoots Recycling Network (2001).
- Wasting and Recycling in the United States 2000, by Institute for Local Self-Reliance for GrassRoots Recycling Network (2000).
- Welfare for Waste: How Federal Taxpayer Subsidies Waste Resources and Discourage Recycling, by GrassRoots Recycling Network, Taxpayers for Common Sense, Friends of the Earth, Materials Efficiency Project (1999).
- Materials Matter: Toward a Sustainable Materials Policy, by Ken Geiser (Cambridge: MIT Press, 2001).

Most items listed above can be previewed and purchased on the GrassRoots Recycling Network website at www.grrn.org/order/order/html.

ZERO WASTE WEB SITES

- GrassRoots Recycling Network

www.grrn.org

- Zero Waste New Zealand

www.zerowaste.co.nz

- Target Zero Canada

www.targetzerocanada.org

ENDNOTES

ENDNOTES

1 This guide may be downloaded from the internet at www.grrn.org/zerowaste/zerowaste/community

2 The GrassRoots Recycling Network (GRRN) is a North American network of waste reduction activists and professionals dedicated to achieving sustainable production and consumption based on the principle of Zero Waste. Founded in 1995 by members of the Sierra Club Solid Waste Committee, the Institute for Local Self-Reliance, and the California Resource Recovery Association, GRRN uses grassroots advocacy, organizing and activism to advance policies and practices based on government, corporate and individual accountability for waste (see footnote on page 1 for contact information).

3 Renine, C., and A. MacLean (1989). Salvaging the Future, Institute for Local Self-Reliance, ISBN: 0917582373.

4 Platt, B., and N. Seldman (2000). Wasting and Recycling in the United States 2000, Prepared by Institute for Local Self-Reliance for the GrassRoots Recycling Network, 64 pages. Seldman, N. (1995). 'History of Recycling in the U.S.,' Encyclopedia of Energy, Technology and Environment (New York, Wiley Brothers).

5 See Zero Waste New Zealand Trust website: www.zerowaste.co.nz. Contact: Warren Snow, email: wsnow@envision-nz.com

6 Murray, Robin, Creating Wealth from Waste, (London: Demos, 1999). Email: postmaster@ecologika.demon.co.uk (see Resources section).

7 Target Zero Canada, Website: www.targetzerocanada.org

8 Arne Schovers, Waste and Environment; Email waste.and.environment@hetnet.nl

9 The mission of Grass Roots and Global Video is to: (1) expose environmental injustice; (2) communicate scientific controversy with integrity and clarity; and (3) spotlight communities, institutions and companies that are pursuing sustainable solutions to environmental problems (see footnote on page 1 for contact information).

10 See website: www.act.gov.au/nowaste

11 Contact: Del Norte County Solid Waste Management Authority at 707-465-1100 or email: recycle@cc.northcoast.com . The Del Norte County Waste Management Authority Zero Waste Plan (February 2000) can be viewed at www.grrn.org/order/order.html#del_norte

12 See website: www.zerowaste.co.nz. Contact: Warren Snow, Email: wsnow@envision-nz.com

13 See website: www.ci.seattle.wa.us/util/solidwaste/SWPlan/default.htm

14 Roumpf, J. (1998). 'Wet- and dry -all over,' Resource Recycling, April 1998, 29-34; Kelleher, M. (1998). 'Guelph's Wet-Dry System. Up-to-date costs are now available,' Solid Waste and Recycling, Feb/ March 1998, 34-35.

15 Contact: Dr. Dan Knapp, Urban Ore, Inc., 6082 Ralston Avenue, Richmond, CA 94805. Phone: 510-235-0172, Fax: 510-235-0198; Website: urbanore.citysearch.com/1.html

16 Glen, J. (1998). 'The State of Garbage in America,' BioCycle, April 1998, 32-43.

17 BioCycle, Journal of Composting and Organics Recycling, published monthly by the JG Press, Inc. ISSN 0276-5055. Subscription offices: 419 State Avenue, Emmaus, PA 18049; Tel: 215-967-4135; Website: www.biocycle.net

18 Contact: Mary Appelhof, Flowerfield Enterprises, Inc., 10332 Shaver Rd., Kalamazoo, MI 49024; Tel: 616-327-0108; Fax: 616-327-7009; Website: www.wormwoman.com

19 See website: www.grrn.org/landfills.html#resources

20 Urban Ore, Inc. (1995). Generic Designs and Projected Performance for Two Sizes of Integrated Resource Recovery Facilities, for the West Virginia Solid Waste Management Board, January 1995 (order at www.grrn.org/order/order.html)

21 See Resource Recovery Parks: A Model for Local Government Recycling and Waste Reduction, by Gary Liss for the California Integrated Waste Management Board, 2000 (www.ciwmb.ca.gov/LGLibrary/Innovations/RecoveryPark). Contact: Gary Liss; Tel: 916-652-7850; Email: gary@garyliss.com; Website: www.garyliss.com

22 Contact: John Moore, UODA, 1970 Broadway, Suite 950, Oakland, CA 94612, 510-893-6300 or jmoore@recyclelaw.com

23 Contact: Michael Bender; Tel: 802-223-9000; Email: MTBenderVT@aol.com; Website: www.mercurypolicy.org

24 Ottawa Take It Back! website: city.ottawa.on.ca/gc/takeitback/index_en.shtml . See also www.grrn.org/resources/ottawa_take_it_back.html

25 Commoner, Barry, et al (1988). 'Intensive Recycling: Preliminary Results from East Hampton and Buffalo,' presented at the Fourth Annual Conference on Solid Waste Management and Materials Policy, Jan 27-30, New York City. Copies available from CBNS, Queens College, Flushing, NY 11367. Phone: 718-670-4192.

26 US EPA (1998), Characterization of Municipal Solid Waste in the US: 1997 Update (EPA 530-R-98-007).

27 Glen, J. (1998). 'The State of Garbage in America,' BioCycle, April 1998, 32-43.

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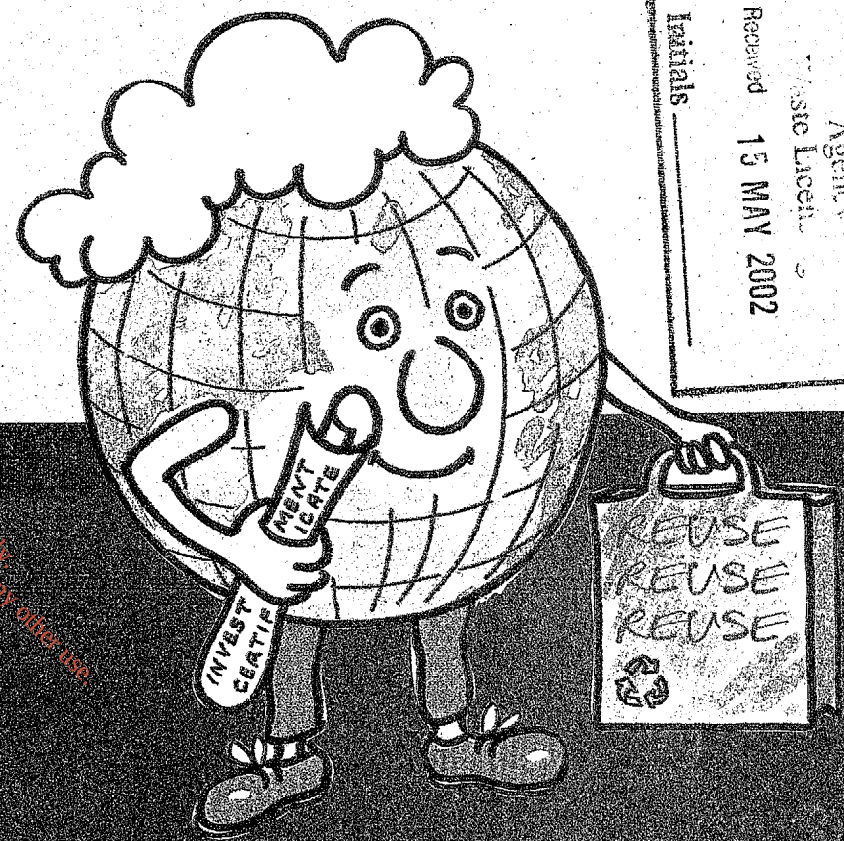

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ORIGINAL CONTRIBUTIONS

Health Effects of Dioxin Exposure: A 20-Year Mortality Study

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Follow-up of the population exposed to dioxin after the 1976 accident in Seveso, Italy, was extended to 1996. During the entire observation period, all-cause and all-cancer mortality did not increase. Fifteen years after the accident, mortality among men in high-exposure zones A (804 inhabitants) and B (5,941 inhabitants) increased from all cancers (rate ratio (RR) = 1.3, 95% confidence interval (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), with no latency-related pattern for rectal or lung cancer. An excess of lymphohemopoietic neoplasms was found in both genders (RR = 1.7, 95% CI: 1.2, 2.5). Hodgkin's disease risk was elevated in the first 10-year observation period (RR = 4.9, 95% CI: 1.5, 16.4), whereas the highest increase for 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) occurred after 15 years. No soft tissue sarcoma cases were found in these zones (0.8 expected). An overall increase in diabetes was reported, notably among women (RR = 2.4, 95% CI: 1.2, 4.6). Chronic circulatory and respiratory diseases were moderately increased, suggesting a link with accident-related stressors and chemical exposure. Results support evaluation of dioxin as carcinogenic to humans and corroborate the hypotheses of its association with other health outcomes, including cardiovascular- and endocrine-related effects. *Am J Epidemiol* 2001;153:1031–44.

accidents, occupational; carcinogens, environmental; chemical industry; dioxins; mortality

Editor's note: An invited commentary on this paper appears on page 1045, and the authors' response is on page 1048.

The health effects associated with exposure to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD, or simply "dioxin") have not yet been fully characterized. Uncertainty exists about whether the extremely potent toxicity of TCDD in experimental animals, including carcinogenicity (1, 2), also applies to humans (3). In addition, the possible risk, if any, to human health of widespread, low-level dioxin contamination of the environment has still to be assessed (4).

One relevant data source that bridges these gaps in knowledge is the study of the Seveso, Italy, industrial accident (5, 6) that exposed several thousand people to substantial quantities of TCDD. The accident took place in the summer of 1976, when the temperature and pressure surged in an improperly maintained reaction vessel in the trichlorophenol production department of a chemical plant near the town of Seveso, 25 km north of Milan; given the concomitant failure of a safety device, the contents of the reactor were vented directly into the atmosphere (7).

The level and extent of the environmental contamination were documented by dioxin soil measurements in a wide area along the direction of the prevailing winds. Three contamination zones were delimited. The most heavily contaminated was called zone A, zone B was its natural continuation along the fallout path of the chemical cloud, and zone R, with

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Abbreviations: CI, confidence interval; RR, rate ratio; TCDD, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin.

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low-level and patchy contamination, represented a gray, circular strip between the highly contaminated zones and the surrounding territory (8).

The earliest accident-related health effect was chloracne in children who were outdoors and in the path of the toxic cloud (9). In the following years, under the supervision of an international steering committee, other health outcomes possibly linked to TCDD exposure were investigated, including spontaneous abortions (10), cytogenetic abnormalities (11, 12), congenital malformations (13, 14), impaired liver function and lipid metabolism (15, 16), and immunologic (17) and neurologic (18, 19) impairment. In 1984, the committee concluded their work and stated that the only ascertained effect of dioxin exposure was chloracne but that long-term studies were needed (20).

The mortality and the cancer incidence studies we designed were implemented in 1985. Results for mortality (1976–1986 and 1976–1991 (21–23)) and for cancer incidence (1977–1986 (24)) have been published. In this paper, we report on extension of the mortality study to the end of 1996. Although other populations with known TCDD exposure have been investigated (e.g., chemical workers (25–28), pesticide manufacturers and applicators (29, 30), and Vietnam War veterans (31)), Seveso remains unique because of several characteristics, including residents' exposure to "pure" TCDD and the presence of persons of both genders and all ages in the exposed populations.

MATERIALS AND METHODS

Methodological aspects of the mortality study have been reported in detail previously (7, 21, 32). The three contaminated zones (A, B, and R) covered parts of the territory that included two health districts encompassing 11 municipalities, with a total population of nearly 300,000. The study population was comprised of all people, both sexes and all ages, residing in on the date of the accident or entering in the 10-year period after the accident, the districts in any of the study zones or in the surrounding noncontaminated area. The population living in this latter territory was adopted as the reference group. In addition to geographic proximity, all available indicators documented the close comparability of the reference population with the exposed one in terms of environmental, social, educational, cultural, and occupational characteristics. Its size (some 250,000 subjects) was also deemed reasonably large. Nevertheless, mortality rates for the reference population were compared with those for the entire Region of Lombardy (nearly 9 million inhabitants) to evaluate their stability (21).

Exposure classification was based on the address of the residence on the date of the accident or when the person first entered the area, if later. The extent and level of soil contamination in zones A and B were measured systematically by using a tight sampling grid, whereas analyses in zone R were scanty (7). Biologic data were also available. TCDD blood levels were measured in small plasma samples, stored immediately after the accident, from subjects living in zones A, B, and R who were reportedly exposed to high levels of dioxin (33) and in the plasma of subjects randomly selected from zone A, zone B, and the reference area who were enrolled in a current molecular epidemiology study (34). Table 1 shows exposure information by zone.

TABLE 1. TCDD* concentrations in soil and in the blood of selected residents in the zones contaminated after the Seveso, Italy, industrial accident in 1976

Study area	Soil concentration†		Lipid-adjusted plasma concentration	
	Minimum	Maximum	No. of subjects	Median‡
Zone A	15.5	580.4	296§	447.0
			7¶	73.3
Zone B	1.7	4.3	80§	94.0
			51¶	12.4
Zone R	0.9	1.4	48§	48.0
Reference zone	NA*	NA	52¶	5.5

* TCDD, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin; NA, not available.
† Mean value (μg/m²); reference 8.
‡ Parts per trillion.
§ Samples collected in 1976–1977; reference 33.
¶ Samples collected in 1993–1994; reference 34.

The search for vital status, and cause-of-death ascertainment for the deceased subjects, was performed on an individual basis by contacting the vital statistics offices of the 11 study towns and of thousands of municipalities throughout the country to reach those subjects who had migrated. The per-year migration rate from the 11 municipalities to other locations within Lombardy was 5.0 per 1,000 in the exposed and 11.5 per 1,000 in the reference population; the migration rates outside Lombardy were 6.2 and 6.3 per 1,000, respectively. Exposed and reference subjects were followed concurrently, as a unique cohort, by using the same means, methods, and criteria. Exposure status was ignored in this research phase.

In successive follow-ups, causes of death were coded by the same trained nosologist, who was unaware of the exposure status of the subjects. Coding accuracy and consistency were evaluated on the basis of criteria from the Italian Central Statistics Institute, located in Rome.

We also compared death rates in the pre- and postaccident periods to identify unusual risks possibly present even before the accident occurred in the exposed population and to highlight possible changes in the background death registration system in the area (35). Each study subject contributed person-time of observation from the date of the accident (July 10, 1976) or, if later, the date of first residence in the study area through the end of follow-up (December 31, 1996) or through the date of death, if earlier. All person-years of follow-up for each person were attributed to the zone of residence on the accident date or later, when he or she first moved to the area. Person-time was computed by stratifying on age, zone, residence on the day of the accident, gender, calendar time, duration of residence, and number of years elapsed since first exposure. For each contaminated zone, age-, gender-, and period-adjusted rate ratios and approximate 95 percent confidence intervals were estimated for each

cause of death by means of Poisson regression techniques 36) in Stata software (37). The expected number of deaths was obtained by multiplying the fitted reference rates (specific period, age, and gender) by the number of stratum-specific person-years in the exposed zones. Data also were analyzed separately by number of years since first exposure ("latency"), gender, age category, calendar time, duration of residence (surrogate for duration of exposure), and residence on the day of the accident.

RESULTS

Information on the study population at the end of follow-up is shown in table 2. Tracing was virtually complete for those in the 11 study municipalities and was approximately 99 percent for those who had moved out of the area.

The person-years accrued during the 20.5-year follow-up period are reported in table 3. The 5-year (1992–1996) extension of the follow-up added to the entire study population, including the reference area, 1,225,644 person-years of observation and 9,570 deaths (26 in zone A, 180 in zone B, 1,203 in zone R, and 8,161 in the reference zone). Distribution by age and gender was fairly uniform across zones, as expected. People in zone B were slightly younger.

Table 4 presents detailed results for the population living in high-exposure zones A and B, including observed and expected numbers of deaths; age-, gender-, and calendar-period-adjusted point estimates of the rate ratios; and 95 percent confidence intervals for the main causes of death in each zone and in both zones combined. All-cause and all-cancer mortality was similar to that in the reference population. Deaths from rectal cancer were elevated, with a nearly twofold increase in zone B. No liver cancer deaths were observed in zone A. An excess of "other" digestive cancer was found in both zones; however, it did not reach statistical significance. Lung cancer also was moderately in excess, but the increase was statistically nonsignificant. One melanoma death in zone A yielded a remarkable increase in the rate ratio estimate. Among lymphohemopoietic neoplasms, two deaths from non-Hodgkin's lymphoma were observed in zone A, which represented a higher-than-threefold excess above expectations. In zone B, nearly twice as many as expected lymphohemopoietic neoplasms occurred, a significant increase that in particular included Hodgkin's disease, multiple myeloma, and myeloid leukemia. No deaths from soft tissue sarcoma were observed in zones A or B (0.1 and 0.7 expected, respectively).

Regarding nonmalignant causes of death, hypertension was nonsignificantly in excess in zone A. Chronic obstructive pulmonary disease increased significantly in zone A and less evidently in zone B. The zone B population also exhibited moderate increases in diabetes and chronic ischemic heart disease.

Zone A results by latency are available on the *Journal* website (www.jhsph.edu/Publications/JEPI/bertazzi.htm (table 1)). Increased mortality was found in the 5–9-year

TABLE 2. Sample size and completeness of follow-up of the study population in the dioxin-contaminated and reference areas, Seveso, Italy, 1976–1996

Study area	No. of subjects		No. of deaths	Not traced	
	Female	Male		No.	%
Zone A	414	390	96	5	0.6
Zone B	2,924	3,017	649	30	0.5
Zone R	19,424	19,200	4,937	202	0.5
Reference zone	118,775	113,970	32,128	1,616	0.7

TABLE 3. Distribution of person-years of observation, by exposure zone, gender, age, and latency, for the population exposed to dioxin after the Seveso, Italy, industrial accident, 1976–1996

Variable	Exposure zone			Reference zone	Total
	A	B	R		
Gender					
Male	7,270	55,648	349,480	2,032,871	2,445,269
Female	7,866	54,861	358,797	2,150,048	2,571,572
Age (years)					
<20	4,946	40,452	243,325	1,354,246	1,642,969
20–29	2,524	20,100	121,936	707,941	852,500
30–39	2,354	17,264	109,845	668,879	798,342
40–49	2,354	14,343	96,151	577,324	690,172
50–59	1,720	10,045	71,259	432,911	515,936
60–69	864	5,487	43,081	279,235	328,667
70–79	321	2,394	19,243	135,726	157,685
≥80	53	424	3,436	26,657	30,569
Latency (years)					
≤4	3,962	29,323	189,774	1,142,625	1,365,684
5–9	3,834	28,562	183,806	1,102,945	1,319,147
10–14	3,634	26,940	171,669	1,008,725	1,210,968
15–20	3,706	25,684	163,028	928,532	1,120,950

TABLE 4. Observed and expected numbers of deaths, rate ratios,* and 95% confidence intervals for selected causes of death in high-exposure zones A and B for the population exposed to dioxin after the Seveso, Italy, industrial accident, 1976–1996

