GREENPEACE

5 September 2001

Dear Councillor

Re: Health impacts of waste incineration

As you are no doubt aware, there is currently a great deal of controversy over the health impacts of municipal waste incineration. Clearly waste incineration is an unpopular practice with the general public, but some waste disposal companies and trade associations have put considerable resources into promoting the alleged benefits of this technique. The most recent contribution to the debate is from the National Society for Clean Air which has published a suite of documents relating to both health impacts and the public acceptability of municipal waste incinerators. This document set is partly financed by organisations associated with waste incineration and argues that small scale facilities can be acceptable if combined with high levels of recycling and composting.

Greenpeace has been very active in the scientific debate over incineration and has made several important contributions to the subject. We therefore felt that on this occasion, and given the high level of interest in the debate, we would circulate some further documentary material to inform future discussions. Consequently, please find enclosed the following documents:

- A recent article from The Lancet[1] concerning an epidemiological study around a municipal waste incinerator along with an associated press cutting
- A recent paper from the Journal [2]of the Air & Waste Management Association which reviews recent epidemiological studies on incineration and health
- A fully referenced Greenpeace response[3] to the National Society for Clean Air report on incineration and health, undertaken on behalf of the NSCA by the Institute for European Environmental Policy (IEEP)
- A briefing on incineration[4] and health based on a recent scientific report[5] by Greenpeace International (www.greenpeace.org/~toxics/ for an electronic version of the full report)

Although these documents come from different sources, they do have two things in common. Firstly, they are all rigorously referenced and constitute a significant scientific contribution to the debate around the health impacts of municipal waste incineration. Secondly, all of the reports highlight significant concerns over the health effects of burning waste and raise very serious questions about the wisdom of pursuing this method of waste treatment.

[1] Link to Lancet article
[2] Link to Hu paper
[3] Link to Exeter rebuttal
[4] Link to GP health briefing
[5] Link to full GP health report

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In the current political situation, where many local authorities are considering the construction of municipal waste incinerators to meet the requirements of the EU landfill directive, it is important that both sides of the debate are heard. Greenpeace will shortly be publishing a practical guide for local authorities which describes how the demands of the landfill directive can be met using existing technologies and without resorting to incineration.

We hope you find the contents of this package interesting and informative and will ensure that the data is placed alongside other contributions to the debate. It would be a tragedy if unwilling communities had incinerators imposed on them when there are clear practical alternatives and the health effects of this technology are a matter of such concern.

Yours sincerely

Head of Toxics Campaign

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Municipal Solid Waste Incineration: Observations on the IEEP Report for the National Society For Clean Air

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Background

In May 2001 the UK National Society for Clean Air published a report entitled "Municipal Solid Waste Incineration: Health Effects, Regulation and Public Communication". The central thesis of this report, written by Andrew Farmer and Peter Hjerp of the Institute for European Environmental Policy (IEEP) has been widely quoted. The authors assert that environmental releases from a modern incinerator are a fraction of those occurring ten years ago. The corollary promoted by the authors, that in consequence relative health impacts consequent from releases from municipal solid waste incinerators are exceedingly low, has also attained a wide currency. This is despite the authors concluding that "…..we cannot discount effects resulting from the small quantities of pollutants emitted by MSW incinerators….". The primary purpose of this report seems to be to allay human health concerns of community residents associated with incinerator operation.

The conclusions (in both cases) are open to considerable question, the subject of this current critique. This argues that the shortcomings and omissions from the IEEP (2001) report render it unusable as a basis for defining a waste management strategy either in whole or in part. In fact, any local authority using the report in this way, on the basis that the IEEP (2001) conclusions are supportable, will find that their position can be immediately undermined by information already published and freely available in the academic literature. It follows that, shouldany future (medium to long term) liabilities be accrued as a result of relying on the IEEP report, it will be impossible to put forward a plausible defense against them. Such liabilities could be substantial.

The significance of any reliance upon the IEEP (2001) conclusions relating to MSW incineration is considerable in the light of the current situation. Local authorities in the UK, faced with a considerable waste management crisis following many years of failing to develop an integrated, sustainable waste management strategy, are looking to MSW incineration as a means of reducing dependence upon landfill. Quite apart from direct environmental considerations arising from the use of incineration technology, there are potential secondary impacts of some considerable importance. The impacts of incineration as a "technology lock in" upon higher elements in a waste management hierarchy are likely to be profound. The burning of compostable waste, of plastics which could be recycled *via* a feedstock route, and of recyclable paper and card is likely seriously to prejudice the recycling of these elements. In addition, the recyclability of other elements of the waste such as steel cans is likely to be compromised. Taken together, the recycling impacts and impacts upon the environment are sound reasons to question thoroughly any waste management strategy based upon incineration as a central technology.

It is of significance that, when confronted by the misgivings and criticisms of a number of parties, the authors of the report have declined to defend their work either verbally or in writing. The reasons given by the NSCA (Brown *pers. comm.)* are trivial. In the main it appears that the authors consider criticisms made to date to constitute a personal attack upon them. For researchers to decline to defend their work in a public debate is highly unusual and inevitably must raise the possibility that they lack the depth of expertise required to enter fully into this debate. An alternative view, of course, is that the authors actually do recognise that their report is in fact deeply flawed and in consequence, indefensible.

The IEEP Report in Context

The IEEP (2001) report is one of at least three recent reports and papers which have set out to examine the impact of MSW incineration upon human health and the environment. It stands alone from the other material published in the literature in largely dismissing potential human health and environmental impacts. It also stands out, however, as a result of its apparently superficial appreciation of the literature base which underpins the overall subject area. The concluding views contrast with many of the conclusions of the wide ranging review conducted recently by the US National Research Council Committee on Health Effects of Incineration (NRC 2000). It contrasts also with the conclusions reached in a review conducted on behalf of Greenpeace International (Allsopp *et al.* 2001) and with the broad conclusions articulated in a review published in the Journal of The Air and Waste Management Association (Hu & Shy , 2001).

The underlying reasons for this divergence of view between the IEEP study and the other studies mentioned probably have their origins in a number of factors. In any such review exercise, the quality and authority of the source materials is of paramount importance. The IEEP report cites 34 sources (some wrongly, many from the "grey" literature) in the bibliography. Hu & Shy (2001) cite 28 sources in their short but highly focussed 10-page review based on a literature search. Allsopp *et al.* 2001 cite over three hundred literature resources while The NRC (2000) bibliography makes over six hundred citations. On this basis alone, it is clear that the IEEP report has not reviewed the subject area holistically, but has drawn on a very limited base of the information resources actually available. Accordingly, oversight of the subject area by IEEP (2001) can be best regarded as extremely superficial.

Of what could be regarded as key recent publications, the IEEP (2001) report understandably does not cite Hu & Shy (2001) since their report preceded the publication of this paper by some two months. Less understandably, IEEP (2001) fail entirely to reference the NRC (2000) report. The NRC report preceded the IEEP report by some months. As probably the most extensive review carried out in this subject area to date, omission of this source reference is entirely mystifying. The NRC (2001) review (in common with other reports produced by this body) has been produced by an authoritative committee and has been subjected to extensive peer review.

Allsopp *et al.* (2001) is cited by IEEP (2001). The citation, however, is misrepresentative of the overall content, and implies that the report was only cursorily considered.. This view is strengthened by the fact that Allsopp *et al.* (2001) refer to the NRC (2000) report and clearly identify it as a work of some importance. This would have been obvious to anyone who had thoroughly read the Greenpeace Report. Indeed anyone truly familiar with this subject domain would have identified the NRC (2000) report as a key contribution to the knowledge base via a thorough search of the literature and included it in their consideration of the subject. The fact that IEEP (2001) appear not to have been aware of this suggests that their approach was less than thorough.

In a general context, therefore, the IEEP report can be considered as the weakest of the recent publications on incineration and environmental issues based upon its failure to consider the most recent other publications in the field. This failure, coupled with questionable data abstracted from other reference sources, particularly on the mass balance of dioxins and other toxic chemicals has led to one extremely important consequence: In considering the potential health consequences of incinerators to be negligible, the IEEP is effectively equating absence of evidence of impact with evidence of absence of such impact. This is naïve at best, while at worst it could be construed as fundamentally dishonest in scientific terms. In fact this thematic pervades the report. Not only does it adopt this view in relation to the health impacts but also in relation to likely releases from incineration plant. In short it chooses effectively to interpret the largely non-existent empirical data set on releases from new plant as positive evidence that these are not a problem. This choice once made clear, in addition to being scientifically poor, effectively undermines the whole of the rest of the report.