Cause of death (ICD-9† codes)	Zone A				Zone B				Total			
	Obs†	Exp†	RR†	95% CI†	Obs	Exp	RR	95% CI	Obs	Exp	RR	95% CI
All causes (001–999)	96	91.9	1.0	0.9, 1.3	649	654.6	1.0	0.9, 1.1	745	746.5	1.0	0.9, 1.1
All cancers (140–208)	27	31.4	0.9	0.6, 1.3	222	208.9	1.1	0.9, 1.2	249	240.2	1.0	0.9, 1.2
Digestive (150–159)	9	12.0	0.7	0.4, 1.4	75	81.7	0.9	0.7, 1.2	84	93.7	0.9	0.7, 1.1
Stomach (151)	3	3.6	0.8	0.3, 2.6	24	24.9	1.0	0.6, 1.4	27	28.5	0.9	0.6, 1.4
Colon (153)	2	2.3	0.9	0.2, 3.4	13	15.8	0.8	0.5, 1.4	15	18.1	0.8	0.5, 1.4
Rectum (154)	1	0.8	1.2	0.2, 8.6	11	5.7	1.9	1.1, 3.5	12	6.5	1.8	1.0, 3.3
Hepatobiliary (155–156)	0	2.6			13	17.3	0.8	0.4, 1.3	13	19.9	0.7	0.4, 1.1
Liver (155)	0	2.2			12	14.7	0.8	0.5, 1.4	12	16.9	0.7	0.4, 1.3
Pancreas (157)	1	1.2	0.8	0.1, 5.9	4	8.1	0.5	0.2, 1.3	5	9.3	0.5	0.2, 1.3
Other digestive (159)	2	0.6	3.2	0.8, 12.9	7	4.3	1.6	0.8, 3.5	9	4.9	1.8	0.9, 3.6
Respiratory (160–165)	9	7.7	1.2	0.6, 2.3	60	49.9	1.2	0.9, 1.6	69	57.6	1.2	0.9, 1.5
Lung (162)	9	6.7	1.3	0.7, 2.6	52	43.4	1.2	0.9, 1.6	61	50.1	1.2	0.9, 1.6
Soft tissue sarcoma (171)	0	0.1			0	0.7			0	0.8		
Melanoma (172)	1	0.2	4.2	0.6, 30.2	2	1.6	1.3	0.3, 5.2	3	1.8	1.7	0.5, 5.3
Breast (174)	2	2.6	0.8	0.2, 3.1	12	16.5	0.7	0.4, 1.3	14	19.1	0.7	0.4, 1.2
Genitourinary (179–189)	2	3.7	0.5	0.1, 2.2	24	24.8	1.0	0.6, 1.4	26	28.5	0.9	0.6, 1.3
Uterus (179–182)	0	0.6			2	3.8	0.5	0.1, 2.1	2	4.4	0.5	0.1, 1.8
Ovary (183)	1	0.6	1.6	0.2, 11.2	2	3.9	0.5	0.1, 2.0	3	4.6	0.7	0.2, 2.0
Prostate (185)	0	0.8			8	6.7	1.2	0.6, 2.4	8	7.5	1.1	0.5, 2.2
Bladder (188)	1	0.8	1.3	0.2, 9.4	5	5.5	0.9	0.4, 2.2	6	6.2	1.0	0.4, 2.2
Kidney (189)	0	0.7			6	4.6	1.3	0.6, 2.9	6	5.3	1.1	0.5, 2.6
Brain (191)	0	0.5			4	3.5	1.2	0.4, 3.1	4	4.0	1.0	0.4, 2.7
Lymphatic and hemopoietic (200–208)	2	2.1	1.0	0.2, 3.9	26	14.0	1.9	1.3, 2.7	28	16.1	1.7	1.2, 2.5
Hodgkin's disease (201)	0	0.2			4	1.1	3.5	1.3, 9.8	4	1.3	3.1	1.1, 8.6
Non-Hodgkin's lymphoma (200, 202)	2	0.6	3.3	0.8, 13.1	5	4.0	1.2	0.5, 3.0	7	4.7	1.5	0.7, 3.2
Multiple myeloma (203)	0	0.4			5	2.5	2.0	0.8, 4.8	5	2.9	1.7	0.7, 4.2
Leukemia (204–208)	0	0.9			12	6.4	1.9	1.0, 3.3	12	7.4	1.6	0.9, 2.9
Lymphatic leukemia (204)	0	0.3			2	1.8	1.1	0.3, 4.4	2	2.1	1.0	0.2, 3.9
Myeloid leukemia (205)	0	0.4			6	2.5	2.4	1.0, 5.4	6	2.9	2.1	0.9, 4.7
Leukemia, unspecified (208)	0	0.3			4	2.0	2.0	0.7, 5.4	4	2.3	1.8	0.6, 4.8
Diabetes mellitus (250)	2	2.4	0.8	0.2, 3.3	24	16.9	1.4	0.9, 2.1	26	19.3	1.3	0.9, 2.0
All circulatory diseases (390–459)	37	33.3	1.1	0.8, 1.5	228	242.7	0.9	0.8, 1.1	265	276.0	1.0	0.8, 1.1
Chronic rheumatic heart disease (393–398)	3	0.4	7.0	2.2, 21.9	0	2.8			3	3.2	0.9	0.3, 3.0
Hypertension (400–405)	4	1.8	2.3	0.8, 6.1	5	12.6	0.4	0.2, 1.0	9	14.3	0.6	0.3, 1.2
Ischemic heart disease (410–414)	10	12.4	0.8	0.4, 1.5	87	89.5	1.0	0.8, 1.2	97	102.0	1.0	0.8, 1.2
Myocardial infarction (410)	5	7.9	0.6	0.3, 1.5	45	54.3	0.8	0.6, 1.1	50	62.3	0.8	0.6, 1.1
Chronic ischemic heart disease (412, 414)	5	4.6	1.1	0.5, 2.6	41	34.8	1.2	0.9, 1.6	46	39.4	1.2	0.9, 1.6
Cerebrovascular disease (430–438)	8	9.8	0.8	0.4, 1.6	80	71.0	1.1	0.9, 1.4	88	80.7	1.1	0.9, 1.3
Respiratory disease (460–519)	9	4.8	1.9	1.0, 3.6	35	37.7	0.9	0.7, 1.3	44	42.5	1.0	0.8, 1.4
Chronic obstructive pulmonary disease (490–493)	7	2.1	3.3	1.6, 6.9	22	16.9	1.3	0.9, 2.0	29	19.1	1.5	1.1, 2.2
Digestive disease (520–579)	5	5.8	0.9	0.4, 2.1	38	39.3	1.0	0.7, 1.3	43	45.2	1.0	0.7, 1.3
Cirrhosis of the liver (571)	2	3.9	0.5	0.1, 2.1	19	25.0	0.8	0.5, 1.2	21	28.8	0.7	0.5, 1.1
Unknown (799.9)	3	1.6	1.9	0.6, 5.9	11	11.2	1.0	0.5, 1.8	14	12.8	1.1	0.6, 1.9
Accidents (800–999)	7	5.7	1.2	0.6, 2.6	45	41.5	1.1	0.8, 1.5	52	47.1	1.1	0.8, 1.5

* Adjusted for age, calendar period, and gender.

† ICD-9, *International Classification of Diseases and Causes of Death*, Ninth Revision; Obs, observed no.; Exp, expected no.; RR, rate ratio; CI, confidence interval.

period only, sustained mainly by suggestive increases in a number of cancer types and sites (including digestive, lung, melanoma, and bladder) and by deaths due to circulatory disease. Lung cancer and non-Hodgkin's lymphoma increased steeply after 15 years. Increased circulatory disease mortality characterized the first 10-year period, with the exception of

hypertension, which peaked between 10 and 15 years. Mortality from respiratory disease, predominantly chronic obstructive pulmonary disease, was elevated immediately after the incident and in the latest observation period.

In zone B (table 2, website), neither all-cause nor all-cancer mortality notably departed from expectations throughout the study period. The increased rectal cancer mortality failed to exhibit a consistent latency-related pattern. Other digestive cancers and lung cancer increased in the 5–9-year period, as they did in zone A. Two melanoma deaths in the 15–20-year period yielded a remarkably high risk ratio estimate. Lymphatic and hemopoietic neoplasms were fairly consistently elevated throughout the observation period. Hodgkin's disease increased in the early period, significantly so in the 5–9-year period, whereas non-Hodgkin's lymphoma showed a later, modest increase. The risk of multiple myeloma was increased in the categories 5–9 and 10–14 years. The numbers of deaths from leukemia were steadily above expectations, and the myeloid leukemia increase was highest in the longest latency period. Among nonmalignant causes, the increase in diabetes was most evident immediately after the accident and for chronic ischemic heart disease and chronic obstructive pulmonary disease in the 5–9-year period.

The results by latency for zones A and B combined (table 5) failed to reveal clear, definite, time-related mortality patterns. Only suggestive was the trend for lymphatic and hemopoietic neoplasms as a whole. As to specific causes, the rate of Hodgkin's disease was high in the early postaccident period: the rate ratio estimate for 0–10 years since first exposure was 4.9 (95 percent confidence interval (CI): 1.5, 16.4). The rates for non-Hodgkin's lymphoma and myeloid leukemia were instead high in the longest latency period and for multiple myeloma in the period between 5 and 15 years. The moderately increased diabetes mortality exhibited no regular time-related pattern. Rates of chronic ischemic heart disease and chronic obstructive pulmonary disease were high in the 5–9-year latency period.

This study provided an almost unique opportunity to examine the health experience of a large female population exposed to dioxin. Therefore, we considered it useful to report results separately by gender.

Among females in zone A (table 3, website), mortality from all causes and all cancers was as expected, with the exception of the 5–9-year latency period, which showed an excess of colon and other digestive cancers and of melanoma. Stomach cancer was increased in the second decade. The single observed case of non-Hodgkin's lymphoma occurred in the 15–20-year period. The risk ratios for hypertension and for chronic obstructive pulmonary disease were elevated. Definite patterns or trends could not be observed, but the population size was small and few events were observed. Among males in zone A (table 4, website), cancer mortality was slightly elevated after 15 years. Lung cancer showed a clear, significant increase, whereas the increases for rectal cancer and non-Hodgkin's lymphoma were significant but only suggestive. The highest mortality pattern from circulatory disease was found in the first postaccident periods; respiratory disease was elevated in the earliest period and after 15 years.

Regarding results by years since first exposure for females in zone B (table 5, website), all-cause and all-cancer mortality overall was as expected. In the 10–14-year period, digestive cancer mortality was elevated, and stomach and liver cancer showed statistically significant increases. Twelve cases of lymphatic and hemopoietic neoplasms made up a twofold statistically significant excess, with a suggestively increasing pattern by latency. The increase involved Hodgkin's disease, non-Hodgkin's lymphoma (significantly increased, as in zone A, in the latest latency period), and multiple myeloma. An excess of leukemia deaths was found, although not significantly so, 15 or more years after first exposure. With respect to nonmalignant causes, diabetes showed an excess that was significant after 15 years, and chronic obstructive pulmonary disease exhibited a pattern of moderately increased mortality with a peak between 10 and 15 years.

In total, males in zone B had moderately increased mortality from cancer causes (table 6, website). Rectal cancer increased significantly, but there was no definite latency pattern. In contrast to women, liver cancer was not increased, five "other" digestive cancer deaths represented a borderline significant excess, and lung cancer exhibited a slight, persistent elevation 5 or more years after first exposure. Lymphatic and hemopoietic neoplasms showed a nearly twofold borderline significant increase: Hodgkin's disease and leukemia mainly contributed to this finding. Most prominent was the increase in myeloid leukemia in the longest latency categories. No increased mortality from diabetes was noted, and no major departures from expectations were found for other nonmalignant causes of death.

When we examined the pooled experience of the highly exposed Seveso population (zone A plus zone B), no obvious departure of all-cause and all-cancer mortality from reference population rates was found for females (table 6). Ten to 14 years after the accident, mortality from digestive cancer (stomach and liver in particular) was borderline significantly increased. Lymphatic and hemopoietic neoplasms were significantly increased, with monotonically increasing risk ratio estimates. The highest risk ratio values were found for Hodgkin's disease, multiple myeloma, and non-Hodgkin's lymphoma in the latest latency category. Leukemia mortality was not elevated. Among nonmalignant diseases, the rate of diabetes was high, with a significant increase in the longest latency period, whereas the elevated mortality from chronic obstructive pulmonary disease peaked in the 10–15-year period. Results for males (table 7) showed an elevated cancer mortality for three sites. Rectal cancer was significantly increased, without a definite latency pattern. Lung cancer showed a moderate increase, which was borderline significant in the second and latest latency periods. The increased mortality from lymphatic and hemopoietic neoplasms was statistically borderline significant, but no particular trend or pattern was evident. The increase for leukemia, most notably myeloid leukemia, was significant. Among nonmalignant causes, an excess of chronic ischemic heart disease and chronic obstructive respiratory disease was found in the 5–9-year category.

In zone R, for none of the cancer sites considered was a mortality rate notably different from that expected found.

TABLE 5. Observed and expected numbers of deaths, rate ratios,* and 95% confidence intervals, by cause of death and years since first exposure (latency), for the population in zones A and B exposed to dioxin after the Seveso, Italy, industrial accident, 1976–1996

Cause of death (ICD-9† codes)	No. of years since first exposure															
	0–4				5–9				10–14				15–20			
	Obs†	Exp†	RR†	95% CI†	Obs	Exp	RR	95% CI	Obs	Exp	RR	95% CI	Obs	Exp	RR	95% CI
All causes (001–999)	170	181.9	0.9	0.8, 1.1	187	177.2	1.1	0.9, 1.2	171	178.0	1.0	0.8, 1.1	217	209.7	1.0	0.9, 1.2
All cancers (140–208)	38	48.2	0.8	0.6, 1.1	63	55.8	1.1	0.9, 1.4	65	63.3	1.0	0.8, 1.3	83	73.9	1.1	0.9, 1.4
Digestive (150–159)	10	19.2	0.5	0.3, 1.0	22	21.1	1.0	0.7, 1.6	26	24.7	1.1	0.7, 1.6	26	29.5	0.9	0.6, 1.3
Stomach (151)	4	6.8	0.6	0.2, 1.6	4	7.1	0.6	0.2, 1.5	10	7.5	1.3	0.7, 2.5	9	7.4	1.2	0.6, 2.4
Colon (153)	0	3.9			5	4.1	1.2	0.5, 2.9	5	4.3	1.2	0.5, 2.8	5	6.0	0.8	0.3, 2.0
Rectum (154)	3	1.5	2.0	0.6, 6.5	1	1.3	0.7	0.1, 5.4	4	1.8	2.2	0.8, 6.1	4	2.0	2.0	0.7, 5.5
Hepatobiliary (155–156)	1	3.0	0.3	0.05, 2.4	5	3.7	1.3	0.6, 3.3	4	6.1	0.7	0.2, 1.8	3	7.0	0.4	0.1, 1.3
Liver (155)	1	2.4	0.4	0.1, 3.0	4	3.2	1.3	0.5, 3.4	4	5.2	0.8	0.3, 2.1	3	6.0	0.5	0.2, 1.6
Pancreas (157)	1	1.7	0.6	0.1, 4.3	2	1.9	1.1	0.3, 4.3	1	2.3	0.4	0.1, 3.1	1	3.4	0.3	0.04, 2.1
Other digestive (159)	1	0.6	1.6	0.2, 11.7	4	1.2	3.4	1.2, 9.3	1	1.3	0.8	0.1, 5.7	3	1.9	1.6	0.5, 5.0
Respiratory (160–165)	10	11.7	0.9	0.5, 1.6	19	13.0	1.5	0.9, 2.3	17	16.0	1.1	0.7, 1.7	23	16.7	1.4	0.9, 2.1
Lung (162)	9	10.0	0.9	0.5, 1.7	16	10.8	1.5	0.9, 2.4	15	14.4	1.0	0.6, 1.7	21	14.9	1.4	0.9, 2.2
Melanoma (172)	0	0.3			1	0.4	2.7	0.4, 20.4	0	0.6			2	0.7	3.1	0.7, 13.0
Breast (174)	3	3.4	0.9	0.3, 2.8	3	5.0	0.6	0.2, 1.9	3	4.5	0.7	0.2, 2.1	5	6.2	0.8	0.3, 2.0
Genitourinary (179–189)	4	5.6	0.7	0.3, 1.9	7	6.9	1.0	0.5, 2.2	5	7.6	0.7	0.3, 1.6	10	8.4	1.2	0.6, 2.2
Bladder (188)	1	1.2	0.8	0.1, 6.1	3	1.3	2.3	0.7, 7.2	0	1.8			2	1.8	1.1	0.3, 4.5
Kidney (189)	0	0.9			0	1.3			3	1.5	2.0	0.6, 6.5	3	1.7	1.8	0.6, 5.7
Brain (191)	0	0.5			2	1.2	1.7	0.4, 7.0	2	1.0	1.0	0.5, 8.1	0	1.3		
Lymphatic and hemopoietic (200–208)	5	3.5	1.4	0.6, 3.5	6	3.6	1.7	0.7, 3.8	8	4.0	2.0	1.0, 4.1	9	5.2	1.7	0.9, 3.4
Hodgkin's disease (201)	1	0.3	3.4	0.4, 26.0	2	0.3	6.1	1.4, 27.5	1	0.4	2.6	0.3, 19.6	0	0.3		
Non-Hodgkin's lymphoma (200, 202)	0	0.6			0	1.0			2	1.4	1.5	0.4, 6.0	5	1.8	2.8	1.1, 7.0
Multiple myeloma (203)	0	0.6			2	0.5	3.8	0.9, 16.2	3	0.5	5.5	1.7, 18.4	0	1.2		
Leukemia (204–208)	4	2.0	2.0	0.7, 5.5	2	1.8	1.1	0.3, 4.6	2	1.7	1.2	0.3, 4.8	4	1.9	2.1	0.8, 5.8
Lymphatic leukemia (204)	1	0.4	2.5	0.3, 18.8	1	0.4	2.6	0.3, 19.8	0	0.6			0	0.7		
Myeloid leukemia (205)	1	0.7	1.5	0.2, 11.3	0	0.7			2	0.8	2.6	0.6, 10.9	3	0.8	3.8	1.1, 12.5
Leukemia, unspecified (208)	2	0.9	2.1	0.5, 8.8	1	0.6	1.6	0.2, 11.5	0	0.3			1	0.3	3.0	0.4, 23.1
Diabetes mellitus (250)	7	3.6	2.0	0.9, 4.2	3	4.7	0.6	0.2, 2.0	8	5.0	1.6	0.8, 3.2	8	5.8	1.4	0.7, 2.8
All circulatory diseases (390–459)	71	70.5	1.0	0.8, 1.3	81	69.7	1.2	0.9, 1.4	52	64.0	0.8	0.6, 1.1	61	71.9	0.8	0.7, 1.1
Hypertension (400–405)	3	3.7	0.8	0.3, 2.5	1	3.3	0.3	0.04, 2.1	3	3.5	0.9	0.3, 2.7	2	3.7	0.5	0.1, 2.2
Ischemic heart disease (410–414)	21	25.0	0.8	0.5, 1.3	30	27.7	1.1	0.8, 1.6	20	23.9	0.8	0.5, 1.3	26	25.2	1.0	0.7, 1.5
Myocardial infarction (410)	11	15.4	0.7	0.4, 1.3	10	16.0	0.6	0.3, 1.2	11	15.3	0.7	0.4, 1.3	18	15.3	1.2	0.7, 1.9
Chronic ischemic heart disease (412, 414)	10	9.4	1.1	0.6, 2.0	19	11.5	1.6	1.0, 2.6	9	8.5	1.1	0.5, 2.0	8	9.9	0.8	0.4, 1.6
Cerebrovascular disease (430–438)	25	21.7	1.2	0.8, 1.7	25	19.7	1.3	0.9, 1.9	18	19.2	0.9	0.6, 1.5	20	20.4	1.0	0.6, 1.5

Respiratory disease (460–519)	11	11.2	1.0	0.5, 1.8	9	10.3	0.9	0.5, 1.7	8	9.8	0.8	0.4, 1.6	16	11.1	1.4	0.9, 2.4
Chronic obstructive pulmonary disease (490–493)	7	4.7	1.5	0.7, 3.1	8	4.1	2.0	1.0, 4.0	6	5.0	1.2	0.5, 2.7	8	5.2	1.5	0.8, 3.1
Digestive disease (520–579)	8	13.5	0.6	0.3, 1.2	13	11.7	1.1	0.6, 1.9	10	9.6	1.0	0.6, 1.9	12	10.3	1.2	0.7, 2.1
Cirrhosis of the liver (571)	5	8.9	0.6	0.2, 1.4	6	8.0	0.8	0.3, 1.7	5	6.3	0.8	0.3, 1.9	5	5.7	0.9	0.4, 2.1
Accidents (800–999)	13	12.3	1.1	0.6, 1.8	10	12.2	0.8	0.4, 1.5	14	10.8	1.3	0.8, 2.2	15	11.7	1.3	0.8, 2.1

* Adjusted for age, calendar period, and gender.
† ICD-9, *International Classification of Diseases and Causes of Death*, Ninth Revision; Obs, observed no.; Exp, expected no.; RR, rate ratio; CI, confidence interval.

Given the large population size, 4 vs. 4.8 expected soft tissue sarcoma deaths were observed. Mortality from diabetes (observed no. = 168, expected no. = 132.2; rate ratio (RR) = 1.3, 95 percent CI: 1.1, 1.5), hypertension (observed no. = 130, expected no. = 99.2; RR = 1.3, 95 percent CI: 1.1, 1.6), and chronic ischemic heart disease (observed no. = 328, expected no. = 266.5; RR = 1.2, 95 percent CI: 1.1, 1.4) increased moderately.

Analyses by zone according to length of stay yielded results very similar to those according to number of years since first exposure; the great majority of study subjects (90 percent in zone A , 81 percent in zone B, 82 percent in zone R, and 78 percent of the reference population) resided in the area at the time of the accident, and the migration rate was limited. Consistently, analysis by residence on the date of the accident marginally influenced the risk ratio estimates.

A special group within the cohort, composed of 182 persons (57 in zone A, 11 in zone B, 69 in zone R, and 45 in the reference area), was diagnosed with chloracne after the accident. All were traced; two had died by the time of this follow-up extension, one zone A resident from myocarditis and one zone R resident from suicide.

DISCUSSION

Extension of follow-up of the population exposed to dioxin after the Seveso, Italy, industrial accident failed to reveal an overall increase in all-cause and all-cancer mortality. However, it suggested that those residents living in the highly contaminated territory were at increased risk from some causes.

When we interpreted the results, major bias and confounding phenomena could be excluded. Follow-up was virtually complete. The reference population was local, closely similar to the index population, and large enough to ensure that the adopted reference rates were stable. Exceptions were pinpointed through comparison with the Region of Lombardy population rates and included other and not specified leukemia (high reference rates for males), digestive diseases (high reference rates for females), and brain cancer (low reference rates for males) (21). Tracing of vital status and coding of causes of death were uniform and were blinded for exposure status of the subjects. The exposed and referent populations belonged to the same health districts and had similar access to the same diagnostic and therapeutic services (from family physicians to hospitals). Therefore, no differential death registration pattern between exposed and referent populations should have occurred. Comparison of pre- and postaccident rates disclosed elevated risks in the exposed population, before the accident, for brain cancer (males and females) and leukemia (females) (35). The ecologic classification of exposure status based on soil levels was not refuted by classification based on available blood dioxin measurements. Blood measurements also strengthened confidence in the nonexposure status of the reference population; their estimated average blood concentration corresponded to background values measured in industrial areas (38).