The IEEP (2001) report, moreover, is grounded generally in risk assessment, without appearing to appreciate the many drawbacks associated with such methodologies (see: Santillo *et al.*2001). Broadly, risk assessments depend upon identifying releases of significance, assessing exposure of the general population/ecosystem and specific subgroups thereof and then using these data to assess the risk of a negative environmental or health impact. If the data used in any of the steps are inadequate, then inevitably the risk assessment is flawed. This includes the wider consideration of the full universe of chemicals released. Moreover, it is commonly impossible to evaluate the validity of the many assumptions regarding *e.g.* chemicals and pathways of concern, exposure and effect concentrations, which are an inevitable part of the conduct of risk assessments. The uncertainties and degree of ignorance which pervade such judgements are central to any understanding and critical appraisal of the assessment conclusions. While the IEEP report acknowledges the existence of uncertainties, the authors fail to appreciate the significance of these in influencing their conclusions.

IEEP (2001) justify an emphasis on the chlorinated dioxins released by incinerators on the basis that Allsopp *et al.* (2001) among others specify this group of chemicals as comprising the main health risk. While it is true that these compounds are important, a premise confirmed by the NRC (2000) report, this

attribution is a clear misrepresentation of the content of the Greenpeace Report. While the dioxins are undoubtedly the best researched of the chemicals released from incinerators, they are not the only ones of significance. In fact Allsopp *et al.* (2001) consider a wide range of metals and organic chemicals to be of potential significance and discuss them in some detail..

The IEEP (2001) report under Section 5.7.2 also contains a number of clear misconceptions and omissions concerning the dioxins and dioxin-like PCBs and the use of TEFs. The most obvious is that while the IEEP consider that the WHO 1998 TEF scheme includes 34 congeners, (unreferenced) the widely cited source reference for this only lists 29. (van den Berg *et al.*1998, see also: Stringer & Johnston 2001). The IEEP source cannot be checked since it is not given but the number 34 appears to be a mistake which, although apparently trivial, speaks volumes about the authors understanding and expertise in this research domain. It is unclear at the time of writing whether similar errors pervade the rest of the body of the report.

Another example of poor understanding of the TEQ issue is furnished by the IEEP (2001) statement that while PCBs are not included in the EU 2000 Directive on Incineration, their TEFs are generally low. The clear implication is that the authors consider that compounds are not of importance toxicologically in relation to incinerator releases. This ignores that fact that PCBs are a significant contributor to overall dioxin and dioxin-like compounds in the general population due to their potentially and actual high concentrations. Moreover, there are no data on quantities of PCBs emitted from the burning of MSW tendered in support of the IEEP view. Finally, the EC & DETR (1999) report cited has been superseded by three more recent EC reports of which the authors should have been aware (EC 2000 a, b& c). While these generally support the assertion that exposure to dioxins and dioxin like PCBs has fallen in some countries, the quoted average trend is far from universal and disguises considerable variation. Moreover, recent reports suggest that the decline may not be continuing in *e.g* Germany and Spain (see: Buchert *et al.*2001) based on ongoing monitoring work. In relation to references in the IEEP (2001) report more generally, a number are cited incorrectly or are untraceable from the information given. This is another telling illustration of lack of attention to detail on the part of the authors.

The overall effect of the IEEP (2001) report's self proclaimed emphasis upon the chlorinated dioxins is to draw attention away from the less well researched toxic materials released from incinerators and thus to subtly unbalance the whole content of the report. This approach also has consequences for the risk assessment process espoused by the authors. Risk assessments for dioxins are generally conducted on a single chemical basis and this inevitably fails to allow for the fact that these chemicals are emitted as mixtures with other chemicals. This is a potentially fatal confounder of the risk assessment process in theoretical terms. No practical means exists of resolving this confounding factor. The failings are illustrated under section 5.9 of IEEP (2001) where the epidemiological studies deemed most relevant considered only dioxins as potential causal agents. The approach taken by the IEEP authors is therefore akin to erecting a straw man. Emphasis upon dioxins as the most significant chemical release , justified by misquoting Allsopp *et al.* (2001) is followed by a consideration of epidemiological studies which fail to draw correlations between dioxin emissions and reported health impacts around incinerators.

Overall, the approach taken by the IEEP is to present data on emissions from incinerators derived on the basis of emission factors. It is important to recognise that these are theoretical, not empirical data. The data are based upon a key assumption that incineration plants will operate within the regulatory limit values, and that emissions per tonne of waste combusted will be consistent across the whole industrial sector. Firstly, there is no basis for the assertion that even the new generation of plant will operate consistently within these limits. Secondly, it is known that releases can vary widely even between installations operating ostensibly under the same conditions. Moreover, substantial numbers of breaches have been identified with currently operating UK incinerators through inspection of the relevant public registers maintained by the Environment Agency for England and Wales (Greenpeace 2001). In addition, the work of de Fré & Wevers (1998) (name spelt wrongly in the IEEP 2001 report text and reference list, together with that of Costner, P.) suggests that the results of dioxin monitoring on a semi-continuous basis give substantially higher values than monitoring conducted on a point basis. Despite being aware of this work, the authors of the IEEP Report fail to accord it any real significance in their analysis and fail to consider the potential impacts upon the release scenarios that they reproduce from other (largely "grey") literature sources.

Admittedly (IEEP 2001) recognise that the release figures that they present are subject to considerable uncertainty, but maintain that the trend data are actually reliable. It is difficult to see how this can be supported in the absence of any empirical data, but nonetheless these estimates are used, in turn, to support the assertion that incinerators are now relatively minor contributors to national and Europe-wide atmospheric releases. The releases to ashes and waters are not considered in detail. In general, however, the emission factor approach will tend to underestimate releases of most chemicals; Allsopp *et al.* (2001) cite research which demonstrates this.

In relation, therefore, to the IEEP (2001) report:

- Overall, it displays a poor appreciation of the importance of the concepts of precaution and sustainability in the formulation of environmental policy, as noted earlier in the text.

- It assumes that new incineration plant releases will be very much lower than old plant, but presents no empirical data in support of this assertion.

- It fails to recognise that many chemicals released by incineration operations are poorly characterised with many remaining unidentified. If a substance is not identified, then its toxicological properties cannot be determined.

- It bases exposure assessment upon the emission factor approach which is known to underestimate releases of chemicals of concern.

- It endorses the application of these theoretically derived estimates in a number a risk assessments reported in the "grey literature"

- It displays an extremely superficial knowledge of the relevant scientific literature.

- The report is poorly and inaccurately referenced, drawing in parts on out of date literature.

- There appear to be numerous errors of fact which indicate a lack of understanding by the authors of the subject area.

- The report does not appear to consider scientific uncertainty and ignorance as legitimate and important factors in environmental decision making.

- The concept of sustainability is largely ignored in the report, despite high relevance to ash disposal amongst other issues.

Taken together, these points go a long way to explaining the difference in the broad conclusions reached by the IEEP (2001) report as compared to the other recent reviews which have been published. Overall, on the basis of this limited reporting of the deficiencies of the report, it would be unwise to consider it either accurate or authoritative or as a basis for the formulation of waste management strategy.

The Other Viewpoints Compared.

a) Allsopp *et al.* (2001)

The report produced for Greenpeace (Allsopp *et al.* 2001) was based initially on an extensive search of the academic literature. As far as could be ascertained, at the conclusion of the search phase, all academic papers of direct relevance to the topic had been identified and obtained. Subsequently. two further papers were published addressing aspects of the incineration/health/environment debate. Hu & Shy (2001) produced a focussed review of epidemiological studies, while Staessen *et al.* (2001) reported on a study of

biomarkers in subject population living in the vicinity of a lead smelter and two incinerators. In addition to the overview of the various studies, concerning health impacts upon workers and the general population, information was provided upon the known substances groups of substances of concern. In relation to the health studies, the detailed findings can be found in the Summary Table in the Executive Summary, as well as in the main body of the report.

The broad conclusions which can be reached from the Allsopp *et al.*(2001) report are as follows:

- The number of studies specifically directed at evaluating human health impacts of incinerators are very few in number relative to the large number of installations in operation and planned for future development.

- Epidemiological studies carried out to date have not been able unequivocally to resolve causality, and have suffered from various confounding factors.