Analyses by exposure zone, time since first exposure, and gender disclosed unusual mortality patterns for some

TABLE 6. Observed and expected numbers of deaths, rate ratios,* and 95% confidence intervals, by cause of death and years since first exposure (latency), for the female population in zones A and B exposed to dioxin after the Seveso, Italy, industrial accident, 1976–1996

Cause of death (ICD-9† codes)	No. of years since first exposure																			
	0–4				5–9				10–14				15–20				Total			
	Obs†	Exp†	RR†	95% CI†	Obs	Exp	RR	95% CI	Obs	Exp	RR	95% CI	Obs	Exp	RR	95% CI	Obs	Exp	RR	95% CI
All causes (001–999)	67	72.2	0.9	0.7, 1.2	74	72.1	1.0	0.8, 1.3	76	71.8	1.1	0.8, 1.3	90	92.4	1.0	0.8, 1.2	307	308.5	1.0	0.9, 1.1
All cancers (140–208)	10	18.1	0.6	0.3, 1.0	20	21.1	0.9	0.6, 1.5	28	22.1	1.3	0.9, 1.8	25	29.8	0.8	0.6, 1.2	83	90.8	0.9	0.7, 1.1
Digestive (150–159)	3	7.7	0.4	0.1, 1.2	9	8.0	1.1	0.6, 2.2	13	8.7	1.5	0.9, 2.6	6	12.0	0.5	0.2, 1.1	31	36.0	0.9	0.6, 1.2
Stomach (151)	0	2.5			1	2.4	0.4	0.1, 3.0	7	2.7	2.6	1.2, 5.5	3	3.0	1.0	0.3, 3.1	11	10.6	1.0	0.6, 1.9
Colon (153)	0	1.7			3	2.0	1.5	0.5, 4.7	2	1.8	1.1	0.3, 4.6	0	2.9			5	8.3	0.6	0.2, 1.4
Rectum (154)	1	0.7	1.4	0.2, 10.5	0	0.4			1	0.7	1.4	0.2, 10.4	1	0.9	1.1	0.1, 7.7	3	2.7	1.1	0.4, 3.5
Hepatobiliary (155–156)	1	1.3	0.8	0.1, 5.7	2	1.5	1.3	0.3, 5.4	3	1.6	1.8	0.6, 5.8	1	2.3	0.4	0.1, 3.1	7	6.7	1.0	0.5, 2.2
Liver (155)	1	0.8	1.2	0.2, 8.7	1	1.2	0.9	0.1, 6.2	3	1.1	2.8	0.9, 8.9	1	1.6	0.6	0.1, 4.5	6	4.7	1.3	0.6, 2.9
Pancreas (157)	0	0.5			1	0.5	2.0	0.3, 15.0	0	0.9			0	1.6			1	3.5	0.3	0.03, 2.0
Other digestive (159)	1	0.4	2.2	0.3, 16.6	2	0.7	2.8	0.7, 11.7	0	0.6			1	0.9	1.1	0.1, 7.9	4	2.6	1.5	0.6, 4.1
Respiratory (160–165)	0	1.4			1	1.3	0.8	0.1, 5.6	1	1.9	0.5	0.1, 3.7	3	2.6	1.2	0.4, 3.7	5	7.2	0.7	0.3, 1.7
Lung (162)	0	1.1			1	1.1	0.9	0.1, 6.9	1	1.8	0.6	0.1, 4.1	2	2.2	0.9	0.2, 3.8	4	6.2	0.6	0.2, 1.7
Melanoma (172)	0	0.2			1	0.3	3.5	0.5, 27.1	0	0.3			1	0.4	2.6	0.3, 20.0	2	1.1	1.8	0.4, 7.3
Breast (174)	3	3.4	0.9	0.3, 2.8	3	5.0	0.6	0.2, 1.9	3	4.5	0.7	0.2, 2.1	5	6.1	0.8	0.3, 2.0	14	19.0	0.7	0.4, 1.3
Genitourinary (179–189)	0	2.6			2	3.1	0.6	0.2, 2.6	4	3.1	1.3	0.5, 3.5	3	3.5	0.9	0.3, 2.7	9	12.2	0.7	0.4, 1.4
Uterus (179–182)	0	1.1			1	1.1	0.9	0.1, 6.4	0	1.1			1	1.0	1.0	0.1, 7.4	2	4.4	0.5	0.1, 1.9
Ovary (183)	0	0.8			0	1.2			1	1.1	0.9	0.1, 6.9	2	1.5	1.3	0.3, 5.4	3	4.6	0.7	0.2, 2.0
Kidney (189)	0	0.3			0	0.4			3	0.5	6.1	1.8, 20.4	0	0.5			3	1.6	1.8	0.6, 5.8
Brain (191)	0	0.3			1	0.4	2.7	0.4, 20.1	2	0.4	4.9	1.1, 21.3	0	0.5			3	1.6	1.9	0.6, 6.0
Lymphatic and hemopoietic (200–208)	1	1.4	0.7	0.1, 5.2	2	1.6	1.3	0.3, 5.3	4	1.8	2.3	0.8, 6.2	6	2.4	2.5	1.1, 5.7	13	7.1	1.8	1.1, 3.2
Hodgkin's disease (201)	0	0.1			1	0.1	8.5	0.9, 76.6	1	0.1	8.0	0.9, 69.2	0	0.2			2	0.5	3.7	0.9, 16.0
Non-Hodgkin's lymphoma (200, 202)	0	0.3			0	0.5			0	0.6			4	0.9	4.6	1.6, 12.9	4	2.2	1.8	0.7, 4.9
Multiple myeloma (203)	0	0.3			1	0.2	5.2	0.7, 40.8	3	0.2	14.0	4.0, 52.8	0	0.6			4	1.3	3.2	1.2, 8.8
Leukemia (204–208)	1	0.7	1.4	0.2, 10.5	0	0.8			0	0.8			2	0.8	2.6	0.6, 11.1	3	3.1	1.0	0.3, 3.0
Myeloid leukemia (205)	0	0.3			0	0.3			0	0.4			1	0.4	2.7	0.4, 20.9	1	1.4	0.7	0.1, 5.1
Diabetes mellitus (250)	4	2.0	2.0	0.8, 5.5	1	2.9	0.3	0.04, 2.5	7	3.2	2.2	1.0, 4.6	8	3.4	2.4	1.2, 4.8	20	11.6	1.7	0.1, 2.7
All circulatory diseases (390–459)	32	30.4	1.1	0.7, 1.5	37	31.4	1.2	0.9, 1.6	20	29.2	0.7	0.4, 1.1	26	35.8	0.7	0.5, 1.1	115	126.6	0.9	0.8, 1.1
Hypertension (400–405)	3	2.1	1.4	0.5, 4.6	1	2.0	0.5	0.1, 3.6	2	2.2	0.9	0.2, 3.6	1	2.1	0.5	0.1, 3.3	7	8.6	0.8	0.4, 1.7
Ischemic heart disease (410–414)	8	8.2	1.0	0.5, 2.0	10	10.1	1.0	0.5, 1.9	6	8.7	0.7	0.3, 1.6	8	10.4	0.8	0.4, 1.5	32	37.4	0.9	0.6, 1.2
Myocardial infarction (410)	5	3.7	1.4	0.6, 3.3	2	4.4	0.5	0.1, 1.8	2	4.9	0.4	0.1, 1.6	5	5.5	0.9	0.4, 2.2	14	18.5	0.8	0.4, 1.3
Chronic ischemic heart disease (412, 414)	3	4.6	0.7	0.2, 2.1	8	5.6	1.4	0.7, 2.9	4	3.8	1.1	0.4, 2.8	3	4.9	0.6	0.2, 1.9	18	18.8	1.0	0.6, 1.5
Cerebrovascular disease (430–438)	13	10.6	1.2	0.7, 2.1	11	9.8	1.1	0.6, 2.0	7	9.9	0.7	0.3, 1.5	9	11.5	0.8	0.4, 1.5	40	41.7	1.0	0.7, 1.3

Respiratory disease (460–519)	4	4.1	1.0	0.4,	2	3.5	.0.6	0.1, 2.3	6	3.6	1.7	0.7, 3.8	5	4.6	1.1	0.4, 2.6	17	15.8	1.1	0.7, 1.7				
Chronic obstructive pulmonary disease (490–493)	3	1.2	2.4	0.8, 7.7	2	1.0	1.9	0.5, 7.8	4	1.3	3.0	1.1 8.1	3	1.7	1.8	0.6, 5.6	12	5.4	2.2	1.2, 4.0				
Digestive disease (520–579)	4	4.1	1.0	0.4, 2.6	6	3.9	1.5	0.7, 3.5	4	3.4	1.2	0.4, 3.2	5	4.3	1.2	0.5, 2.8	19	15.9	1.2	0.8, 1.9				
Cirrhosis of the liver (571)	2	2.3	0.9	0.2, 3.5	1	2.2	0.5	0.1, 3.2	0	2.0			2	2.0	1.0	0.2, 4.0	5	8.6	0.6	0.2, 1.4				
Accidents (800–999)	3	3.1	1.0	0.3, 3.1	4	3.8	1.0	0.4, 2.8	5	3.7	1.4	0.6, 3.3	5	3.8	1.3	0.5, 3.2	17	14.4	1.2	0.7, 1.9				

* Adjusted for age and calendar period.
† ICD-9, *International Classification of Diseases and Causes of Death*; Ninth Revision; Obs, observed no.; Exp, expected no.; RR, rate ratio; CI, confidence interval.

diseases. In the zones A and B merged male population, all-cancer deaths were significantly in excess after 15 years. The magnitude of the excess was similar to that estimated in previous long-term studies of high-exposure, male occupational cohorts (30, 39–41). Lung cancer mortality also was elevated; the increase was significant in the highest-exposed zone A male population after 15 years of latency. Several independent studies examining occupational cohorts with biologically documented exposure to high levels of dioxin found elevated lung cancer risks (25–27, 29, 42, 43). The lung is also one of the organs targeted by the carcinogenic action of TCDD in rats (44) and mice (45). Individual tobacco smoking habits were not known, but the known homogeneity of educational and cultural features between the exposed and reference populations makes systematic differences quite improbable. Although rectal cancer was not considered a priori among the possible health outcomes associated with dioxin exposure, the hypothesis of a dioxin-related increase in rectal cancer is backed by at least one occupational cohort study (43) that found a statistically borderline significant increase, with a relative risk of 2.3 (95 percent CI: 1.0, 4.4). Among zone A plus zone B males, the magnitude of the risk ratio and the persistence of the excess over time converged to lend credibility to the finding. The dietary habits of the exposed and reference populations are known for their commonalties: meat consumption is frequent and includes beef, pork, and courtyard animals (chicken, rabbits); high consumption of vegetables, often grown in backyard gardens, is common. Possible involvement of other digestive sites (the stomach in both zones A and B and, less evidently, the liver in zone B) was suggested by results of latency analysis of females.

The clearest and most consistent excess in zones A and B was for lymphohemopoietic neoplasms, for which the numbers were elevated in both genders, with a latency-related pattern among females. The few observed deaths limited interpretation for specific causes. Non-Hodgkin's lymphoma was significantly elevated in the small, but highly exposed zone A population and increased nonsignificantly in zone B. In zone B, the increases in Hodgkin's disease were significant in the first postaccident decade, as were those for leukemia, in particular myeloid leukemia, in the longest latency category. Suggestive was the increase in multiple myeloma, and the excess risk became significant when females were analyzed separately. On the other hand, leukemia deaths showed the highest increase among males. In previous experimental studies, a dose-related increased occurrence of lymphoma was found in both sexes of mice (45, 46). In occupational cohorts with high levels of exposure to TCDD, non-Hodgkin's lymphoma and, less evidently, Hodgkin's disease were elevated (25, 29, 42, 47). An association with TCDD exposure was also seen for multiple myeloma (40, 42, 43) and possibly for leukemia (48). It is difficult to hypothesize about any systematic difference between the exposed and referent populations in terms of exposure to known biologic, chemical, or radiologic risk factors for hematologic neoplasms (49).

Mortality from noncancer deaths also showed some unusual features. An increase in diabetes mellitus was present

Respiratory disease (460–519)	7	7.1	1.0	0.5	7	6.8	1.0	0.5, 2.2	2	6.2	0.3	0.1, 1.3	11	6.5	1.7	0.9, 3.1	27	26.5	1.0	0.7, 1.5					
Chronic obstructive pulmonary disease (490–493)	4	3.5	1.1	0.4, 3.1	6	3.0	2.0	0.9, 4.5	2	3.6	0.6	0.1, 2.3	5	3.5	1.4	0.6, 3.5	17	13.6	1.2	0.8, 2.0					
Digestive disease (520–579)	4	9.4	0.4	0.2, 1.1	7	7.7	0.9	0.4, 1.9	6	6.2	1.0	0.4, 2.2	7	5.9	1.2	0.6, 2.5	24	29.2	0.8	0.5, 1.2					
Cirrhosis of the liver (571)	3	6.6	0.5	0.1, 1.4	5	5.7	0.9	0.4, 2.1	5	4.3	1.2	0.5, 2.8	3	3.7	0.8	0.3, 2.6	16	20.2	0.8	0.5, 1.3					
Accidents (800–999)	10	9.1	1.1	0.6, 2.1	6	8.3	0.7	0.3, 1.6	9	6.9	1.3	0.7, 2.5	10	7.9	1.3	0.7, 2.4	35	32.4	1.1	0.8, 1.5					

* Adjusted for age and calendar period.
† ICD-9, *International Classification of Diseases and Causes of Death*, Ninth Revision; Obs, observed no.; Exp, expected no.; RR, rate ratio; CI, confidence interval.

among females in all exposure zones, and the increase was suggestively time related. This finding should be interpreted with caution, however. The diagnostic accuracy of death certificates for this condition is poor, but inaccuracy should have affected exposed and nonexposed subjects nondifferentially. Systematic differences in dietary habits, as noted already, were improbable. A hypothetical role for dioxin is biologically plausible in light of the known, although not completely understood, interaction of dioxin with hormonal factors (50). An elevated prevalence of diabetes and a positive association between TCDD serum levels and fasting serum glucose levels were found in a survey of US chemical workers exposed to dioxin, but confounding by other variables could not be excluded. (51). Follow-up of a large cohort of US male chemical workers instead failed to detect any excess mortality from diabetes (40). In an accidentally exposed German industrial cohort, mean fasting glucose levels increased slightly with current, but not back-extrapolated, dioxin levels (52). In addition, highly exposed Vietnam veterans were found to have a high prevalence of diabetes and a decrease in time-to-diabetes onset with dioxin exposure (53); in addition, serum dioxin levels were associated with insulin and sex hormone-binding globulin (54). A merely suggestive increase also was found in an international cohort of chemical workers exposed to TCDD or higher chlorinated dioxins (55).

Among males, circulatory disease mortality (chronic ischemic heart disease in particular) was elevated in zone A in the early postaccident period. The previously mentioned similarities between the exposed and reference populations make differences in smoking and dietary habits a highly improbable explanation for this finding. A possibly differential cause-of-death certification in the early postaccident period can be hypothesized. However, health referral conditions were common in the exposed and reference areas; in addition, cardiovascular disease was not considered among the expected effects of dioxin exposure. That dioxin can adversely affect the cardiovascular system is well documented. TCDD has been shown experimentally to alter cardiac function and morphology (44, 56–60). It increases serum triglycerides and cholesterol, well-established risk factors for cardiovascular disease (61), in both experimental animals (62–64) and humans (65, 66). In an international cohort of pesticide manufacturers and applicators, exposure to TCDD and higher-chlorinated dioxins was associated with significantly increased ischemic heart disease mortality (55). One German (41) and one Dutch (29) study found a significant excess of ischemic heart disease associated with dioxin exposure, whereas another German study (27) did not.

Occurrence of the unusual circulatory disease mortality in the short postaccident period suggests another possibly relevant disease determinant, that is, the heavy psychosocial impact of the accident (67, 68). For months and possibly years after the accident, people experienced intense social rejection, deep anger and frustration, acute fear for their future and the health of their children, anxiety about relocation of their houses and work activities, and so forth. The burden of these disaster-linked psychosocial stressors might have precipitated early deaths from preexisting ill-health

TABLE 7. *Observed and expected numbers of deaths, rate ratios,* and 95% confidence intervals, by cause of death and years since first exposure (latency), for the male population in zones A and B exposed to dioxin after the Seveso, Italy, industrial accident, 1976–1996*

Cause of death (ICD-9† codes)	No. of years since first exposure																			
	0–4				5–9				10–14				15–20				Total			
	Obs†	Exp†	RR†	95% CI†	Obs	Exp	RR	95% CI	Obs	Exp	RR	95% CI	Obs	Exp	RR	95% CI	Obs	Exp	RR	95% CI
All causes (001–999)	103	109.4	0.9	0.8, 1.1	113	104.1	1.1	0.9, 1.3	95	105.7	0.9	0.7, 1.1	127	117.3	1.1	0.9, 1.3	438	436.2	1.0	0.9, 1.1
All cancers (140–208)	28	30.1	0.9	0.6, 1.4	43	34.7	1.2	0.9, 1.7	37	41.2	0.9	0.6, 1.2	58	44.2	1.3	1.0, 1.7	166	149.7	1.1	1.0, 1.3
Digestive (150–159)	7	11.5	0.6	0.3, 1.3	13	13.0	1.0	0.6, 1.7	13	16.0	0.8	0.5, 1.4	20	17.5	1.1	0.7, 1.8	53	57.6	0.9	0.7, 1.2
Stomach (151)	4	4.2	0.9	0.4, 2.6	3	4.7	0.6	0.2, 2.0	3	4.7	0.6	0.2, 2.0	6	4.3	1.4	0.6, 3.1	16	17.9	0.9	0.5, 1.5
Colon (153)	0	2.1			2	2.1	0.9	0.2, 3.8	3	2.5	1.2	0.4, 3.8	5	3.1	1.6	0.7, 3.9	10	9.8	1.0	0.5, 1.9
Rectum (154)	2	0.8	2.6	0.6, 10.8	1	1.0	1.0	0.1, 7.6	3	1.1	2.8	0.9, 9.0	3	1.0	2.9	0.9, 9.5	9	3.8	2.4	1.2, 4.6
Hepatobiliary (155–156)	0	1.8			3	2.2	1.4	0.4, 4.3	1	4.4	0.2	0.03, 1.6	2	4.8	0.4	0.1, 1.7	6	13.2	0.5	0.2, 1.0
Liver (155)	0	1.6			3	2.0	1.5	0.5, 4.8	1	4.1	0.2	0.03, 1.7	2	4.4	0.5	0.1, 1.8	6	12.1	0.5	0.2, 1.1
Pancreas (157)	1	1.2	0.8	0.1, 6.1	1	1.4	0.7	0.1, 5.2	1	1.4	0.7	0.1, 5.1	1	1.8	0.6	0.1, 4.1	6	5.8	0.7	0.3, 1.9
Other digestive (159)	0	0.2			2	0.5	4.2	1.0, 18.3	1	0.7	1.5	0.2, 10.9	2	1.0	2.0	0.5, 8.5	4	2.3	2.2	0.9, 5.4
Respiratory (160–165)	10	10.4	1.0	0.5, 1.8	18	11.8	1.5	1.0, 2.5	16	14.1	1.1	0.7, 1.9	20	14.1	1.4	0.9, 2.2	64	50.3	1.3	1.0, 1.6
Lung (162)	9	8.9	1.0	0.5, 2.0	15	9.7	1.5	0.9, 2.6	14	12.7	1.1	0.6, 1.9	19	12.8	1.5	0.9, 2.4	57	43.9	1.3	1.0, 1.7
Melanoma (172)	0	0.1			0	0.1			0	0.3			1	0.3	3.5	0.4, 27.2	1	0.7	1.5	0.2, 12.5
Genitourinary (179–189)	4	3.1	1.3	0.5, 3.5	5	3.9	1.3	0.5, 3.2	1	4.6	0.2	0.03, 1.5	7	4.9	1.4	0.7, 3.0	17	16.5	1.0	0.6, 1.7
Prostate (185)	3	1.5	2.0	0.6, 6.3	2	1.7	1.2	0.3, 4.9	1	2.0	0.5	0.1, 3.6	2	2.3	0.9	0.2, 3.5	8	7.5	1.1	0.5, 2.2
Bladder (188)	1	0.9	1.1	0.2, 8.2	3	1.2	2.6	0.8, 8.4	0	1.5			2	1.4	1.4	0.3, 5.9	6	5.0	1.2	0.5, 2.7
Kidney (189)	0	0.6			0	0.9			0	1.0			3	1.2	2.5	0.8, 8.0	3	3.7	0.8	0.3, 2.6
Brain (191)	0	0.2			1	0.8	1.2	0.2, 9.0	0	0.6			0	0.8			1	2.4	0.4	0.1, 3.0
Lymphatic and hemopoietic (200–208)	4	2.1	1.9	0.7, 5.3	4	2.5	1.9	0.7, 5.3	4	2.2	1.8	0.7, 4.9	3	2.8	1.1	0.3, 3.4	15	9.1	1.7	1.0, 2.8
Hodgkin's disease (201)	1	0.2	5.3	0.7, 43.4	1	0.2	4.6	0.6, 37.2	0	0.3			0	0.1			2	0.8	2.6	0.6, 10.9
Non-Hodgkin's lymphoma (200, 202)	0	0.3			0	0.5			2	0.8	2.6	0.6, 11.0	1	0.9	1.1	0.1, 7.9	3	2.4	1.2	0.4, 3.9
Multiple myeloma (203)	0	0.3			1	0.4	2.5	0.3, 18.4	0	0.4			0	0.6			1	1.6	0.6	0.1, 4.3
Leukemia (204–208)	3	1.3	2.3	0.7, 7.5	2	1.0	1.9	0.5, 8.1	2	0.9	2.3	0.5, 9.6	2	1.1	1.7	0.4, 7.2	9	4.3	2.1	1.1, 4.1
Lymphatic leukemia (204)	1	0.3	4.0	0.5, 31.6	1	0.2	4.9	0.6, 39.2	0	0.3			0	0.5			2	1.2	1.6	0.4, 6.8
Myeloid leukemia (205)	1	0.3	2.9	0.4, 22.3	0	0.4			2	0.3	6.0	1.3, 26.7	2	0.4	4.6	1.0, 20.0	5	1.5	3.4	1.3, 8.4
Leukemia, unspecified (208)	1	0.7	1.4	0.2, 10.6	1	0.4	2.4	0.3, 18.5	0	0.2			0	0.2			2	1.5	1.3	0.3, 5.3
Diabetes mellitus (250)	3	1.6	1.8	0.6, 5.8	2	1.8	1.1	0.3, 4.4	1	1.7	0.6	0.1, 4.1	0	2.5			6	7.7	0.8	0.3, 1.7
All circulatory diseases (390–459)	39	39.9	1.0	0.7, 1.3	44	37.5	1.2	0.9, 1.6	32	34.3	0.9	0.7, 1.3	35	36.2	1.0	0.7, 1.4	150	148.3	1.0	0.9, 1.2
Hypertension (400–405)	0	1.6			0	1.3			1	1.2	0.8	0.1, 5.9	1	1.5	0.7	0.1, 4.8	2	5.8	0.3	0.1, 1.4
Ischemic heart disease (410–414)	13	16.6	0.8	0.5, 1.4	20	17.3	1.2	0.7, 1.8	14	15.0	0.9	0.5, 1.6	18	14.8	1.2	0.8, 1.9	65	63.9	1.0	0.8, 1.3
Myocardial infarction (410)	6	11.7	0.5	0.2, 1.1	8	11.4	0.7	0.3, 1.4	9	10.3	0.9	0.5, 1.7	13	9.9	1.3	0.8, 2.3	36	43.5	0.8	0.6, 1.2
Chronic ischemic heart disease (412, 414)	7	4.8	1.5	0.7, 3.1	11	5.8	1.9	1.0, 3.5	5	4.7	1.1	0.4, 2.6	5	5.0	1.0	0.4, 2.4	28	20.3	1.4	0.9, 2.0
Cerebrovascular disease (430–438)	12	11.0	1.1	0.6, 1.9	14	9.7	1.4	0.8, 2.5	11	9.2	1.2	0.7, 2.2	11	8.9	1.2	0.7, 2.2	48	38.9	1.2	0.9, 1.6

conditions. Disruption of the social environment following a disastrous event is a well-known cause of distress and can also influence coronary heart disease risk factors (69–72).