- Findings in different studies have been inconsistent

- Nonetheless, where such studies have been carried out, they provide highly suggestive evidence of negative impact upon human health such that extensive follow up studies are warranted.

- Most studies reported impacts resulting from exposures from older generation plant. Few data are available for new generation installations.

- At the same time, few data exist in the literature to support the assertion that new installations operate to higher standards than old.

Accordingly, on the basis of a precautionary approach in the face of the numerous uncertainties and indeterminacies which exist Allsopp *et al.* (2001) recommended a moratorium on the construction of new incineration plant and the formulation of a waste management strategy based upon the axiomatic principles of reduce, re-use and recycle. It was pointed out that such an approach would also accord with principles of sustainability.

b) Hu and Shy (2001)

This review of health impacts of waste incineration was published in July 2001 and as such would not have been captured by any of the other studies on this subject referred to. The review appears, like that of Allsopp *et al.* (2001), to be based upon an extensive interrogation of literature databases, albeit with a specific focus on epidemiological evidence. In this case Medline was used. The information obtained was broadly comparable to that found by Allsopp *et al.* (2001), although two studies not recovered by these authors were reported. One of these (Schecter *et al* 1995) was also not reported in IEEP (2001) despite being one of the very few studies which suggest that worker exposure to chlorinated dioxins in modern incinerator plant may be lower due to improved ash and slag handling procedures. Equally, the Hu and Shy (2001) review did not consider several papers included by Allsopp *et al.* (2001). Such inconsistencies in data retrieval are not unusual, but it must be noted that, in comparison, the IEEP (2001) provides little evidence of a systematic literature search prior to writing of the report.

The review by Hu and Shy (2001) overall emphasises the considerable uncertainties and limitations in the epidemiological studies reported to date. Risk assessments and case studies were not included. It noted that there were inconsistencies between the findings of the various studies of community residents with results for reproductive effects conflicting. Three studies reviewed reported significant positive correlations with lung cancer incidence and deaths, or laryngeal cancer mortality. Two studies, however, found no such correlation. Incinerator worker studies, on the other hand, showed consistently perturbed urinary and blood biomarkers. Study of cancer risks also showed inconsistencies similar to the community studies between reports.

The review noted the difficulty of evaluation and comparison of inconsistencies between these studies which can be summarised as follows:

- Different exposure pathways for incinerator workers and exposed community residents.
- The studies investigated different types of incinerator or similar types burning different wastes leading to inconsistent release and, hence, exposure profiles.
- Community resident exposures were largely evaluated using an approach which precluded determination of individual, as opposed to broad community, exposure.
- Occupational exposures were generally assessed by job description rather than empirical chemical determination
- Occupational exposures are likely to vary from plant to plant.
- Different end-points were evaluated in various of the different studies and exposure levels were not well defined.

Far from considering these inconsistencies as a justification for complacency, Hu and Shy (2001) conclude as follows.

"There is an increasing trend toward using incineration to manage waste; therefore, more people will be at risk of exposure to incinerator emissions. It is important to investigate the health effects of waste incinerators currently in operation. More descriptive studies, which use existing disease registration data, can be conducted to compare the incidence of cancers, cardiovascular diseases, reproductive outcomes and hospital visits of respiratory diseases in areas with and without an incinerator and also for communities before and after construction of an incinerator. "

The review goes on to call for more in depth evaluation of many of the pollutants emitted from incinerators such as the dioxins, mercury and cadmium, pointing out that the health effects of such emissions have not been extensively investigated. The report concludes that:

"….more hypothesis-testing epidemiologic studies, such as case control studies and cohort studies, are needed to assess the associations between waste incineration and the risk of cancers, cardiovascular diseases, respiratory health and reproductive outcomes among incinerator workers and community residents."

The above interpretation of the existing research data is more in line with Allsopp *et al.* (2001) than with the thinking of IEEP (2001) insofar as it recognises that uncertainty and ignorance should spark further investigation rather than less. Certainly, the review makes no statements to the effect that incineration operations will not cause impacts upon human health. On the contrary, the lack of information in this subject area is highlighted as a specific concern to be addressed.

c) NRC (2000)

As noted above, this particular report was produced by a specialist committee (including C.M.Shy who produced the review above) under the auspices of the United States National Research Council. Although referred to by Allsopp *et al*. (2001), it was not considered by IEEP (2001). As possibly the most extensive and relevant review of the incineration/human health issue to date, it is scarcely creditable that the authors failed to consider it. Consideration of the conclusions reached by the expert committee concerned would have made it unlikely that the IEEP (2001) report would have drawn the conclusions that it did.