The increased chronic obstructive pulmonary disease mortality was especially apparent among males in zone A, but without a distinct time-related pattern, and it also affected women in zones A and B. Previous studies of TCDD-exposed populations do not support this association. It is difficult to hypothesize such an extreme and systematic difference in smoking habits between the otherwise-so-similar index and reference populations that would explain a threefold increased relative risk. In addition, such a difference would have affected the results for smoking-associated cancer. The most plausible way in which TCDD might have contributed to this finding is through its recognized immunotoxic activity (73, 74). Impaired protection and defense against episodes of respiratory infection play a major role in the natural history of chronic obstructive pulmonary disease (75). As for chronic ischemic heart disease, even among people with preexisting chronic obstructive pulmonary disease, the accident-related stressors might have been relevant in precipitating early deaths.

The limited number of available blood dioxin measurements did not allow individual categorization by TCDD dose. Therefore, no appropriate dose-response estimates were possible, and population exposure characterization remained ecologic. The noted increased risks became apparent in the high-exposure zones. Results for the least-contaminated zone R failed to suggest increased cancer risks, whereas a possible modest excess mortality from diabetes and chronic ischemic heart disease could not be excluded.

Extrapolation of these high-exposure risk estimates to current environmental dioxin levels is problematic. Instead, results of this study are informative with regard to hazard identification. They add further evidence in support of the recent evaluation (76) of dioxin as a human carcinogen, and they corroborate the hypotheses of its association with cardiovascular and endocrine-related health effects. To further elucidate the TCDD action mechanism, epidemiologic studies that use biochemical and molecular markers are being conducted in subsamples of the study population (77). Additional insight into cancer risk is also expected from the concurrent incidence study (24). The increased relative risk estimates for several causes of cancer in the >15-year latency period make continuation of the follow-up mandatory.

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Articles

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The Belgian PCB and Dioxin Incident of January-June 1999: Exposure Data and Potential Impact on Health

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Abstract
In January 1999, 500 tons of feed contaminated with approximately 50 kg of polychlorinated biphenyls (PCBs) and 1 g of dioxins were distributed to animal farms in Belgium, and to a lesser extent in the Netherlands, France, and Germany. This study was based on 20,491 samples collected in the database of the Belgian federal ministries from animal feed, cattle, pork, poultry, eggs, milk, and various fat-containing food items analyzed for their PCB and/or dioxin content. Dioxin measurements showed a clear predominance of polychlorinated dibenzofuran over polychlorinated dibenzodioxin congeners, a dioxin/PCB ratio of approximately 1:50,000 and a PCB fingerprint resembling that of an Aroclor mixture, thus confirming contamination by transformer oil rather than by other environmental sources. In this case the PCBs contribute significantly more to toxic equivalents (TEQ) than dioxins. The respective means ± SDs and the maximum concentrations of dioxin (expressed in TEQ) and PCB observed per gram of fat in contaminated food were 170.3 ± 487.7 pg, 2613.4 pg, 240.7 ± 2036.9 ng, and 51059.0 ng in chicken; 1.9 ± 0.8 pg, 4.3 pg, 34.2 ± 30.5 ng, and 314.0 ng in milk; and 32.0 ± 104.4 pg, 713.3 pg, 392.7 ± 2883.5 ng, and 46000.0 ng in eggs. Assuming that as a consequence of this incident between 10 and 15 kg PCBs and from 200 to 300 mg dioxins were ingested by 10 million Belgians, the mean intake per kilogram of body weight is calculated to maximally 25,000 ng PCBs and 500 pg international TEQ dioxins. Estimates of the total number of cancers resulting from this incident range between 40 and 8,000. Neurotoxic and behavioral effects in neonates are also to be expected but cannot be quantified. Because food items differed widely (more than 50-fold) in the ratio of PCBs to dioxins, other significant sources of contamination and a high background contamination are likely to contribute substantially to the exposure of the Belgian population. **Key words**: Belgium, cancer, dioxin, food chain, polychlorinated biphenyls, risk assessment. **Environ Health Perspect** 109:265-273 (2001). [Online 1 March 2001]

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Introduction

In Belgium, approximately 20 companies collect animal fat from slaughterhouses and melt it into a homogenous substance, which is sold to animal-food producers. It is a common practice to include household waste fat collected at community waste recycling centers in this product. In January 1999, at the Flemish fat-melting company Verkest, 40-50 kg of mineral oil containing polychlorinated biphenyls (PCBs; most likely oil from discarded transformers originating from a waste recycling center) was admixed to the fat delivered to 10 animal-feed producers. Between 15 and 31 January, the resulting 500 tons of contaminated animal feed, containing approximately 60-80 tons of fat contaminated with 40-50 kg of PCBs and almost 1 g of dioxins, were distributed to poultry farms and to a lesser extent also to rabbit, calf, cow, and pig breeding and raising farms, mostly in Belgium. Small quantities were exported to the Netherlands, France, and Germany. In Belgium, 445 poultry farms, 393 bovine farms, 746 pig farms (or a quarter of this type of farm in Belgium) and 237 dairy farms (representing 1.5% of the total number of dairy farms in Belgium) used animal feed from the 10 contaminated animal-feed producers. The 500 tons of contaminated feed represent a limited percentage of the total amount of feed produced and used in Belgium, which is estimated to exceed 28,000 tons/week.

Pathologic conditions were first recorded on 4 February on Belgian chicken farms. They included a decrease in egg production and hatching and an epidemic of chicken edema disease. After excluding other causes of the epidemic, samples of animal feed, chicken carcasses and eggs were sent for toxicologic analysis on 18 March. The authorities were informed of the presence of increased concentrations of dioxins in animal feed, chicken, and eggs on 26 April. The first measures to protect public health were taken, but the public was not informed. On 27 May the Flemish television broke the news; the incident became public and resulted in a political, communication, and economic crisis. The authorities tried to identify the extent of the crisis by identifying the companies involved in the contamination. All available laboratories in Belgium and neighboring countries were mobilized for analytical work. The analyses revealed that the dioxins were part of a PCB contamination, and average PCB/dioxin ratios were determined. During the first days of June, the Ministry of Public Health ordered removal of poultry, derived products (meat, eggs, mayonnaise, custards, cakes, etc.), and all meat products with a fat content > 25% from the market. A widespread product sampling and analysis was organized, resulting in the data used in the present study. Only products with a concentration of < 200 ng PCBs/g fat were released for human consumption. Products with excessive levels were destroyed, including some 2 million chickens. The duration of exposure of the population can hence be estimated as 4 months (February-May).

Dioxin Exposure in Belgium before the Incident

Atmospheric pollution by identified and disperse emission sources of dioxins in Flanders (northern Belgium) has been covered in several studies (*1,2*). These studies show a gradual reduction in emissions from > 600 g international toxic equivalents (I-TEQ)/year in 1994 to approximately 100 g I-TEQ/year in 1999. Whereas municipal solid waste incineration accounted for nearly 60% of dioxin emissions in 1993, its contribution is now limited to a low percentage. The nonferrous, ferrous, and

steel industries remain major sources, together with household heating and traffic. More stringent regulations and continuous monitoring will further reduce the emissions, although the past levels will contribute to background soil pollution and body burdens for a long time.

Table 1

In Belgium in 1995, 1,151 g I-TEQ were emitted (*3*). As shown in Table 1, 60% of this value is due to emissions to air. In this figure emissions to water are likely to be underestimated, as the emission inventory identifies only a limited number of sources. The polychlorinated dibenzodioxin/polychlorinated dibenzofuran (PCDD/PCDF) flux in Belgium is high. A comparison of inventories for 15 countries (*4*) shows that the per capita emission in Belgium is the second highest after Japan.

Background air immission measurements are scarce, but existing information shows values ranging between 100 and 255 fg TEQ/m^3 (*5*).

More information is available on deposition, which is measured systematically at 20 sites. These include a few rural and urban reference areas but are concentrated around specific emission sources such as municipal waste incinerators and steel mills. During 1993-1997, the values ranged between 0.26 to 374 ng I-TEQ/m^2 per year (*5*).

Limited data on the food load of dioxins and PCBs are available. Cox (*6*) reviewed the existing data. Incomplete data were completed by information on Dutch measurements of dioxins in the food basket. Cox concluded that the average total daily intake was 179 pg I-TEQ/day. These data are a rough estimate because the dioxin concentrations in food are only partially known, and because the official food basket composition as used by the government (*7*) is outdated.

Of particular interest are the data on dioxin concentrations in milk. The prevailing mean concentrations are 2.1 pg I-TEQ/g milkfat (range: 1.3-2.5 pg I-TEQ/g milkfat). However, in cows grazing close to municipal waste incinerators, values up to 21 pg I-TEQ/g milkfat have been recorded.

Dioxins and furans in milk of lactating women were studied in 1988 and 1993 by the World Health Organization (WHO) (*8*). Belgian samples in this international study originated from women living in Brussels, the rural area south of Brussels, and Liège, capital of the eastern Belgian province. In these samples an average value of 34.4 pg I-TEQ/g milkfat (range 27.3-43.2 pg I-TEQ/g milkfat) was found. The dioxin concentrations in the Belgian samples are approximately twice as high as those measured in Austria, Denmark, Croatia, or Canada. Levels were stationary between 1988 and 1993.

Table 1 summarizes the data mentioned above and compares them with the available reference values. This comparison illustrates that dioxin background values in Belgium often exceed the reference values. The situation is most pronounced for babies. Assuming the 34.4 pg I-TEQ/g fat in mother's milk is representative of the country as a whole, babies are fed an average of 43 pg I-TEQ/kg per day. This is substantially more than the 1-4 pg/kg per day that has been proposed by the WHO. It is also 20 times higher than the average intake by an adult and indicates that during 3 months of breast-feeding, Belgian babies take in 6% of their lifetime dioxin dose.

The mean preincident concentrations in breast milk samples of mothers nursing their firstborn baby (*9*) also allow one to estimate the body burden. Assuming that fat amounts to 20% of body weight, 34.4 pg I-TEQ/g fat corresponds to a body burden of 6.88 ng I-TEQ/kg body weight in these young women. The global picture which emerges is that Belgians are exposed to high background concentrations of dioxins and furans.

Objectives of This Study

During the initial period (January to end of May) of the PCB-dioxin contamination incident in Belgium, only a few dioxin measurements were performed as the incident was treated in a confidential manner. Large-scale sampling and laboratory testing happened after the crisis went public on 27 May. We assessed the results concerning the samples taken from the end of May until August 1999. The data were used to estimate the overall population exposure and the exposure that might have resulted from selected diets. On this basis the potential impact on public health was estimated. More than 40,000 additional samples were analyzed in the period after August 1999, and monitoring is still ongoing. These results will be dealt with in a separate follow-up paper.

Materials and Methods

Dioxin and PCB Measurements in Animals and Food Products

Samples and measurements. The data assessed here are based on the analyses ordered by the Belgian Ministries of Health and Agriculture. They were presented to the Belgian Parliament in September 1999. As shown in Table 2, 20,491 samples from cattle, pigs, poultry, eggs, milk, and various fat-containing food items (ranging from mayonnaise to Belgian chocolates) were collected by different control departments of the Ministries of Public Health and Agriculture. The sample record form included information on the commercial origin of animal feed for meat or the dairy products, or of dairy ingredients in food.

Table 2

In this study, samples were divided into three classes:

- "Incident-related samples" for which the origin could be traced back to farms that used feed from one of the 10 animal-feed producers who bought contaminated fat from the Flemish fat-melting company Verkest; such samples are not necessarily contaminated by the incident, but might be so

- "Samples unrelated to the incident," for which the origin could be traced back to farms that did not use feed from one of these 10 feed producers

- "Samples without a track record leading to the incident"; these samples could not be traced back to farms that did use feed from one of these 10 feed producers, or to farms that did not use feed from one of these 10 feed producers.

Results of 20,290 PCB measurements and 446 dioxin measurements are available; for 245 of the samples, both PCB and dioxin measurements are available. PCB measurements included seven marker congeners--namely, PCBs 28, 52, 101, 118, 138, 153, and 180--and are expressed as the sum of these congeners in nanograms per gram fat. For PCDD/PCDF the "dirty 17" congeners with

chlorine substitution of at least the 2, 3, 7, and 8 positions were measured, and the total dioxin content of the sample was expressed in picogram TEQ per gram fat, using the WHO-toxic equivalence factor (TEF) values (*10*).

Quality control. Dioxins were measured using mass spectrometry. PCBs were quantified using gas chromatographic techniques followed by electron capture or mass spectrometry.

All but 18 of the PCB measurements were performed by a pool of 23 laboratories accepted by the Belgian authorities after a technical assessment; dioxin measurements were performed by 18 accredited laboratories, 5 of which accounted for 398 of all 446 measurements. Twenty-two of the laboratories that participated in PCB measurements were also included in a quality-ring test of PCB measurements, organized by the dioxin-PCB unit of the Belgian Ministries of Public Health and Agriculture (*11*).

The results showed that for contaminated feedstuff, egg yolk, and pork fat, coefficients of variation for reproducibility ranging between 16 and 35% were found. However, for slightly contaminated lyophilized milk, unacceptably high coefficients of variation were recorded.

Table 2 reviews the number of PCB and PCDD/PCDF samples analyzed: 71% of the samples tested for PCBs showed values below the detection threshold, which was specific for each laboratory. For dioxins only 10% of the samples had concentrations lower than the detection limits of the individual congeners.

Estimation of Cancer Risk

We estimated the incremental risk of cancer associated with the calculated incremental levels of exposure of the Belgian population to PCBs and/or to dioxins during the 1999 incident. First, we calculated the body burden incurred by a given population, resulting from a defined exposure, whether through a certain diet or by assuming that a certain amount of PCBs and/or dioxins was shared between a defined number of persons. Because the available quantitative risk estimates are based on daily intake values of these persistent chemicals, and since their half-life time in humans is on the order of several years, probably about 7 years for dioxins (*12,13*) and between 4.1 and 34.2 years for PCBs (*14*), we divided the incident-associated body burden by 25,550 (the number of days in 70 years), yielding a corresponding averaged daily intake. This approach assumes that no dioxin is eliminated during the exposure period of 4 months. This daily intake value was then compared to the risk estimates for dioxins as provided by Becher et al. (*15*), and for PCBs as provided by Cogliano (*16*). For academic reasons, an assessment based on a cancer risk estimate of 1 in 1 million for a life-time exposure of 0.006 pg I-TEQ/kg body weight per day, is also added for dioxin-related effects.

Results

Nature of the Contamination and Impact on the Food Chain

The contamination chain is illustrated in Figure 1. It shows how 40-50 kg of PCBs and almost 1 g of dioxins were distributed over an estimated 500 tons of animal feed and a still undefined amount of animals and derived animal food products. During the initial response to the incident, a limited number of mostly heavily contaminated samples were analyzed for both dioxins and PCBs.

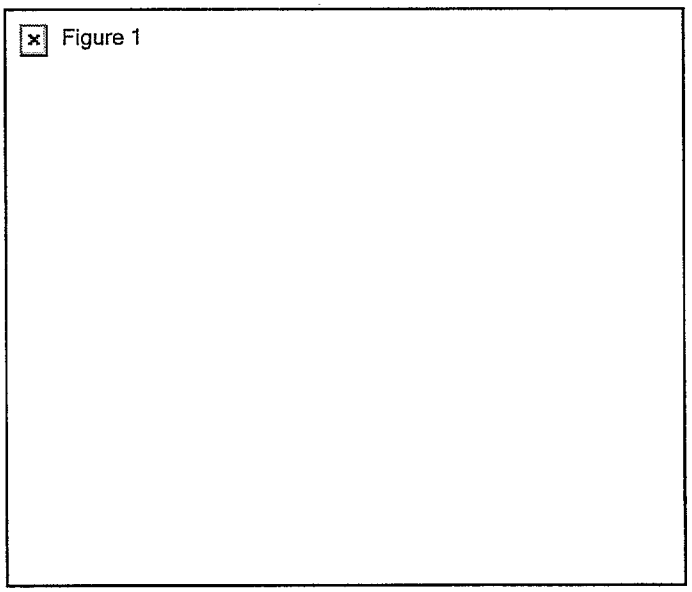


Figure 1. Fate of dioxin and PCBs in the food chain during the contamination incident (*34*).

The PCDD/PCDF congener distributions in four samples of animal feed heavily contaminated by the incident are shown in Figure 2. The congener profile shows a clear predominance of PCDF over PCDD congeners. This is compatible with a PCB contamination by a substance (such as transformer oil) containing PCBs rather than by dioxins originating from thermal processes such as waste incineration. The fingerprint of PCDD/PCDF-emissions by municipal waste incinerators is substantially different from the one found in animal feed (Figure 2). The furans-to-dioxins ratio is 1.7 for the incinerators and 16 for the animal food.

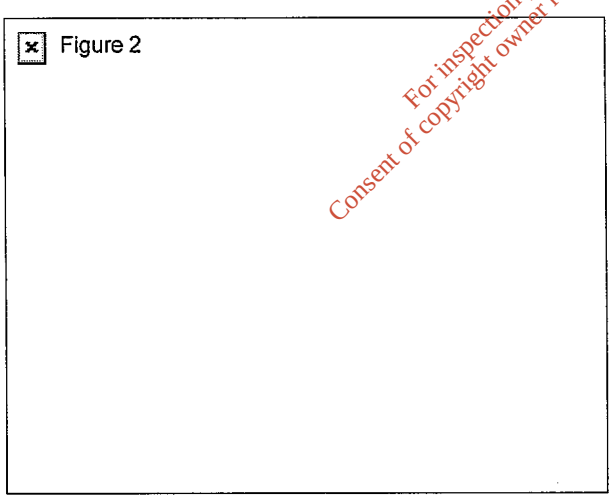
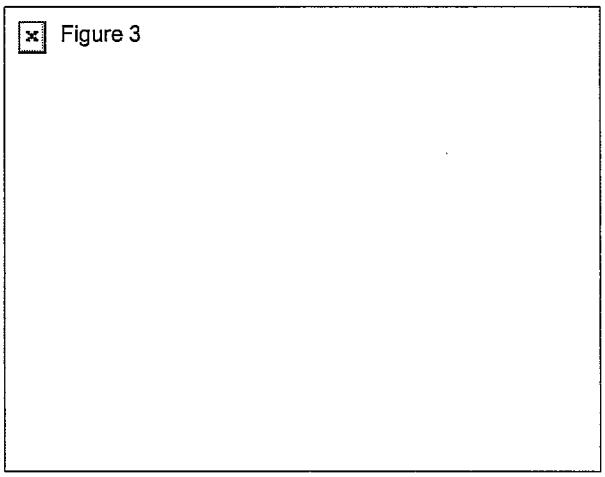


Figure 2. PCDD/PCDF congener distributions (in percentage of weight) measured in four samples of contaminated feed (mean ± SD). The results are compared with the congener distribution as found in emissions of municipal waste incinerators (MWI).