The NRC (2000) report, which can be read without subscription or other charges on the NRC website at URL: http://www.nap.edu/catalog/5803.html, contains a number of important conclusions which run

counter to the broad conclusions reached by the IEEP (2001) and confirm the generally incomplete and superficial nature of this latter report. The NRC considered both old (existing) plant and installations designed to operate under Maximum Achievable Control Technology (MACT) *i.e* new incineration plant. The following text considers the US report in comparison with the IEEP (2001) document, drawing on those conclusions considered to be of greatest significance.

Recommendations begin in the Executive Summary of the report where governments and government agencies are enjoined *inter alia* to begin research operations to characterise optimal operating parameters relative to emissions and ash quality. The emissions during start-up, shut-down and upset conditions, when the greatest emissions are expected to occur, are considered particularly important given that NRC (2000) note that emissions are generally measured under steady state conditions. The fact that the NRC (2000) regard this area as requiring research (and hence an area where data are lacking) significantly undermines the credibility of the simplistic IEEP acceptance of incinerator outputs based upon regulatory limits, and hence undermines the whole report.

This is the first of many points made by the NRC (2000) report which throw into question the central assumptions made in the IEEP (2001) document. The NRC further consider that future environmental assessment and management strategies for individual incineration facilities should include a regional-scale framework for assessing chemical dispersion, persistence and potential long-term impacts upon human health. Better material balance information is also required, suggests the NRC. This contrasts with the diametrically opposed IEEP view that only dioxins are of potential importance and that even in these cases the smaller amount will be lost in the greater (Section 5.10). The IEEP presumably considers that material balance data is satisfactorily addressed by the dubious emission factor figures presented in Section 5.

The Executive Summary of the NRC (2000) report also suggests that combined site epidemiologic assessments should be conducted as well as a strengthening of the regulatory regime designed to protect workers, with a particular emphasis on lead, mercury, dioxins and furans. Significantly, the Committee concluded that while compliance with MACT regulations could be expected to improve the exposure profile for local community residents, paradoxically, substantial concerns which exist regarding regional dioxin and furan exposures might not be allayed. This is due to the regulations (*i.e.* new incineration emission standards) not being far reaching enough to address the impact of cumulative emissions on a regional basis. This then further undermines the IEEP (2001) report which considers that, since general population exposure is predominantly through food, inhalation exposures are not likely to contribute much. The simple fact that such chemical releases can enter the food chain and are therefore potentially significant on a regional basis, while recognised in the NRC (2000) and Allsopp *et al.* (2001) report, appears to have eluded the IEEP (2001) authors entirely.

Finally, the NRC (2001) report addresses the need for developing an information base on the socio-economic aspects on the basis of geographical areas likely to be impacted rather than simple jurisdictional boundaries. These considerations are central to a second report produced by the NSCA on the public acceptability of incineration. (NSCA 2001a). The author(s) of this report also appear to be unaware of the NRC (2000) document. Curiously this report bears the same (supposedly unique) ISBN number as the IEEP (2001) report and another report (NSCA 2001b) although both are obviously different documents. This seems to be evidence of further inattention to detail on the part of the publishers.

On the basis of the Executive Summary of the NRC (2000) report, the following points emerge which undermine and wholly discredit the IEEP (2001) report:

- The NRC identifies a need for research of incinerator emissions under off-normal operational circumstances. The IEEP report does not consider this aspect as significant..

- The NRC regards lead, mercury, dioxins and furans as requiring research emphasis

- The NRC recognises the significance of regional impacts of incinerator releases from multiple facilities while the IEEP does not consider this as significant

The IEEP partly developed position that individual incinerators pose little in the way of a health hazard receives scant support in the NRC (2000) document which states (Page 179):

"On the basis of available data a well designed and properly operated incineration facility emits relatively small amounts of these pollutants, contributes little to ambient concentrations and so is not expected to pose a substantial health risk. However such assessments of risks under normal conditions may inadequately characterize the risks or lack of risks because of gaps in and limitations of existing data or techniques used to assess risk, the collective effects of multiple facilities not considered in plant- by -plant risk assessments, potential synergisms in the combined effects of the chemicals to which people are exposed, the possible effect of small increments in exposure on unusually susceptible people, and the potential effects of short-term emission increases due to off-normal operations."

Not one of the qualifiers outlined in the conclusion above is considered in the IEEP (2001) report.

On page 180 the NRC document states:

"The Committee's evaluation was performed based only on emissions under normal operating conditions. Data are not available for levels during off-normal conditions, or the frequency of such conditions. Such information is needed to address whether emissions resulting from off-normal conditions are a concern with respect to possible health effects."

And also on page 180:

"The committee's evaluation of waste incineration and public health has been substantially impaired by the lack of an adequate compilation of the associated ambient concentrations resulting from incinerator emissions. The evaluation was also impaired by the inadequate understanding of the overall contribution of incinerators to pollutants in the total environment and large variables and uncertainties associated with risk-assessment predictions, which in some cases, limit the ability to define risks posed by incinerators."

This paragraph is in stark contrast to the assured comments of the IEEP (2001) on this area of the subject which promote the view that emission factor estimates are sufficiently accurate and that the trend data which they present has considerable evaluative utility.

The NRC Report also conflicts with the IEEP (2001) conclusion that results from epidemiological studies show little or no evidence for health effects for MSW incinerators operating to new (comparable to MACT) standards. Laying aside the observation that no such studies addressing "new" plant have been carried out and that there are therefore no data to support this view, the NRC (2000) evaluation notes (Page 179):

"Epidemiologic studies assessing whether adverse effects actually occurred at individual incinerators have been few and were mostly unable to detect any effects. That result is not surprising, given the small populations available to study; the presence of effect modifiers and potentially confounding factors (such as other exposures and risks in the same communities); the long periods that might be necessary for health effects to be manifested; and the low concentrations (and small increments in background concentrations) of the pollutants of concern. Although such results could mean that adverse health effects are not present, they could also mean that the effects may not be detectable using feasible methods and available data sources."

In other words, the findings from epidemiological studies should not be construed as evidence of absence of impact which as noted earlier, is precisely what IEEP (2001) have done.

Moreover, although the NRC (2000) accept that MACT requirements will have substantially lower emissions and that potential exposures to community residents will be lower as a result leading to lower risks from local impacts of releases under normal operational circumstances, they point out (Page 181):

"It is unlikely whether implementation of MACT will substantially reduce the risks at the regional level posed by persistent environmental pollutants dioxin, lead and mercury."

Further:

"MACT was not designed to protect workers and MACT regulations are unlikely to reduce worker exposures."

These points are illustrated by the information contained in Table 5-8 on Page 166 of the NRC (2000) report. This indicates that even after MACT compliance, in the view of the evaluating committee, although the impacts of emissions of single facilities upon a local population fall largely to minimal levels from substantial or moderate levels of concern, they do not fall to negligible levels except in the case of acidic gases. By contrast, the concern elicited by multiple facilities on broader populations remains substantial in the case of dioxin emissions while for lead, mercury and other metals, concerns only fall to moderate levels. The multiple installation/ broader population impacts are completely omitted by the IEEP (2001) report.

Taken together, the NRC statements describe rather different impact scenarios and sets of uncertainties to the rather optimistic, simplistic IEEP (2001) report.

Conclusions

The IEEP (2001) report must be considered as deeply flawed. As well as containing some factual errors it is based upon a limited information base and has failed to consider key reference and review works on the subject of incineration and human health. It contains a number of contradictory elements and has signally failed to take into account the potential impact of multiple facilities on regional populations as opposd to local community residents.

The approach adopted by the IEEP (2001) is essentially a risk assessment based approach. Unfortunately, the authors of the report have failed to appreciate the significant limitations to the quality of the data used to estimate exposure, and have failed in turn, to recognise that this inevitably fatally compromises the validity of their risk judgements. Their conclusion that incinerators operating to modern standards exert no health impacts remains unsubstantiated and unproven.

In promoting this report, the National Society For Clean Air is acting in a highly irresponsible manner. This report is superficial and ill-informed to the point that decisions made on the basis of its content will represent high risk decisions which may attract substantial long term liabilities.