To further identify the nature of the PCB oil, different commercial PCB formulations were analyzed under the same conditions as the contaminated feed samples. The results for the 7 marker PCBs in 47 contaminated animal feed samples (containing > 1,000 ng PCB/g fat) are given in Figure 3, which also includes the profiles for a 50/50 mixture of Aroclors 1254 and 1260. There is a good agreement between the contamination pattern found in animal feed and the profile of the mixture of both Aroclors. The contamination pattern shown by these 47 samples is called the "incident-related PCB profile of animal feed."

Figure 3. Weight percent distribution of seven PCB marker congeners in 47 contaminated animal feed samples (mean ± SD) and a 50/50 mixture of Aroclors (A) 1254



and 1260.

The congener distribution in 11 egg samples contaminated with > 1,000 ng PCB/g fat is shown in Figure 4. The data for eggs, reflecting the result of metabolic conversion, are compared with the profiles found in the 47 samples of animal feed. The comparison shows that the higher PCBs (PCB 118, 138, 153, and 180) are the most persistent ones. The lower chlorinated PCBs (PCB 52 and 101) are more easily metabolized or excreted. The contamination pattern shown by these 11 samples is called the "incident-related PCB profile of eggs."

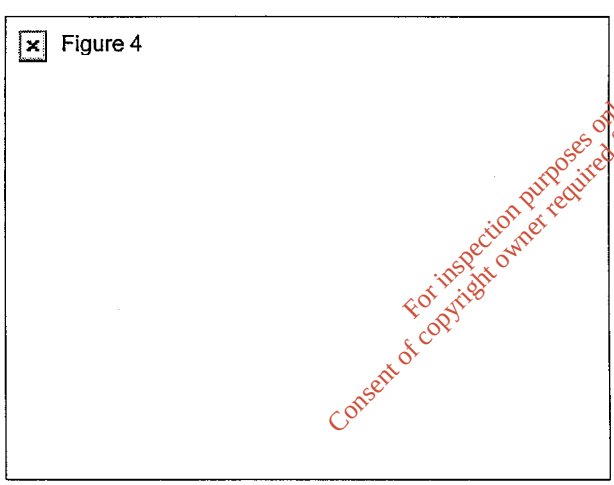


Figure 4. PCB congener distribution (weight %) in contaminated eggs, compared to distribution in contaminated animal feed (mean ± SD).

Figure 5 shows the concentration of PCBs plotted against the ratio between the sum of the seven marker PCBs over the dioxins (in I-TEQ) found in the respective samples, for all samples for which both PCB and dioxin data are available. The figure reveals that for most (12 out of 16) samples with concentrations of PCBs > 5,000 ng/g fat, about 50,000 times more PCBs than dioxins are found. This coincides with the ratio found in transformer oil. Numerous less-contaminated samples show substantially different ratios.

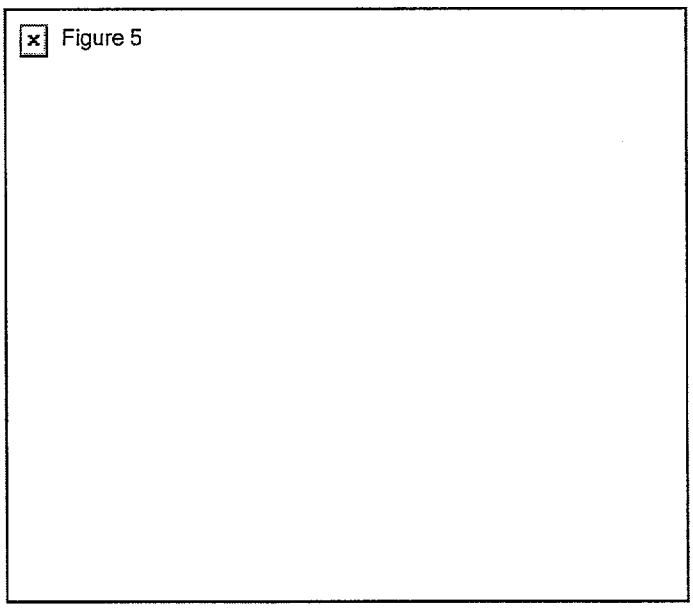


Figure 5. PCB/dioxin ratio in 246 samples for which both dioxin and PCB concentrations were measured.

The PCB congener distribution in many samples differs from the typical incident-related PCB profile. Figure 6 compares the incident-related PCB profile for animal feed with the PCB profile in four animal-feed samples unrelated to the incident. The figure shows that these five profiles differ. Five egg samples unrelated to the incident but showing PCB concentrations ranging between 1,111 and 1,405 ng/g fat likewise showed a PCB profile that differed substantially from the incident-related PCB profile for eggs.

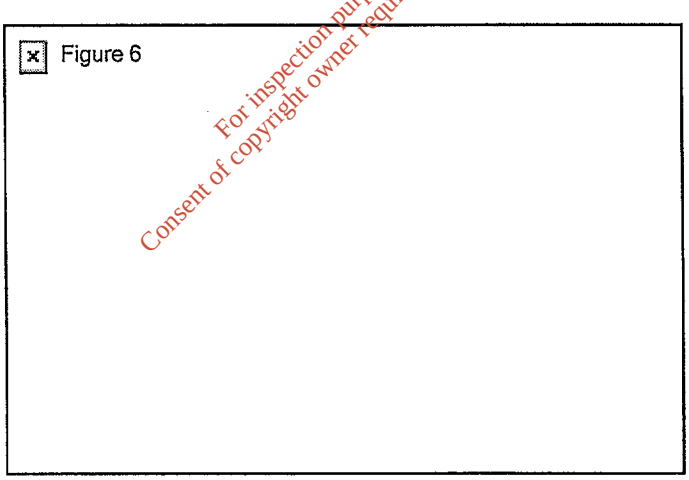


Figure 6. PCB congener distribution of animal feed samples. The results of four contaminated animal feed samples, unrelated to the incident, are compared to the incident-related PCB profile (mean ± SD). Contamination levels are 1,869 ng PCBs/g fat for sample 1; 11,251 ng PCBs/g fat for sample 2; 809 ng PCBs/g fat for sample 3; and 407 ng PCBs/g fat for sample 4. These samples are considered unrelated to the incident.

Table 3 provides concentrations of PCBs and dioxins in a series of food items sampled between the end of May and August 1999. The data show that the highest dioxin concentrations were found in poultry fat and eggs. Pork, beef, and milk were also contaminated, but to a much lower extent. It should be realized that contamination levels in the period February-May might have been higher. Also 4.1% of poultry, 3.94% of egg, and 1.03% of pork samples without a track record leading to the incident contained > 200 ng PCBs/g fat. Samples unrelated to the incident also showed dioxin levels > 2 pg TEQ/g fat in 60% of the beef samples, 31.9% of the milk samples, 16.7% of the poultry meat samples, 58.3% of the eggs, 9.38% of the pork samples, and 27.9% of the processed food samples.

Table 3

To obtain an indication of the contribution of non-*ortho* and mono-*ortho* PCBs to the toxic potency of contaminated samples, we analyzed two animal feed samples, two egg samples, three chicken samples, and two pork samples for both marker PCBs and dioxin-like PCBs. Table 4 lists the details from seven of these analyses. Taking the WHO-TEF value into account, the TEQ values for the different PCBs were calculated. The ratio between PCB TEQ and the sum of marker PCBs amounted to 1:15,622 and 1:11,373 for the two feed samples; 1:11,702 and 1:11,750 for the two egg samples; 1:17,618, 1:16,722 and 1:17,735 for the chicken samples; and 1:42,892 and 1:44,651 for the pork samples. Taking into account a median value of 16,722, this would imply that the incident brought, with an exposure to 15 kg of marker PCBs, also an exposure to 0.9 g TEQ from PCBs.

Table 4

Individual Exposure

The individual exposure that might have resulted from the incident was assessed using two exposure models. The first model assumes an even distribution of the pollution over the Belgian population. The second model is based on selected diets.

Model based on the total amount of PCBs and dioxins released during the incident. Fifty kilograms of PCBs were introduced in the food chain through this incident. It is assumed that 30% of this amount (i.e., 15 kg) was ingested by 10 million Belgians. With a mean body weight of 60 kg for the modal citizen (children included), this represents an average intake of 25,000 ng/kg body weight.

As the body burden for PCBs of the Belgian population before the incident was about 300 ng/g fat (*8*), corresponding to 60 µg/kg body weight, the incident, responsible for an additional estimated

mean of 25 µg/kg body weight, would have increased the average PCB body burden of the Belgian citizen by about 42%. It is assumed that this contribution to the body burden would correspond to a daily intake of 1 ng/kg per day for 70 years (= 25,550 days).

Using similar assumptions, the 1 g I-TEQ of dioxins would have resulted in an increase in body burden with 500 pg I-TEQ/kg body weight. This represents an increase of 7%, assuming a mean baseline dioxin body burden of 6.88 ng I-TEQ/kg. This value also corresponds to an intake corresponding to 0.02 pg/kg per day over 70 years.

Model based on selected dietary patterns. Belgian adults consume an average of 75 g animal fat per day (*6*). As shown in Table 3, 6.5% of the poultry meat, 8.1% of the eggs, and 16.5% of the pork samples contained > 200 ng PCBs/g fat. Two percent of the chicken and 7% of the pork samples analyzed were shown to contain > 1,000 ng PCBs/g fat. The maximum dioxin values of 2,613 and 713 pg I-TEQ/g, respectively, were measured in poultry and eggs.

Levels of contamination up to hundreds of picograms TEQ per gram of fat probably prevailed in a much higher percentage of food items from February until the end of May than from the end of May until August, when measurements took place. Therefore, a certain percentage of the Belgian population could have incurred exposures as those calculated below.

An adult (70 kg body weight) who consumed 15 g of animal fat per day, contaminated with 200 pg I-TEQ dioxins/g, during the 4-month period (120 days) in which the contaminated food was distributed would increase his or her body burden by 360 ng I-TEQ, or 5.14 ng I-TEQ/kg body weight. This corresponds to an increase of 75%, assuming a mean baseline dioxin body burden of 6.88 ng I-TEQ/kg. This intake of 15 g contaminated animal fat per day corresponds to a daily intake of 42.8 pg/kg body weight, 10-40 times the total daily intake (1-4 pg/kg body weight per day) accepted by the WHO.

An adult who consumed 150 g chicken meat contaminated with 700 pg TEQ/g fat three times a week during the same 4-month period would increase his or her body burden by 201.6 ng I-TEQ, or 2.88 ng I-TEQ/kg body weight, which corresponds to an increase of 42%.

An average adult with a body weight of 70 kg would consume the following quantities weekly during a 4-month period: seven servings of meat (150 g/serving with 10% fat), contaminated with 70 pg I-TEQ/g; two eggs, contaminated with 200 pg I-TEQ/g; and one serving of chicken (150 g/serving), contaminated with 700 pg I-TEQ/g.

This diet would increase the total dioxin load by 232 ng I-TEQ (i.e., 3.31 ng/kg body weight). The estimated body burden before the incident of 6.88 ng I-TEQ/kg is increased by 48% in this scenario (*9*). Some persons might have incurred even higher exposures because consumption of milk and derived food products such as sauces and pastry were not included in the above estimations.

Cancer Risk Assessment

Cancer risk assessment for dioxins. Based on the cancer risk estimate of 1 in 1 million for a lifetime exposure of 0.006 pg I-TEQ/kg body weight per day, the incident-specific incremental exposure, estimated above to be equivalent to an intake of 0.02 pg I-TEQ/kg per day during 70 years, would entail a risk of 32 additional cancer deaths in the total Belgian population of 10 million.

Becher et al. (*15*) published a considerably higher risk estimate based on a cohort of workers who were primarily exposed to TCDD. They found that a lifetime exposure of 1 pg TCDD/kg body weight per day entails an incremental lifetime cancer mortality risk of between 1.3 and 7.7 per 1,000. This risk estimate can be applied to an exposure quantified in terms of TCDD-like I-TEQ, as is implicit in the TEF and TEQ concepts. Applying the risk estimates of Becher et al. (*15*) to the dioxin

I-TEQ load due to the incident and assuming a linear dose-risk model, between 260 and 1,540 additional cancer deaths would be expected in the total Belgian population of 10 million.

Cancer risk assessment for PCBs. PCB mixtures have tumorigenic effects that do not necessarily depend on their dioxin-like TEQ value. Cogliano (*16*) developed equations based on animal experiments allowing extrapolation to calculate the risk for humans. Again, assuming a linear dose-risk relationship based on the central slope of the dose-effect curve for Aroclor 1254 (indicating a relative risk of 1.2 for 1 mg/kg body weight per day), the intake of PCBs of 1 ng/kg body weight per day due to the incident would cause an estimated 12 additional cancer deaths in the Belgian population of 10 million individuals. Taking the upper bound slope for Aroclor 1260 (indicating a relative risk of 2.2 for 1 mg/kg body weight per day), this risk amounts to 22 cancer deaths in a population of 10 million.

Weathered PCB mixtures, after chemical modifications caused by the use of the oil, have an increased toxic activity, usually by a factor of 3 (*16*). In the Belgian incident, the PCB mixtures, before being admixed to animal feed, were repeatedly exposed to heat during their industrial use and storage, and afterward during the fat melting process. As a result, I-TEQ values of 1:11,373 and 1:15,622 of its content in marker PCBs were found for animal feed (Table 4); the egg samples, in which the PCB mixtures underwent metabolic reactions in the chicken, contained PCB-derived I-TEQs corresponding to respectively 1:11,702 and 1:11,750 of their content in marker PCBs. For chicken, ratios were 1:17,618, 1:16,722, and 1:17,735, and for pork ratios were 1:42,892 and 1:44,651. Taking the I-TEQ content of the contaminating PCBs into account leads to a considerably higher estimate of cancer risk. Using the cancer risk estimate of 1 death in 1 million for a lifetime exposure of 0.006 pg/kg body weight, the PCB exposure of 1 ng/kg per day, equivalent to between 0.088 and 0.022 pg I-TEQ/kg per day leads to a risk of 37-147 cancer deaths in a population of 10 million. However, if the risk estimation of Becher et al. (*15*) for TCDD is applied to the PCB-dependent I-TEQ load, between 286 and 6,776 additional cancer deaths can be expected for the total Belgian population of 10 million.

A combined risk resulting from the combination of both dioxins and PCBs would range between 44 and 8,316 cancer deaths. As the risk figures are based on cancer deaths, they do not include the increasing number of curable cases.

Discussion

Population Exposure

Uncertainties. Although more than 20,000 PCBs and hundreds of dioxin measurements on animal feed, animal fat, and different food items have been performed, there is still an important uncertainty about the extent to which the Belgian population has been exposed to these toxicants. These uncertainties derive essentially from the fact that almost no measurements of body burdens before the crisis are available and that until now no measurements of body burdens during or after the crisis were performed.

There is also uncertainty about the extent to which consumed food was contaminated, as sampling of animal fat and food items was not performed in a systematic way, but evolved rather haphazardly during the crisis in response to many different needs and pressures, some from national or European regulatory authorities, and others were commercial in nature. Some of these demands have biased sampling to the more suspect items, others to less suspect products.

Further uncertainty about the extent to which the consumed food was contaminated originates from the period of sampling. The results presented in this paper relate to the food available from end of May until August 1999. The contamination episode started, however, in January 1999. During January until the end of May, no systematic sampling of the food chain was performed. Food items

may have been contaminated more often and at higher levels than is evident from the data presented in this paper.

Comparison with other contamination episodes. In the Yusho (Japan 1968) and Yucheng (Taiwan 1979) incidents, 1,700 and 2,000 victims, respectively, ate contaminated rice oil and ingested respectively, 600 mg PCBs (equivalent to 10 mg/kg body weight) plus 3.5 mg PCDFs, and 1,000 mg PCBs (equivalent to 16.6 mg/kg body weight) plus 3.8 mg PCDFs. In the Belgian crisis we estimated that a modal Belgian ingested 1.5 mg PCBs (equivalent to 0.025 mg/kg body weight). In the Seveso accident (Italy), in which the main toxicant involved was TCDD, individuals studied in the different areas were exposed to between 16 and 78 ng TCDD/kg body weight. The Ranch Hand study on U.S. Air Force veterans concerned individuals with an average exposure of 10 ng TCDD/kg body weight. The mean exposure due to the Belgian incident is estimated at approximately 0.5 ng TCDD I-TEQ/kg body weight. In conclusion, exposure during the Belgian incident amounts to only a fraction of that during other episodes, but far more people were involved.

Incident-related and background exposure. The more than 20,000 measurements reported in this paper provide information not only on contamination as a result of the incident but also on background contamination. This structural contamination is probably similar to the situation in other countries of the European Union and might also be similar in other industrialized countries.

Figure 5 shows a peak of measurements with increased PCB concentrations and a PCB:dioxin ratio of approximately 50,000:1 in samples that could be traced back to the feed producers who used ingredients from the incriminated fat-melting company. Figure 5 also shows samples with increased concentrations of PCBs, but with a PCB/dioxin ratio which is clearly different from the 50,000:1 ratio. Because of the stability of the measured dioxin and PCB congeners, these most variable PCB/dioxin ratios point to the existence of other contamination sources, different from the transformer oil. The wide range of dioxin/PCB ratios in contaminated products suggests that many smaller unidentified contamination events occurred, some of which resulted in high levels of contamination. This hypothesis is equally supported by the congener pattern found in the heavily contaminated animal feed samples shown in Figure 6. These PCB profiles are clearly different from the incident-related PCB profile of animal feed.

Analogously, four egg samples, with a PCB content between 1,111 and 1,405 ng/g fat, with no known link to the incident, also showed a PCB pattern that is different from the incident-related PCB profile of eggs. This indicates that in human food, too, high contamination levels are observed independently from the incident.

The joint occurrence of PCBs and dioxins is commonly encountered in thermal processes where precursors and *de novo* synthesis generate PCDDs and PCDFs. Hence one would expect that the PCB incident resulting from the use of discarded transformer oil in recycled fat would be characterized by a nearly constant ratio of PCBs to PCDD/PCDFs. This is observed for the data in Figure 5, which cluster around the PCB/dioxin ratio of 50,000:1. The other points do not coincide with the expected pattern and at higher ratios, PCB levels exceed by far what is expected. In fact, when measurements corresponding to the PCB/dioxin ratios between 25,000 and 100,000 are omitted, an obvious relationship can be observed between the logarithm of the PCB concentration and the logarithm of the PCB/dioxin ratio. This linear regression can be described as $\log_{10}$ PCB = -0.964 + 0.638 □ $\log_{10}$ PCB/dioxin (r^2 = 0.368). These findings point toward a latent contamination by PCB mixtures with a very low dioxin content, clearly different from the thermally degraded transformer oil. This contamination comes on top of the background contamination with environmental dioxins and can be responsible for high PCB levels.

Milk and beef are less contaminated with PCBs and dioxins than pork, chicken, and eggs (Table 3). Although during the incident the European Union used 200 ng PCB/g fat as a guideline for

contaminated food, it is reasonable to consider that food for human consumption should contain < 75 ng marker PCBs and < 1 pg dioxins/g fat. The percentage of samples with dioxin levels above 2 pg/g fat is high. This applies to all samples, as well as to samples unrelated to the contamination incident. These percentages are also higher than the percentages of samples with > 200 ng PCBs/g fat. Dioxins stemming from environmental contamination are probably responsible for background levels up to about 5 pg/g fat. Environmental PCBs probably only rarely lead to contamination levels above 100 ng PCBs/g fat (*17*). These data provide strong support for the hypothesis that, independent from the incident, the use of recycled fats, oils, and animal waste can lead to high PCB levels in human food.

Measures are urgently needed to reduce the overall PCB and dioxin burden for the population. Known sources independent from the incident are fish, imported vegetable products from countries where DDT is still in use (*17*), recycled animal fat, and all kinds of waste fat that might contain mineral oils. Especially the recycled animal fat and the waste containing mineral oils appear to account for PCB levels above 200 ng/g fat in a few percentages of the meat samples, as shown by measurements on export meat that originated from farms that did not obtain animal feed from producers contaminated by the transformer oil (*17*).

Individual Exposure

Significant numbers of Belgians who consumed contaminated products have temporarily increased their intake of PCDDs/PCDFs up to a factor 100 over the WHO guidelines [1-4 pg I-TEQ/person per day, with 1 pg as the limit in the future (*18*)].

In interpreting the chronic toxicity of persistent chemicals, the body burden is important (*19*). Here we estimated that the average incident-related increase in body burden in Belgium was 7% for PCDD/PCDF and 42% for PCBs. However, because of geographic and dietary reasons, it is likely that the PCB/dioxin burden of the crisis was unevenly distributed among the Belgian population. The U.S. Environmental Protection Agency has estimated that dioxin body burdens might be 3-4 times above the average in about 10% of the population. It is therefore likely that for some subpopulations the increase in body burdens and the associated risk has been substantial. It is important to identify these subpopulations to study the long-term effects associated with these levels of body burdens.

Effects

Acute clinical health effects have not been reported during the Belgian incident. In view of the type of contamination, this is not expected for acute effects as ischemic heart disease (*20*), chloracne, or conjunctivitis (*21*). However, it is most likely that this contamination episode will have delayed effects on the health of exposed individuals.

We estimated the stochastic incremental cancer risk associated with the incident to vary between 44 and 8,316 cancer deaths. This is a first estimation based on a simple model in which a sudden increase in body burden was transformed in a lifelong daily exposure. More complex models relate exposure to age, energy intake, sex, the acute nature of the body burden increase, and the subsequent elimination of PCBs and dioxins from the body. Cancer risk estimates based on these exposure parameters and the unit cancer figures proposed by Becher et al. (*15*) result in figures of the same order of magnitude as the ones presented here (*22*). Moreover, huge uncertainties exist because of the wide variation in the reference estimates of cancer risk and a manifest lack of precision in estimating the individual dose received. At present there is no indication of how the toxic load is distributed over different groups in the Belgian population, but the main uncertainty probably concerns the pathogenic potency of the chemicals involved. Different risk factors are reported in the literature, accounting for differences in risk by a factor 250 for PCBs (*23*) and 100 for dioxins (*15,24,25*).

Moreover, noncancer effects in neonates, infants, and children are important. Four groups of effects are known to be influenced at relatively low-dose exposure to PCBs, dioxins, and/or co-contaminants. First, several studies point to a lack of optimal neurologic function (*26-28*). Second, changes in thyroid hormone levels include increased plasma thyroid-stimulating hormone (TSH) levels, lower plasma TSH levels, and lower plasma tetraiodothyronine (T_4) levels. In particular, the higher TSH levels are indicative for hypothyroidism (*29*). Third, changes in T-cell subpopulations and lower monocyte and granulocyte counts in the blood (*30*) have been reported. Fourth, these contaminants may interfere with the vitamin K metabolism associated with a late form of hemorrhagic disease of the newborn (*31*). This late form is due to vitamin K deficiency at the age of 1 month to 1 year (*32*).

Table 5 relates these health effects in babies with concentrations of PCBs and dioxins in breast milk. It is important to compare these concentrations to the levels found in the milk of lactating women in Belgium (mean for dioxins only: 34.4 pg I-TEQ/g milk fat; range: 27.3-43.2 pg I-TEQ/g). These concentrations are higher than the threshold value at which thyroid changes occur. Therefore, if these mothers double or triple their body burden, thyroid hormone and immunologic changes in their babies should be expected. Hemorrhagic disease of the newborn also occurs at concentrations that probably have been reached as a result of the Belgian dioxin incident.

Conclusion

We assessed data until August 1999 (i.e., during the last phase and immediately after the incident). Subsequent data became available meanwhile. These will be assessed and compared with the current conclusions in a follow-up paper, which will also address issues of latent PCB contamination.

When dioxin-like PCBs are included, Dutch breast milk samples collected in 1990-1991 contained a mean concentration of 72.3 pg I-TEQ/g milk fat (sum of dioxins and dioxin-like PCBs). This corresponds to a body burden of about 14.5 ng I-TEQ/kg body weight (*18*). These levels are of the same order as those causing morbidity in animals (*10*). Furthermore, prenatal exposure or postnatal exposure through breast milk to background concentrations of PCBs and dioxins, as present in a sizeable portion of the Dutch population, was associated with adverse health effects in the children of healthy Dutch women with a normal pregnancy outcome (*33*). Therefore, it is most likely that dioxin and PCB concentrations as they occur in industrialized countries such as Belgium are an important issue in public health.

The Belgian "dioxin crisis," which probably entailed a higher exposure to TEQ through its PCB content (estimated value equivalent to 0.9 g I-TEQ) than through its PCDDs/PCDFs (estimated value 300 mg I-TEQ), should be considered a potentially important public health event. According to our estimations, the incident had a significant impact on the body burden of the modal citizen and has probably doubled or tripled the body burdens of selected subpopulations who were intensely exposed to contaminated food. Dioxin-like compounds are among the reactive or hormone-disturbing substances whose long-term effects may be insidious and particularly hard to detect because of the high background levels. These high background levels should go down. Individuals exposed during the incident to high PCB and dioxin amounts should be traced, and their health status should be monitored. As a precautionary measure, the exposure to these PCBs and dioxins should be decreased by the promotion of chemical and physical hygiene. This form of hygiene is necessary for the primary prevention of cancer and other health problems related to pollution of the environment and the food chain.

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[Table of Contents] [Citation in PubMed] [Related Articles]

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Does exposure to landfill waste harm the fetus?

Perhaps, but more evidence is needed

Papers p 363

In this issue Elliott and colleagues report on the risks to fetuses associated with residence near landfill sites: they compared pregnancy outcomes among British women living within 2 km of any of 9565 landfill sites operational between 1982 and 1997 with outcomes among those who lived at least 2 km away from all known sites (the reference area).[1] They found excess risks for some adverse pregnancy outcomes, a finding consistent with previous literature, but a clear pattern of excess did not emerge and the excesses were small enough (generally less than 10%) that they could be due to study bias, a problem that is difficult to rule out in this type of study. Therefore, the question whether these results represent a causal connection between residential exposures to landfill and adverse outcomes is unresolved.

The authors found that 80% of the British population lives within 2 km of a current or closed landfill site. This remarkable finding has several implications. Firstly, it suggests that even small excess risks near landfill sites would be important in public health terms. Secondly, it raises the question of whether the reference population is unusual, and thus whether the landfill and reference areas were comparable in terms of other risk factors for adverse birth outcomes—for example, poor nutrition. Thirdly, the main study findings relate to the risk of living near any sort of site, while the potential for harm may vary greatly between sites. Higher risks associated with a small number of sites could be lost in the overall comparison.

This large study, based on about eight million pregnancies between 1983 and 1998, was made possible through the use of postcoded national databases such as birth data and the National Congenital Anomaly System. Postcoding provided a means not only of measuring distance of residence from landfill sites but also of addressing comparability of the two populations in terms of other risk factors for adverse outcomes. It was found that 34% of the landfill area and 23% of the reference area were in the most deprived third of the Carstairs deprivation index—a classification of areas based on social class, unemployment, access to a car, and overcrowding. All analyses were adjusted for this difference; however, it cannot be assumed that the three category Carstairs index is an adequate proxy measure for all underlying risk factors. Failure to account for an unmeasured risk factor could have artificially inflated or deflated the relative risks for the landfill versus reference areas. For example, an absolute difference of 10% in the prevalence of a factor that doubles risk could increase or decrease the relative risk for landfill versus reference areas by around 5-9%.

Residence near a landfill site and excess exposure to hazardous chemicals cannot be assumed to be equivalent. There is little published information about the likely exposure concentrations for nearby residents,[2] although local authorities may have carried out risk assessments.[3] Contamination of water or soil may affect a much wider population, and the impact of air dissemination may depend on prevailing winds. Thus, if there is substantial exposure of the population, some of the more exposed people will live more than 2 km away, and some of the less exposed within 2 km. Furthermore, since the study was based on residence at pregnancy outcome, misclassification could occur because women moved home between the critical time window for exposure[4] and the end of pregnancy. Misclassification in terms of exposure or residence would tend to cause underestimation of differences in risk between landfill and reference populations.

Underreporting by district health authorities of anomalies among live born and stillborn children to the National Congenital Anomaly System is well known, and data on terminations of pregnancy for congenital anomaly were available to the study only from 1992. Underreporting might explain why in this study the prevalence of children needing surgical corrections for abdominal wall defects was 40 per 10 000 births whereas the reported prevalence of these anomalies at birth or termination was only 26 per 10 000. The important question here is whether reporting levels for anomalies would have differed systematically between the landfill and reference areas, thereby biasing the results.

Uncertainty about the meaning of epidemiological results, because of potential bias, is not resolved by arguments about statistical significance. In any case, the 99% confidence intervals quoted in the paper are too narrow since there was no allowance for sampling error in the reference population. However, some types of epidemiological comparison offer a stronger basis for inference than others. Evidence of an exposure-response relationship—whereby risk increases with increasing (markers of) exposure—can be convincing. Unfortunately, further classification of the landfill group according to distance from sites (such as <1 km and 1-2 km) was not carried out; the authors felt that inaccuracies in the recorded location of some sites would undermine this. An alternative approach that compared sites licensed to receive hazardous waste.

BMJ 2001;323:351–2

and the remaining sites is probably not helpful in this regard if, as the authors suggest, the former sites were subject to stricter design and management.

A final set of comparisons concerned the area surrounding the 5260 sites that opened in the study period: relative risks for the population of this area compared to the reference area were calculated both before and after the new sites opened. For most outcomes the relative risks (landfill *v* reference) decreased after opening or remained the same. The exceptions were low birth weight and neural tube defects, for which the relative risks increased by 6% and 7% respectively. These comparisons offer an alternative assessment of the influence of landfill sites on risk without entirely solving the problems mentioned above.

It is important that we gain a better understanding of the health impact of different waste management options. Future studies ought to give attention to better estimates of the exposure of residents.

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Improving endothelial vasomotor function

May reduce cardiovascular risk, but the current evidence is circumstantial

The vascular endothelium is a confluent, cellular monolayer that lines the entire vascular compartment at the interface between blood and the vessel wall. This "organ" possesses complex endocrine and paracrine functions and is intimately concerned in controlling vasomotor tone and preventing atherosclerosis and thrombosis.[1] Indeed, endothelial dysfunction plays a key part in the pathogenesis and progression of atherosclerosis.[2]

An important and relatively recently discovered endothelial product is nitric oxide, a simple, highly reactive gas previously known as endothelium-derived relaxing factor. Endothelial nitric oxide itself possesses potent antiatherogenic properties, inhibits platelet aggregation, and regulates vascular tone.[1] Bioavailable nitric oxide may be increased either by enhancing its production or by reducing its inactivation—for example, by reactive oxygen species, which are thought to damage the endothelium and promote atherosclerosis. Indirect measurement of bioavailable nitric oxide, through its vasodilating properties, is an extensively investigated surrogate of endothelial (vasomotor) function in clinical and experimental studies. In this context, endothelial vasomotor dysfunction occurs in the coronary arteries of patients with coronary atherosclerosis[3] and with standard risk factors for atherosclerosis,[4] and more recently it has been associated with the novel risk factors hyperhomocysteinaemia and low birth weight.[5]

Coronary endothelial vasomotor function may be assessed using quantitative angiography to measure vasodilatation induced by agonists (such as acetylcholine) or mechanical stimuli (increased flow) that stimulate the endothelium to produce nitric oxide; impaired function is associated with reduced dilatation. This assessment, although informative, is invasive and potentially hazardous and so not applicable to routine clinical practice. However, coronary endothelial vasomotor dysfunction has been shown to correlate closely with endothelial function measured in large peripheral arteries.[6] Measurement of endothelial function in accessible peripheral vessels, such as the brachial artery, is therefore a useful surrogate of coronary endothelial vasomotor function and can be measured by changes in forearm blood flow induced by nitric oxide releasing agonists (using venous plethysmography) or by flow mediated dilatation (using high resolution ultrasound).

Many studies have shown that endothelial vasomotor dysfunction is reversible with risk factor intervention (such as smoking cessation, physical exercise) and drugs (angiotensin converting enzyme inhibitors, statins, vitamin C, folic acid, fish oils, and spironolactone).[7–10] Until recently, however, we lacked clear evidence of a prognostic link between coronary endothelial vasomotor dysfunction and cardiovascular events. Two recent prospective studies have, for the first time, shown that coronary endothelial vasomotor dysfunction predicts cardiovascular events.[11 12]

Thus, if endothelial vasomotor dysfunction is associated with standard risk factors, can its measurement further improve risk stratification? This question has not been conclusively answered, though data from these prospective studies suggest that it may be more predictive of cardiovascular events than standard risk factors.[11] Furthermore, in people with mild coronary atheroma those with the greatest endothelial vasomotor dysfunction had a worse prognosis than those with mild dysfunction, there being no significant difference in risk factors or disease severity between the groups.[12] The observation that standard risk factor scoring in general practice in the United Kingdom will identify only 59% of men at risk of myocardial infarction or sudden death over a five year period is further evidence that standard risk factor detection will not reveal all those at risk of cardiac events.[13]

At present, clear prospective evidence for benefit, in terms of decreased cardiovascular events, after improving endothelial vasomotor function does not exist, although there is circumstantial evidence to support this link. Several large secondary prevention studies (4S, HOPE, RALES, GISSI Prevenzione study) have shown clear benefit in patients treated with different

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Papers

Risk of adverse birth outcomes in populations living near landfill sites

Editorial by McNamee and Dolk

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Abstract

Top
Abstract
Introduction
Methods
Results
Discussion
References

Objective: To investigate the risk of adverse birth outcomes associated with residence near landfill sites in Great Britain.

Design: Geographical study of risks of adverse birth outcomes in populations living within 2 km of 9565 landfill sites operational at some time between 1982 and 1997 (from a total of 19 196 sites) compared with those living further away.

Setting: Great Britain.

Subjects: Over 8.2 million live births, 43 471 stillbirths, and 124 597 congenital anomalies (including terminations).

Main outcome measures: All congenital anomalies combined, some specific anomalies, and prevalence of low and very low birth weight (<2500 g and <1500 g).

Results: For all anomalies combined, relative risk of residence near landfill sites (all waste types) was 0.92 (99% confidence interval 0.907 to 0.923) unadjusted, and 1.01 (1.005 to 1.023) adjusted for confounders. Adjusted risks were 1.05 (1.01 to 1.10) for neural tube defects, 0.96 (0.93 to 0.99) for cardiovascular defects, 1.07 (1.04 to 1.10) for hypospadias and epispadias (with no

excess of surgical correction), 1.08 (1.01 to 1.15) for abdominal wall defects, 1.19 (1.05 to 1.34) for surgical correction of gastroschisis and exomphalos, and 1.05 (1.047 to 1.055) and 1.04 (1.03 to 1.05) for low and very low birth weight respectively. There was no excess risk of stillbirth. Findings for special (hazardous) waste sites did not differ systematically from those for non-special sites. For some specific anomalies, higher risks were found in the period before opening compared with after opening of a landfill site, especially hospital admissions for abdominal wall defects.

Conclusions: We found small excess risks of congenital anomalies and low and very low birth weight in populations living near landfill sites. No causal mechanisms are available to explain these findings, and alternative explanations include data artefacts and residual confounding. Further studies are needed to help differentiate between the various possibilities.

What is already known on this topic

Various studies have found excess risks of certain congenital anomalies and low birth weight near landfill sites

Risks up to two to three times higher have been reported

These studies have been difficult to interpret because of problems of exposure classification, small sample size, confounding, and reporting bias

What this study adds

Some 80% of the British population lives within 2 km of known landfill sites in Great Britain

By including all landfill sites in the country, we avoided the problem of selective reporting, and maximised statistical power

Although we found excess risks of congenital anomalies and low birth weight near landfill sites in Great Britain, they were smaller than in some other studies

Further work is needed to differentiate potential data artefacts and confounding effects from possible causal associations with landfill

Introduction

Waste disposal by landfill accounts for over 80% of municipal waste in Britain.[1] Human exposure to toxic chemicals in landfill (which include volatile organic compounds, pesticides, solvents, and heavy metals[2-4]) may occur by dispersion of contaminated air or soil,[2] leaching or runoff,[5] or by animals and birds, although evidence for any substantial exposures is largely lacking.[6] Excess risks of congenital anomalies and low birth weight near landfill have been reported,[6-9] including from recent European and UK studies,[10 11] although some have reported less significant[12] or negative findings.[13] The aim of our present study was to examine risk of adverse

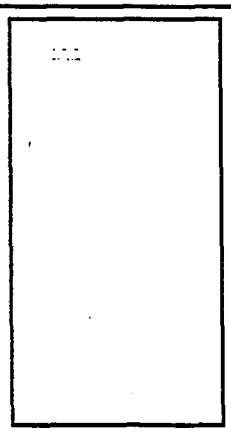
birth outcomes associated with residence near landfill using data on all known sites in Great Britain.

Methods

Top
Abstract
Introduction
• Methods
Results
Discussion
References

Classification of populations near landfill sites

Data provided by the national regulatory agencies were merged in a geographical information system to give a database containing 19 196 sites. Data on boundaries were unavailable for most sites, so point locations had to be used. These comprised the site centroids for 70% of sites and, for the remainder, the location of the site gateway at the time of reporting. Data for site locations were of low accuracy (often rounded to 1000 metres), and data on area were inadequate to allow estimation of the extent of most sites. Landfill sites also change considerably over time as old areas are closed and new areas develop, while postcodes (used to define the location of cases and births) give only an approximation of place of residence, accurate to 10-100 metres in urban areas but >1 km in some rural areas; also, landfill sites are highly clustered, so that individual postcodes may lie close to 30 or more sites. Therefore, distance from nearest landfill site was not regarded as a meaningful proxy for exposure. As a compromise between the need for spatial precision and the limited accuracy of the data, we constructed a 2 km zone around each site (figure), giving resolution similar to or higher than that of previous studies, [10 11] and at the likely limit of dispersion for landfill emissions.[14] Postcodes within the 2 km buffer zone were classified hierarchically by operational status, year on year, such that sites still operating took precedence over those closed earlier in the study period, which took precedence over sites opening later in the study period.[15] People living more than 2 km from all known landfill sites during the study period comprised the reference population.



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Map of Great Britain showing 2 km zones around landfill sites and reference area

Because of concerns about the quality of landfill data for earlier years, and because health data

were available only to 1998, we excluded 9631 sites (25% of the population) that closed before 1982 or opened after 1997 (to allow a one year lag period for the birth outcomes) or for which there were inadequate data. The remaining 9565 sites comprised 774 sites for special (hazardous) waste, 7803 for non-special waste, and 988 handling unknown wastes. The 2 km surrounding these sites included 55% of the national population; 20% were included in the reference area.

Health and denominator data

We used national postcoded registers held by the Small Area Health Statistics Unit. These comprised the National Congenital Anomaly System in England and Wales, 1983-98, and data on terminations, 1992-8, performed for "grounds E" of the 1967 Abortions Act ("where there is a substantial risk that if the child were born it would suffer from such physical or mental abnormality as to be seriously handicapped"); congenital anomaly and terminations data for Scotland, 1988-94; hospital admissions data for England and Scotland, 1993-8 (Welsh data were considered unreliable); and national births and stillbirths data, 1983-98.

Cases were coded to ICD-9 (international classification of diseases, ninth revision) from 1983 to 1994, and to ICD-10 thereafter. Outcomes were all congenital anomalies combined (ICD-9 740-59; ICD-10 Q00-Q99); neural tube defects (ICD-9 740.0-740.2, 741.0-741.9, 742.0; ICD-10 Q00.0-Q00.2, Q05.0-Q05.9, Q01.0-Q01.9); cardiovascular defects (ICD-9 745.0-747.9; ICD-10 Q20.0-Q28.9); abdominal wall defects (ICD-9 756.7; ICD-10 Q79.2-Q79.4); hypospadias and epispadias (ICD-9 752.6; ICD-10 Q54.0-Q54.9, Q64.0); surgical correction of hypospadias and epispadias (M731, M732); and surgical correction of gastroschisis and exomphalos (T281). Multiple anomalies were counted under each outcome (once only for all anomalies combined).

Surgical corrections (England and Scotland only) were analysed by date of birth, not date of surgical procedure. For hypospadias and epispadias, we included only procedures carried out before the age of 3 years, and, for gastroschisis and exomphalos, in the first year of life only. Low and very low birth weights were defined as <2500 g and <1500 g respectively. The relevant denominators and years of analysis are shown in table 1.

View this table: [in this window] [in a new window]	**Table 1.** Denominators and years for analyses of birth outcomes near landfill sites (within 2 km) and in reference area (≥2 km from any site), and before opening and during operation and after closure for sites that opened during the study period

Statistical methods

We calculated risks for the population within 2 km of landfill relative to the reference population by indirect standardisation, assuming a common relative risk for all landfill sites. We used model predictions from Poisson regression of data from the reference area to provide standard rates. The regression function included year of birth, administrative region (n=10), sex (for birth weight and stillbirths), and deprivation. We obtained deprivation by assigning postcodes to tertiles of the national distribution of the Carstairs' deprivation index[16] based on 1991 census statistics at enumeration district level (we used tertiles rather than quintiles of the Carstairs index because of

the small number of events for the rarer outcomes in the most deprived part of the reference area). We used a descending stepwise selection procedure starting from the fullest model including all possible interactions. This was repeated without deprivation, and then the two models were constrained (where necessary) to differ only in terms of deprivation (table 2). For the hospital admissions data (where there were fewer years), unadjusted and deprivation-adjusted results only were obtained, and no modelling was done.

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Table 2. Models chosen by the stepwise selection procedure in the reference area for each outcome*

Some degree of overdispersion and a widening of the confidence intervals is to be expected if our model assumptions fail to hold (for example, because of data anomalies, unmeasured confounding, or sampling variability of the rates). We therefore calculated Poisson 99% (rather than 95%) confidence intervals, but this does not necessarily ensure that all additional variability has been captured—we emphasise estimation of relative risks and their stability (or otherwise) to choice of model confounders rather than significance testing.

We assessed the sensitivity of our results to model choice by using an alternative model for each birth outcome (table 2). We also included urban or rural status and examined risks for rural areas only, and for birth weight (where data were sufficient) we examined sensitivity to the use of quintiles (rather than tertiles) of the Carstairs index. For abdominal wall defects, we also examined maternal age (<20 and ≥20 years, available 1986-98 for England and Wales only).[17]

The main analysis identified at outset was for all landfill sites for the combined period during their operation and after closure. Subsidiary analyses examined risks separately for special and non-special waste sites, and in the period before and after opening for the 5260 landfill sites with available data.[17]

Results

Top
Abstract
Introduction
Methods
Results
Discussion
References

Urban or rural status and Carstairs index were strongly correlated. Within the reference area, 49% of the most affluent tertile of areas was classified as rural (7% for the most deprived tertile), while for all outcomes rates were higher in the most deprived areas compared with the most affluent areas: the ratio ranged from 1.02 (surgical correction of hypospadias and epispadias) to 1.52 (very low birth weight).[17] The area within 2 km of the 9565 landfill sites tended to be more deprived than the reference area: 34% (*v* 23%) of the population were in the most deprived tertile of Carstairs score (36% for special waste sites). The area near landfill also had a higher proportion of births to mothers under 20 years of age (7.7% *v* 6.1%) and, among women aged 15-44, included (1991 census) a higher proportion of women of Indian, Pakistani, or Bangladeshi

origin (4.8% *v* 3.2%) and a lower proportion of black women (2.0% *v* 3.4%).

Table 3 shows the numbers of cases for each birth outcome and relative risks for the area near landfill compared with the reference area. The relative risk for all congenital anomalies combined was 0.92 (99% confidence interval 0.907 to 0.923) unadjusted, and 1.01 (1.005 to 1.023) adjusted for deprivation and other confounders. After adjustment for deprivation (which reduced excess risks) relative risk was 1.05 (1.01 to 1.10) for neural tube defects, 1.08 (1.01 to 1.15) for abdominal wall defects (and 1.07 (0.98 to 1.18) for hospital admissions), 1.19 (1.05 to 1.34) for surgical correction of gastroschisis and exomphalos, and 1.05 (1.047 to 1.055) and 1.04 (1.03 to 1.05) for low and very low birth weight respectively. The risk was 0.96 (0.93 to 0.99) for cardiovascular defects and 1.07(1.04 to 1.10) and 0.96 (0.90 to 1.02), respectively, for hypospadias and epispadias and their surgical correction (for which deprivation adjustment had little or no effect).

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Table 3. Risks of congenital anomalies, stillbirths, and low and very low birth weight in populations living within 2 km of a landfill site (all waste types) during operation or after closure compared with those in the reference area (≥2 km from any site)

Table 4 summarises findings (adjusted for deprivation) for the special and non-special waste sites, and for the sites that opened during the study period. For special waste sites, risks above one were found for all but two outcomes, ranging up to 1.11 (1.03 to 1.21) for cardiovascular defects and for hypospadias and epispadias. For the specific anomalies, except neural tube and cardiovascular defects, risks were higher in the period before opening of a landfill site compared with after opening, especially for hospital admissions for abdominal wall defects. For birth weight and stillbirth, risks were higher after opening.

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Table 4. Estimated relative risks (99% confidence intervals) of birth outcomes for populations living within 2 km of a landfill site, adjusted for deprivation and other variables* according to waste type and to operating status for those sites that opened during the study period

Sensitivity analysis showed that the risk estimates were robust to the different models used.[17] Urban or rural status did not materially alter results with deprivation included, though modelling of data for rural areas only (where numbers of cases were much lower than in the main analysis) did reduce risk estimates for neural tube defects and hypospadias and epispadias—relative risks (for all waste types, deprivation adjusted) were 0.99 (0.89 to 1.10) and 1.01 (0.94 to 1.09) respectively. Inclusion of maternal age as a confounder had only a small effect on risk of abdominal wall defects.[17]

Discussion

Top
Abstract
Introduction
Methods
Results
Discussion
References

This is by far the largest study of associations between residence near landfill and adverse birth outcomes. We found a small excess risk of neural tube defects, abdominal wall defects, surgical correction of gastroschisis and exomphalos, low and very low birth weight. Findings for cardiovascular defects and hypospadias and epispadias were inconsistent, and there was no association with stillbirth. By including all landfill sites in Great Britain and using routine data sources, we avoided the possibility of bias from selective reporting [18 19] and maximised statistical power, but problems with data quality and confounding could have led to spurious associations.[20] These merit further discussion.

Exposure classification and data quality issues

In the absence of information on site or geological factors affecting emissions from landfill, we examined data for special waste sites as a proxy for potential hazard. The UK practice of co-disposal of special and non-special wastes (in contrast, for example, with US "superfund" sites[3]) means that most special waste sites handle small volumes of hazardous wastes. They are subject to stricter management and design standards than other UK sites, while hazardous wastes may have been disposed of, unreported, in non-special sites. Thus exposure risks from special waste sites may be no greater than from other sites. Exposures to environmental contamination from sources other than landfill may also be relevant because sites tend to be located in old mineral or other excavations, often on old industrial or contaminated land or close to current industrial activities.

A key issue was the possibility of misclassification from use of a 2 km zone to define proximity to landfill sites. However, in view of the low spatial resolution of the landfill data (hundreds of metres) and complex nature of landfill sites, using finer subdivisions of the 2 km zone or distance as a continuous measure to examine proxy dose-response relationships would not yield meaningful results. Misclassification of potential exposure to landfill may also have occurred if mothers moved home during the relevant period after conception.[21]

While the data for births and stillbirths are well recorded, the national congenital anomaly system in England and Wales is known to be incomplete[22] (though we found relative over-reporting in Scotland), and there were marked fluctuations in rates of anomalies over the study period, partly because of coding changes[23] and the dates that the terminations data became available. We adjusted for calendar year to deal with fluctuating rates, but ascertainment artefacts could have biased our results (in either direction) if they were differential with respect to landfill locations. Though we had no reason to suspect that this had occurred, such inconsistencies could explain differences of the order detected in this study. On the other hand, we included data on terminations to improve ascertainment, especially for neural tube defects, and included data on hospital admissions and surgical corrections to give an independent source of data for those specific anomalies.

Confounding

We addressed confounding in two ways. Firstly, analysis included potential confounders, with and without adjustment for deprivation. Residual confounding may persist if the adjustment did

not account completely for relevant individual characteristics such as smoking,[24] drug use,[25] and infections during pregnancy.[26] As in the Eurohazcon study,[10] maternal age (for risk of abdominal wall defects[27]) did not seem to be a strong confounder, and, unlike in the United States,[28] location of waste sites near ethnic minority communities was not a key feature. Increased risks (about 1.5 to 2) of low and very low birth weight, [29 30] and (more weakly) of certain congenital anomalies (especially neural tube defects[31]) have been reported among offspring of women of South Asian origin,[32] but the higher proportions of women of Indian, Pakistani, or Bangladeshi origin living near landfill sites compared with the reference area would explain only around 1% excess in our study.

Secondly, we examined rates both before and after the opening of landfill sites that opened during the study period. Because this analysis is restricted to one set of areas, it is less subject to confounding by sociodemographic factors than comparisons between different areas—although confounding by temporal trends (which are strong for some of the health outcomes studied here[17]) is possible. Consequently, we did not compare the risks before and after opening directly but estimated each with respect to the reference region. We found excess risks for some specific anomalies in the period before opening (and which were higher than in the period during operation or after closure, especially for hospital admission for abdominal wall defects). This implies that factors other than landfill might be responsible. The Nant-y-Gwyddon study also noted an excess risk of all congenital anomalies combined before the site was opened.[11]

A possible causal association with landfill should also be considered. Given the large heterogeneity between landfill sites and the likelihood that the effect of any emissions would be greatest close to the sites,[33] causal effects related to particular landfill sites might have been greatly diluted. None the less, we know of no causal mechanism that might explain our findings, and there is considerable uncertainty as to the extent of any possible exposure to chemicals found in landfills.[6] Further understanding of the potential toxicity of landfill emissions and possible exposure pathways is needed in order to help interpret the epidemiological findings.

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Contributors: PE and LJ initiated the project and, with DB and SM, drafted the paper. DB, CdH, CH, and IM performed the analysis of landfill sites. SM, CH, and IM performed the statistical analysis, overseen by JW and SR. TKJ contributed to the epidemiological analysis and interpretation. All authors contributed to and approved the final paper. PE is guarantor for the paper.

Footnotes

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References

Top
Abstract
Introduction
Methods
Results
Discussion
References

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bmj.com, 17 Aug 2001 [Response]

Paternalistic, illogical reassurance
Mitzi Blennerhassett, secretary cancer concern support group, royal college patient liaison group member , *home*
bmj.com, 17 Aug 2001 [Response]

Length of residence.
Gareth Lloyd MD MRCOG, retired
bmj.com, 18 Aug 2001 [Response]

Socioeconomic factors of landfill sites
Dr Geoff Helliwell, Consultant Occupational Physician , *WellWork Ltd.*
bmj.com, 18 Aug 2001 [Response]

Oxygen
Patricia Theisen, Perceptual Specialist , *India*
bmj.com, 18 Aug 2001 [Response]

Alternative presentation of risks from landfill sites
Professor Dick Heller, Professor of Public Health , *Evidence for Population Health Unit, Medical School, University of Manchester*
bmj.com, 20 Aug 2001 [Response]

Related editorials in BMJ:

Does exposure to landfill waste harm the fetus?.
R McNamee and H Dolk
BMJ 2001 323: 351-352. [Full text]

Other related articles in BMJ:

EDITOR'S CHOICE
The passage from the *BMJ* to the *Scunthorpe Daily Echo* or CNN.
BMJ 2001 323: 0. [Full text]

EDITOR'S CHOICE [GP]
The passage from the *BMJ* to the *Scunthorpe Daily Echo* or CNN.
BMJ 2001 323: 0. [Full text]

WEBSITE OF THE WEEK
Website of the week: Landfill sites.
Gavin Yamey
BMJ 2001 323: 406. [Full text]

2001-03-21

Government incineration policy reduced to ashes

Greenpeace today welcomed the report on sustainable waste management by the House of Commons Select Committee on the Environment, Transport and the Regions. The report condemns the Government's policy on waste incineration and raises real doubts about the safety and regulation of the technology.

The report itself says that:
"incineration will never play a major role in truly sustainable waste management" and that "the health effects, which result from an incinerator's emissions, are not yet fully known".

Commenting on the report, Mark Strutt, the Toxics Campaigner at Greenpeace, said:
"This report is a serious body-blow to incineration and the Government's plans to unleash a wave of incinerators across the country. No planning authority will be able to grant planing permission for an incinerator when a respected body such as the Environment Committee concludes that the health effects are unknown. Building incinerators is like playing Russian roulette with people's health."

The Environment Committee report comes on the same day that Greenpeace revealed that of 84 current and proposed incinerator schemes, 48 are in Labour controlled planning authorities and 61 are in Labour held parliamentary constituencies.

Mark Strutt commented:
"Labour is completely isolated as the only mainstream political party which favours incineration despite massive public opposition. It's deluded scheme to fill the country with waste incinerators is creating a political time bomb and voters will ultimately not forgive them."

The Environment Committee also attacked the Environment Agency for poor regulation of incinerators. The Committee concluded that:
"Where recurrent breaches of limit values are found to occur, the operator should be fined. If breaches continue to occur, the plant should be closed down."

Mark Strutt said:
"The Environment Agency has a lamentable record in regulating incinerators. There have been 899 breaches of emission limits since 1996 and no prosecutions. Enough is enough, it is time that the Government abandoned incineration forever and committed itself to an intelligent waste strategy which adopts a reduce, reuse and recycle approach to waste management along with the composting of biodegradable waste."

Further Information:
Contact:
Greenpeace Press Office on 020 7865 8255/6/7/8
Mark Strutt on 020 7865 8226

LATEST PRESS RELEASES

- Trials of GM vines underway in key wine growing regions
- Toxics: Unliver admits to mercury dumping
- BP loses out through declining petrol sales and expanding green fuel technology

Brussels, 14 IX. 2000
PMA/lr/A-5131/D: 16 26

Dear Mr Crowley,

Thank you for your letter of 28 June 2000. My opinion is that in most cases incinerators are not the answer to waste management. The incineration of waste helps reduce the volume of waste to be disposed of. However, the environmental impact of incineration is significant, given that, by incinerating waste, pollutants are only transformed - for example, they are concentrated in the incineration ashes, which in turn must be disposed of.

Some pollutants which are present in waste - such as heavy metals - cannot be destroyed. Furthermore, incineration plants which operate in full respect of air and water emission requirements are by definition extremely expensive equipment. Thus, in order to be profitable, they need to rely on a significant and constant inflow of waste. This may, depending on the specific circumstances, undermine waste minimisation programmes.

The ultimate goal of any local authority should in fact be waste avoidance. This objective has been recognised by the Community Strategy for Waste Management (COM 96(399)), echoed by European Parliament Resolution of 14.11.1996 (A4-0364/96). This Strategy is based, *inter alia*, on the so-called 'hierarchy' of waste management principles (first: prevention, followed in order by reuse, recycling - including composting - incineration and landfilling).

The management of waste nevertheless requires the use of a mix of tools and techniques. In this context, the incineration of certain waste fractions is probably unavoidable, in particular such waste fractions whose generation is necessary and which are difficult to recycle as materials. What is important is to plan carefully the necessary treatment capacities, in order to avoid situations where waste fractions are necessary in order to justify the existence of waste treatment equipment.

I trust that the above is of assistance.

Yours sincerely,

Margot WALLSTRÖM

Zero Waste Update

September 2001, Issue 18

Please pass on

☐ _____
☐ _____
☐ _____
☐ _____

Mission Statement: "To encourage and motivate all sectors of New Zealand society to work towards a target of zero waste."

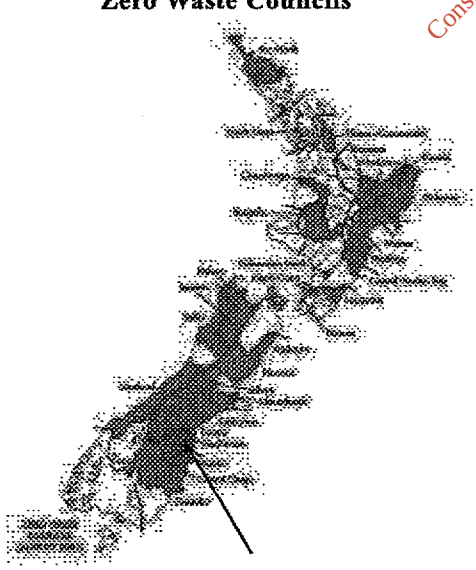
Inside this issue:

Zero Waste Website

www.zerowaste.co.nz

■ A list of NZ's Waste Exchanges is onsite under "Zero Waste Solutions"/"Businesses"

Zero Waste Councils



In August, Waimate District Council adopted a target of Zero Waste by 2015

Our Clean Green Image: What's it really worth?

The Ministry for the Environment investigates the value of conserving New Zealand's Clean Green image. A recently released report commissioned by the Ministry, concludes that underpinning our clean green image with a clean green reality "will not only benefit New Zealanders' quality of life directly, but will have clear long-term economic benefits."

The report states in its 'Assessment of Risks': "The evidence available to date suggests that the generation of waste, including hazardous wastes within New Zealand, continues to pose a threat to the New Zealand environment. It is plausible that this in turn could impact on the perception of environmental quality that underpins New Zealand exports. The sectors most likely to be affected include:

- Tourism, particularly if the quality of waste management is such that the waste stream is allowed to degrade the quality of marine and freshwater resources (including groundwater)
- The agricultural and horticultural sectors (including organics) particularly if the hazardous waste stream results in chemical residues being found in food exports."

The full report, "Valuing our Clean Green Image", is available at www.mfe.govt.nz or by contacting the Ministry on 04 917 7490.

WasteMINZ Conference

Two leading international Zero Waste proponents will attend the 2001 WasteMINZ Conference in Christchurch this year. Dr Dominic Hogg, a leading UK economist, and Graham Mannell, the driving force behind Canberra No Waste by 2010 Strategy, are being sponsored by Zero Waste New Zealand Trust, to share their expertise with the WasteMINZ attendees. Dr Hogg's visit is being co-sponsored by the NZ British Council.

Zero Waste New Zealand Trust is hosting a half day workshop on the first day of the conference, providing an opportunity for Councils and community groups to network and discuss progress, views and ideas. Graham Mannell will join the workshop, to provide insight into Canberra's strategy. Dr Dominic Hogg, Director of Eunomia Research and Consulting, UK, will be providing a keynote speech on the economics of waste minimisation. Other keynote speakers include Brian Richards –New Zealand's well known Brand Strategist, discussing "Repositioning Waste" and Hon Marian Hobbs, outlining how the government intends to meet its commitment to reduce the waste stream.

The WasteMINZ Conference will run from the 31st of October to the 2nd of November. For more information, please contact: WasteMinz Conference 2001, PO Box 31

Updating Buy it Back Guide

Free listing for businesses making products out of recycled materials.

The Buy it Back Guide is currently being updated. This guide is a database of businesses making products out of recycled materials. If your business uses recycled materials to produce products, or if you know of a business currently using recycled materials, please contact Zero Waste New Zealand Trust to be added to the database.

Contact 09 486 0734 or mailbox@zerowaste.co.nz

Zero Waste Council: Kawerau District Council

Kawerau District Council adopted a target of Zero Waste to landfill in 1999, and hopes to reduce waste by 20% this year and 50% in the 2002/2003 year.

To achieve these reductions the council is recommending the introduction of a 50-litre kerbside-recycling bin, a 240-litre wheelie bin for fortnightly greenwaste collections and a 60-litre wheelie bin for other waste, to be introduced in October 2002.

On adopting a Zero Waste target, the Council's first initiative was to employ a person at the landfill to strip car bodies for scrap and spare parts. In January 2001 a drop off centre was set up at the landfill for paper, plastic, glass, metals and cardboard. This is currently being replaced by a Resource Recovery Centre, where staff members will assist the public in the separation of recyclable commodities and resalable items from residual waste. Green waste is being stockpiled and then chipped for use as an energy source for the boilers at the local pulp and paper industry.

In early 2002, a kerbside-recycling scheme will be introduced and the community will be consulted to determine the favoured waste collection system.

The Kawerau community has already undertaken a number of waste reduction initiatives:

* Worm farming was introduced into primary and intermediate schools in the latter part of 2000 and has proven very successful.
* Recycled fashion shows have been organised by the local Soroptomist Club for the last two years.
* A small kitchen industry has also been set up to produce reusable calico bags to replace plastic shopping bags in the area.

For further information, contact Tom McDowell, 07 323 8779 or tom.mcdowall @kaweraudc.govt.nz

Radio Campaign

A radio campaign raises environmental awareness in Timaru.

Timaru District Council is running a radio campaign made up of different environmental messages.

These messages are aired at the same time every week and have been an excellent and cost effective way to increase awareness of environmental issues, reaching up to 15,000 people.

The environmental messages covered topics ranging from recycling and Zero Waste to global warming and resource depletion. The campaign costs Timaru District Council $400 a month, for a weekly message proceeded by six advertisements.

The transcript of this radio campaign is freely available from Timaru District Council, to anybody interested in conducting a similar campaign. Contact Briony Woodnorth on 03 684 8199 or brionyw@timdc.govt.nz

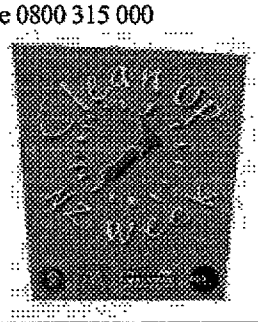
North Shore City Council and Raglan's X-treme Waste have also used radio campaigns to promote their waste reduction initiatives.

Clean Up New Zealand

Clean Up New Zealand Week, 2001, is being held from 17th – 23rd September. Clean Up New Zealand Week brings together schools, families, friends and colleagues to tackle dump sites, polluted streams, littered walk ways and plant native trees.

For the past 12 years the Clean Up the World campaign has mobilised millions of people who have made a positive difference to their local environment and to the planet. New Zealand is just one of over 200 participating countries.

To find out how to get into action, check out the Clean up New Zealand website: www.cleanupnz.org.nz or phone 0800 315 000



Auckland Down-Scales

Auckland City Isthmus residents' 240-litre wheelie bins have been swapped for 120-litre red-lidded wheelie bins, as part of Auckland City Council's strategy to reduce waste by 50% over the next three years.

Auckland City residents currently produce more waste per household than anywhere else in New Zealand.

An innovative new greenwaste recycling scheme has also been set up, called "green.cycle". The 240-litre wheelie bin that was previously used for general waste is being kept on as a greenwaste recycling bin. Through "green.cycle", ratepayers are provided with six rates-funded coupons, which can be redeemed for greenwaste collections, compost and worm bins, or at green waste drop off centres. Auckland City Council has also encouraged the use of recycling bins, allowing each household to order up to three 45 litre bins, for tin, aluminium, glass and plastic grade 1, 2 and 5. A paper recycling collection is also provided.

To assist people to understand the new waste disposal system, Auckland City Council has employed eleven 'Waste Doctors'. These 'Doctors' have been touring the city in a travelling road show, educating people on the changes, and visiting some individual households.

Changes have also been made on the gulf islands, particularly Waiheke which now has official refuse bags and an improved recycling collection service. Changes are also coming for the inner central business district area.

Auckland's new wheelie bin

VCU Launch in Waitakere

In a first for New Zealand, Waitakere City has commissioned a new composting facility to turn its mountains of green waste into organic compost.

The new facility, called a vertical composting unit (VCU), has been installed at the Council's main waste transfer station in Henderson.

The VCU is capable of processing 30 tonnes of green waste into organic compost every day and could reduce the amount of domestic waste going to landfill by up to 52%.

The VCU is an enclosed, self bio-filtering system, which means the composting process does not smell. As well as green waste, the system can process food scraps, paper and cardboard, sewerage, waste cooking oils, and problem waste such as offal and meat scraps.

Current site consents mean Waitakere will process only green waste, although the possibility of handling a wider range of recyclable matter is being considered.

Waitakere City Council is also establishing a resource recovery park at the transfer station, where materials such as timber, tyres and furniture are being recycled on a daily basis. Native timbers are de-nailed, machined and re-sold, and pine timber is de-nailed, tanalised and resold. Currently 14,000 tonnes of timber enters the transfer station each year.

For more information, contact: Mr Peter Higgs, Manager,Perry Waste Services Ltd, Ph: (07) 829 9678, Mobile: (025) 968 200 or

Mr Paul Brown, Director, VCU Technology Ltd, Ph: (09) 571 0266, Mobile: 029 609 543

VCU at Waitakere Transfer Station

BusinessCare Trust

BusinessCare was established as a non-profit trust in December 2000, to promote cleaner production, waste minimisation and sustainable management to businesses nationwide.

Launched with funding from the Ministry for the Environment's Sustainable Management Fund and Zero Waste New Zealand Trust, BusinessCare's aim is 'For cleaner production and complementary programmes to be locally championed by community stakeholders groups all over the country, to help New Zealand companies implement cleaner production as mainstream business practice'.

BusinessCare seeks to provide support and assistance in three ways:

1) Through the establishment of a nationwide network of Local Coordinators whose role is to work with and support local businesses efforts. BusinessCare is developing relationships with local councils nationwide to encourage in-kind support to establish these coordinator positions.

2) Through the provision of Training Programmes focused on the use of Cleaner Production to achieve waste minimisation and implement sustainable management practices. Programmes are being designed both for (a) local coordinators and (b)business managers and staff.

3) Through the collation and dissemination of information on Cleaner Production and business waste minimisation and sustainable management practices. BusinessCare intends to offer a "One Stop Shop" website and database for this information.

Sponsorship recently received from Industry New Zealand is enabling BusinessCare to upgrade their website and online database and also place their newly developed Cleaner Production training online. These changes are due to go live in October. For further infomration contact Peter Crosland, 04 801 9162 or peterc@businesscare.org.nz or visit their website www.businesscare.org.nz

Zero Waste School Award

A set of criteria has been developed to help schools work towards Zero Waste. The criteria, devloped by Zero Waste New Zealand Trust in association with Zero Waste Advisors, outlines the steps a school should take to implement a Zero Waste Strategy and allows a schools to apply for a Zero Waste Award.

The criteria will be available on the Zero Waste website (Under "Zero Waste Solutions"/ "Schools") or by contacting the Zero Waste office (09 486 0734).

The criteria can be used in conjunction with other waste minimisation programmes, and provides schools with the opportunity to outline their success on the Zero Waste website.

Resources

Software for Businesses

Ecology Action in Santa Cruz, California has placed a 30-day trial version of its WasteNot Assessment and Recommendation software on its web site at: http://www.ecoact.org

This software has been used for over 500 Industrial/Commercial/Institutional waste assessments, assisting businesses to reduce waste and measure existing diversion.

International News

Zero Waste in Italy

A 'pay as you throw' scheme it Italy investigated a number of people who were reporting zero residual waste. A survey of families with a particularly low delivery of bags for disposal (16% of total householders), reported some 43% of zero waste cases were "true" or "justified", 29% were due to misbehaving (attempts to avoid waste disposal costs) and another 28% were suspected to be misbehaving.

This means 43% of 16% (6.5% or so) are already 'zero-wasters' in this community in Italy!

Used Nappies get Useful

A Dutch city has started collecting disposable nappies for recycling. The city of Arnhem is asking parents to drop disposable nappies sealed in plastic bags into special bins outside two nurseries and a recycling firm has been hired to extract wood pulp and plastic from the waste. The company (Knowaste BV) will treat waste water and turn sludge into compost during recycling.

Knowaste BV plans eventually to collect Arnhem's annual 200,000kg of nappies from 40 bins around the city, many outside childcare centres.

It is estimated that disposable nappies in landfills could take up to 500 years to decompose. *From NZ Herald, Thursday, August 16, 2001.*

Landfills linked to birth defects

Women living near landfills run a greater risk of giving birth to deformed babies than those who live elsewhere, a British study has shown. It found that 80 per cent of the British population live within 2 km of a landfill and babies of women in those areas have 5 per cent more neural tube defects such as spina bifida, 7 per cent more genital defects in boys and 8 per cent more abdominal wall defects.

The study, based on about eight million pregnancies between 1983 and 1998, is the biggest of its kind done in Britain and has been published in the British Medical Journal. NZ Herald, Monday, August 20, 2001.

Events

EcoFest 2001

Nelson City Council and Tasman District Council have this year joined forces to organise their first Ecofest, Festival of the Environment. It will run over two weeks, from 27 October - 11 November, with a whole range of events, walks, talks, seminars and workshops, on a variety of topics, including Zero Waste.

The aim is to increase environmental awareness and participation.

There will also be two EcoExpos, in Takaka and Nelson, which will be a one-stop shop of stalls and displays of all things environmental. If you want more information about the Ecofest or EcoExpos or want to book a stall, contact Susan Corry, at TDC, (03) 544 3484.

The NZ Association for Environmental Education

is holding a national environmental education conference in Hamilton in January 2002. The conference will bring together environmental educators to discuss, debate and demonstrate the practice and theory of environmental education in New Zealand today. The NZAEE is currently calling for papers to be submitted. If you would like to be involved, or would like further information, please contact NZAEE at

PO Box 9584, Hamilton, phone: 07 838 2442, or email: sixhats@wave.co.nz.

Website: www.nzaee.org.nz

International Ecological Engineering Society (IEES) Conference

on Ecological Engineering for Landscape Services and Products. 25-29 November 2001. Lincoln University, Christchurch http://events.lincoln.ac.nz/iees/default.htm

Lincoln University Environmental Management and Design Seminar Series

* Sept 12th, Ian Luxford, "Energy conservation and efficiency: developing rational policy"
* Sept 26th, Caroline Larkin, "Recycling in rural areas: A process model for substantive design"
* Oct 10th, Rowan Hegglun, "Storytelling and environmental education"
* Oct 17th, John Reid, " Organics for Maori community development"
* Oct 24th, Scott Freeman, "Conservation management in New Zealand: control or care?"
* Oct 31st, Shane Roberts, " Place and sustainability"
* Nov 7th, Fiona Cox, "A Compost Toilet for Rural Samoans: a cost effective and environmentally sound sanitary solution."
* Nov 14th, Andrew Dakers, "EcoSummit 2000 Outcomes: Interdisciplinary projects essential for more sustainable relationships with ecosystem services"
* Nov 21st, Mark McGrath, "EM and an innovative composting toilet"

John Hayward Room in John Burton Building 3.40, Wednesdays. For further information contact Wayne McCallum mccallw@lincoln.ac.nz

==========Electronic Edition==========

RACHEL'S ENVIRONMENT & HEALTH WEEKLY #270

---January 29, 1992---

HEADLINES:

EPA'S DIOXIN REASSESSMENT--PART 2:

DIOXIN DAMAGES HUMAN IMMUNE SYSTEM

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Environmental Research Foundation

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EPA'S DIOXIN REASSESSMENT--PART 2:
DIOXIN DAMAGES HUMAN IMMUNE SYSTEM

U.S. Environmental Protection Agency [EPA] is presently reassessing the dangers of dioxin, one of the most toxic chemicals ever tested on laboratory animals. As a result of animal tests, EPA has declared only exceedingly small amount dioxin "safe" for the human food supply. Americans eat food routinely containing roughly 10 to 100 times more dioxin tha considers safe. [SEE RHWN #269.]

No one makes dioxin intentionally, but many industries creat dioxin as a byproduct of their main activity. Industries tha emit dioxin into the environment (paper, plastics, chemicals solid waste incinerators) are being sued for their emissions citizens claiming harm from exposure, and one industry--paper--faces billions of dollars in lawsuits.

Partly to please the paper industry, and partly because ther were new scientific findings worth considering, EPA chief Wi Reilly announced a year-long dioxin reassessment to begin ir April, 1991.

Now, nine months into the year-long process, EPA scientists responsible for parts of the reassessment have begun talkinc openly about new findings that make dioxin seem as bad as, c worse than, EPA used to think.

For two decades dioxin has baffled toxicologists. They are u to seeing cancer-causing chemicals that predominantly cause cancer in one organ or another--like asbestos, which chiefly affects the lungs, or benzene, which chiefly affects the blood-forming cells, causing leukemia. But dioxin seems to c cancer in many organs, raising the general level of cancer i population without causing a huge increase in any one type c cancer. In addition, dioxin causes certain toxic effects in species and other effects in another species. Likewise, diox low doses causes one kind of illness, and at higher doses it causes different illnesses. Only recently have EPA scientis concluded that this puzzling pattern occurs because dioxin a like an "environmental hormone." Hormones are potent natural chemicals that send messages via the bloodstream, turning or off chemical switches throughout the body, creating an array

effects in different organs. Dioxin behaves this way. Hormc are present in the body in tiny amounts, yet they can trigge huge changes in various bodily systems. For example, it is hormones that trigger the different stages of growth in a fe and that cause young humans to go through puberty.

New Information

EPA chief William Reilly was right--there IS new informatior about dioxin. But it won't be reassuring to the paper indust On the contrary, two studies of workers exposed to dioxin, published during the past year, have shown unmistakable incr in cancers of several types. A study of 5172 American worker revealed a cancer rate 46% above the norm.[1] Likewise, a st of 1583 German workers revealed a cancer rate 39% above the among German workers 20 years on the job, the rate was 82% a the norm, and among the most heavily exposed Germans workers cancer rate was three times the norm.[2] Notably, among fema German workers, the risk of breast cancer was doubled. Wher year ago one might have argued whether dioxin had ever been to cause cancer in humans, now such arguments are only voice the kind of people who say it still isn't proven that cigare cause lung cancer.

Linda Birnbaum, one of the scientists conducting EPA's reassessment of dioxin, says these two studies have convince that dioxin causes cancer in humans, at least at relatively exposures. But, she told SCIENCE NEWS (January 11, 1992, pgs 24-27.), she has an even greater concern about dioxin: "I'm concerned that much lower exposure to dioxin may result in adverse health effects that are very subtle and difficult tc detect." She was talking about dioxin's impact on the immune

system.

The immune system is an exceedingly complex network of organ cells, and chemical secretions (hormones) that react to pres health in the face of a vast array of hostile microorganisms toxicants that our bodies encounter every day. The immune sy fights against common colds, influenza, and the body's own c that go haywire and start to multiply uncontrollably (a definition of cancer).

A degraded immune system leaves the body less able to defend itself against hostile forces in the natural environment. Di attacks the immune system.

EPA's dioxin reassessment will "focus much greater attention toxicological data revealing TCDD's [dioxin's] reproductive, developmental, and immunotoxic effects," says SCIENCE NEWS. Immunotoxic means toxic to the immune system. Furthermore, " document [EPA's draft reassessment] will also establish TCDI the first pollutant to be regulated on the basis of toxicity observed at the cellular level."

This is one reason why the dioxin controversy is being follc so carefully by industry and by environmentalists. It promis set precedents in the way chemicals are regulated in the fut In the past, chemicals were considered harmless if they caus "clinical" damage (damage your family doctor might detect). with the dioxin reassessment, evidence of chemical changes i individual cells is being considered important to a person's being.

"So far, studies in mice suggest that dioxin's immunotoxic p

occurs in extremely low doses and may well be more important cancer in determining dioxin's primary health risk," says Birnbaum."

To study TCDD's toxicity to the immune system, researchers u mice, whose immune systems model those of humans. For exampl EPA researchers have measured how well TCDD-treated mice withstand the influenza virus. Mice pre-treated with TCDD re die after exposure to a quantity of virus that rarely kills healthy mice.

Naturally, it would be very difficult to detect such effects people. If people exposed to unusually high levels of dioxin from a solid waste incinerator, had damaged immune systems a consequently experienced various illnesses, no one might eve suspect dioxin as a cause.

People might question whether some of dioxin's low-level eff represent real harm to people, but "...few people will conte that suppression of the immune system is not an adverse heal effect," Birnbaum told SCIENCE NEWS.

Unlike hormones, which remain in the body only a few hours, dioxin has a half-life in the body of seven years. At the en one half-life, half the initial dioxin remains. What this me is that dioxin has, relatively, a very long half-life in the body, unlike the hormones that it mimics, so it stays around play havoc with the body's chemical systems year after year. "Thus one TCDD [dioxin] molecule can continuously disrupt nc cell physiology," says SCIENCE NEWS, citing work by well-knc dioxin researcher Thomas A. Gasiewicz at the University of Rochester (NY) Medical School. EPA's Birnbaum, and Michael

Holsapple, a well-known di-oxin researcher at the Medical Cc of Virginia, say studies of humans at Times Beach, Missouri, of Vietnam veterans, were essentially bungled. Holsapple say "If I were to take mice and ask the same [research] questior that are routinely asked of the populations of Times Beach c the Ranch Hand study [of Vietnam vets exposed to dioxin-contaminated herbicide], I would come up with a very nebulous picture [of dioxin's immunotoxicity]," says Holsapp "But when we ask different questions [in mice], we can certa show very strong effects on the immune response," he says.

Is there a threshold for dioxin's damage to the human body? there a level of dioxin below which no effects can be observ George Lucier of the National Institute of Environmental Hea Sciences in Research Triangle, North Carolina, has been aski this question in his laboratory. His data show no evidence c threshold. "My data might not prove that a threshold doesn't exist," he told SCIENCE NEWS, "but there's also no evidence any thresholds." In other words, any amount of dioxin does s damage, according to Lucier's findings. This means the only amount is zero.

This conclusion is not what the paper industry wanted to hea when its executives urged William Reilly to initiate EPA's c reassessment. As the reassessment reaches its draft stages e this summer, we'll have new measures of the potency not only dioxin, but also of industry's muscle in a contest with unwe scientific conclusions.

For all of us, much is riding on the outcome.

--Peter Mont

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[1] Marilyn Fingerhut and others, "Cancer Mortality in Worke Exposed to 2,3,7,8-tetrachlorodibenzo-P-dioxin," NEW ENGLANI JOURNAL OF MEDICINE Vol. 324 (1991), pgs. 212-218.

[2] A. Manz and others, "Cancer Mortality Among Workers in Chemical Plant Contaminated With Dioxin," THE LANCET Vol. 33 (October 19, 1991), pgs. 959-964.

Descriptor terms: dioxin; dioxin reassessment; immune system epa; cancer; pulp and paper industry; william reilly;

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Dioxin & Incinerators

The industrial accident at Seveso, Italy, in the summer of 1976, provides the strongest evidence for the carcinogenic (cancer-causing) potential of dioxin. A recent report from the follow-up of the exposed populations around Seveso *(Bertazzi et al, Amer. J. Epi. 2001;153:1031-44)* documents excess deaths from cancers such as Hodgkins disease, non-Hodgkins lymphoma and myeloid leukemia. This report further extends dioxin's toxic potential by associating deaths from endocrine causes (diabetes) and from cardiovascular diseases in both men and women with dioxin exposure.

Exposure to dioxin should now be considered toxic and to be avoided at all costs. The evidence is so compelling that the International Agency for Research on Cancer (IARC) classifies TCDD (dioxin) as a group 1 human carcinogen, based on the following evidence:

(1) 2,3,7,8-TCDD is a multi-site carcinogen in experimental animals that has been shown by several lines of evidence to act through a mechanism involving the Ah receptor;

(2) this receptor is highly conserved in an evolutionary sense and functions the same way in humans as in experimental animals;

(3) TCDD tissue concentrations were similar both in heavily exposed human populations in which an increased overall cancer risk was observed and in rats exposed to carcinogenic dosage regimens in

bioassays. (*Polychlorinated dibenzo-para-dioxins and polychlorinated dibenzofurans. IARC monographs on the evaluation of carcinogenic risk to humans. Vol 69. Lyon, France: International Agency for Research on Cancer, 1997)*

It would not be appropriate to conclude that the risks from dioxin exposure documented in a highly exposed population would not apply at lower doses. Incinerators produce dioxins. No lower dose has been established for dioxin, below which no adverse health effects can be documented. We cannot say that dioxin in small amounts is not harmful. Why risk it at all? Far better to choose environmentally friendly solutions to our waste management problems than to further poison our land and our citizens.

Mise le meas,

Julianne Byrne, PhD

Drogheda

Full Report

2000-10-12

Government figures show Edmonton incinerator responsible for fifteen deaths per year

Government figures from the Department of Health show that current emissions from Edmonton incinerator are expected to result in fifteen deaths every year. The figure will increase to twenty-two if the proposed expansion of Edmonton incinerator goes ahead.

Yesterday, Cllr Rupert Perry (Chair of North London Waste Authority, the co-owners of Edmonton) said on BBC Newsroom South East that the Edmonton incinerator is "very clean" but this view has already been contradicted by Environment Minister Michael Meacher who has said that there is 'no safe threshold' for incinerator emissions.

Stephen Tindale, Policy Director with Greenpeace in the UK, said: "Rupert Perry seems to think that a clean incinerator only kills fifteen people every year but local residents of Edmonton incinerator would disagree. His absurd optimism about Edmonton is even contradicted by the Minister for the Environment. It's time we abandoned hazardous incinerator technology and opted for recycling schemes which are popular with the public and will generate jobs for Londoners."

To read the full press release, containing key facts about Edmonton click on 'Full Report' above. The file is prepared as a PDF (40k)

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Incinerator cancer threat revealed

Dioxins from waste burning and industry far more dangerous than was thought

Sarah Boseley, John Vidal and Julian Borger in Washington
Guardian

Thursday May 18, 2000

Dioxins, the highly toxic chemicals produced by waste incineration and industrial processes which tests have shown to be lingering in the bodies of people all over the planet, have been identified as the cause of many cancers in a new report from the US Environmental Protection Agency.

A draft of the EPA report, leaked yesterday to the Washington Post, has taken the US by surprise and is likely to send shockwaves throughout the rest of the world, forcing an upgrade in the assessment of the hazard posed by dioxins. It had been thought that the risk was diminishing because levels of the chemicals in the environment were dropping.

The report will fuel mounting opposition in communities across Britain to a new generation of up to 160 major waste incinerators that the government is expected to encourage over the next 20 years.

Dioxins are chemical compounds unintentionally released by incinerators burning sewage sludge and household, hazardous and medical waste. They are also released in industrial processes such as steel making.

Among the most poisonous man-made chemicals, they accumulate in fat and milk and work their way up the food chain. Even low-level exposure is known to interfere with the immune, reproductive and endocrine systems. The latter is involved in the secretion of hormones. Dioxins also effect the early growth and development of humans and animals.

By far the greatest dioxin producer in Britain, according to the Environment Agency, is British Steel, whose works at Llanwern, Port Talbot, Scunthorpe and Teesside pump out almost as many dioxins as the next 15 most polluting companies.

That dioxins are already widely present in the environment and food supplies of all industrialised countries is well-known,

but evidence has been slowly accumulating about how widespread and carcinogenic some are. Yesterday's EPA report is remarkably similar to a more low key report from a group of German scientists, which last year concluded that dioxins might be responsible for 12% of human cancers in industrialised countries.

The British government is gradually accepting that dioxins pose real health threats.

In 1994, the Department of Health published a report saying that "despite insufficient evidence for clear causal links", it would be prudent to regard dioxins as possible human carcinogens".

Health hazard

The proposed incineration plants will be needed, it is claimed, to handle the growing mountains of household waste that the EU is banning from landfill sites. In response to the EU directive, the government is expected to announce that by 2020 it will recycle a third of household waste and burn a third.

Some communities are already arguing that these incinerators will pose a health hazard and that money should be spent on more expensive recycling and composting schemes.

Such alternatives, say Friends of the Earth, would be popular, provide more jobs and be easy for people to understand. They say that £250m collected yearly in waste tax could be used to build new recycling centres.

Chris Pilbury, who works with a coalition of 25 community groups in north-east Wales that oppose a massive proposed incinerator and cement kiln expansion scheme near Wrexham, said: "People will not tolerate these risks. Feelings are running high and this report con firms that we are right to to be concerned."

The document, nine years in preparation, says that for those who eat large amounts of fatty meat and dairy produce the risk - on top of any others they may be exposed to - of getting cancer could be as high as one in 100.

Yesterday the EPA said that at least one scientist involved disputed the statistic and that there was a possibility it would be amended before official publication in June. But there will be no dilution of the message of acute concern about dioxins in the report, which for the first time names the most toxic of the group, TCDD (the infamous Agent Orange of Vietnam) as a human carcinogen.

In 1997, the International Agency for Research on Cancer (IARC) categorised TCDD as a "known human carcinogen" after analysing the epidemiological evidence. In 1998, the World Health Organisation decided to slash the safe level for human exposure. Even at the new level of between one and four picograms per kilogram of body weight (a picogram is a millionth of a millionth of a gram) - they were still anxious that "subtle effects may already occur in the general population in developed countries".

Cancer is not the only worry, and other health damage from dioxins has been slightly easier to substantiate. The EPA report will link low-grade dioxin exposure to a variety of problems, including hormonal changes and developmental defects in babies. It states: "It is likely that part of the general population is at, or near, exposure levels where adverse effects can be anticipated."

Risk to babies

Rick Hind, the legislative director for Greenpeace's toxics campaign, which yesterday wrote to the EPA demanding a Dioxin emergency action plan, said: "This means that dioxin levels in the bodies of newborn babies are already at levels that put them at risk of serious illness."

There have been concerns for some time about the high levels of dioxins in human breast milk, although environmental and health groups continue to urge women that the risks do not outweigh the benefits of breastfeeding.

Experts from the Imperial Cancer Research Fund (ICRF) and the Cancer Research Campaign (CRC) in London yesterday agreed that dioxins were a cause of anxiety.

"We know that dioxins are in general highly toxic and can cause cancer," said Tim Key of the ICRF cancer epidemiology unit in Oxford. But more is unknown than known.

"The whole area is full of uncertainty and particularly in relation to cancer," said Lesley Walker of the CRC.

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