Accordingly, the NSCA should issue a statement in the form of *corrigenda* or, as a more responsible course of action, withdraw the report from circulation immediately.

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Renal function, cytogenetic measurements, and sexual development in adolescents in relation to environmental pollutants: a feasibility study of biomarkers

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Summary

Background Human exposure to chemicals is normally monitored by measurement of environmental pollutants in external media. We investigated whether biomarkers in adolescents can show exposure to, and health effects of, common environmental pollutants.

Methods We recruited 200 17-year-old adolescents (120 girls) from a rural control area and from two suburbs polluted by a lead smelter and two waste incinerators. We measured biomarkers of exposure and of effect in blood and urine samples, and obtained questionnaire data. School doctors measured testicular volume and staged sexual maturation.

Findings 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 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. Biomarkers of glomerular or tubular renal dysfunction in individuals were positively correlated with blood lead. Biomarkers of DNA damage were positively correlated with urinary metabolites of PAHs (polycyclic aromatic hydrocarbons) and VOCs.

Interpretation Biomarkers can be used to detect environmental exposure to pollutants and measure their biological effects before overt disease develops. Our findings suggest that current environmental standards are insufficient to avoid measurable biological effects.

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Introduction

People worldwide are exposed to many environmental pollutants, which are usually monitored by measurements in air, food, water, soil, or dust. Extrapolation from these data to assess the total internal exposure of human beings or to the possible health effects is uncertain.[1] People are exposed via different routes. Variability between individuals in absorption, distribution, metabolism, and excretion of xenobiotics is huge. Several chemicals can act on the same target organs. Diseases caused by chronic exposure to low concentrations of pollutants might become clinically evident only after a long period of time. Concentrations of pollutants or their metabolites in blood, urine, or tissues show current or lifetime exposure via all routes. Biomarkers of exposure are more directly associated with biomarkers of effects than are measurements of pollutants in external media, and provide better estimates of health risk before onset of disease.[2]

Exposure to chlorinated pesticides has been compared between women aged 50–65 years in rural areas and in suburbs: serum-sample concentrations of pentachlorophenol, lindane, and active p,p'-DDT (dichlorodiphenyl-trichloroethane) and its inactive metabolite p,p'-DDE were significantly higher in rural areas than in suburbs (100 women per area), but the opposite was noted for hexachlorobenzene (Department of Welfare, Health and Equal Opportunities, Ministry of the Flemish Community, Brussels, 2000).

We have therefore investigated whether biomarkers can reveal exposure and early health effects in relation to four main classes of environmental pollutants: heavy metals, polychlorinated biphenyls (PCBs), volatile organic compounds (VOCs), and polycyclic aromatic hydrocarbons (PAHs). We chose 17-year-old adolescents as our target population, because in a society with a life expectancy of more than 74 years, biomarkers in young people show recent exposure, even for cumulative toxins such as heavy metals,[3] polychlorinated biphenyls,[4,5] or dioxins.[4] Moreover, in Belgium, school attendance is compulsory until age 18 years and school doctors routinely examine adolescents. Hence, our study benefited from professional expertise and infrastructure.

Methods

Geographical areas

The suburbs Hoboken and Wilrijk are 11–13 km south-east of the chemical industry in the seaport of Antwerp.[6] We selected them for our study area because they included a large non-ferrous smelter,[7,8] two waste incinerators,[9] a crematory,[9] a printing works, and other various industries. Both suburbs are crossed by motorways that carry over 80 000 vehicles per day.[6] In 1998, the mean air concentrations of benzene, toluene, and ethylbenzene were 3·2, 13·0, and 3·6 μg/m^3, respectively (Vlaamse Milieumaatschappij; Erembodegem, Belgium). The waste incinerators (in Wilrijk) started working in 1971 and 1980. In 1997, they had annual turnovers of 23 000 and 110 000 tonnes,[9] and were shut down because dioxin emissions exceeded recommendations (>2·0 *vs* <0·1 ng toxicity equivalents/m^3).[9] Dioxin concentrations in topsoil samples from 15 sites in a radius of 0·5–3·0 km around the incinerators, ranged from 3·9 to 27·2 ng toxicity equivalents per kg dry weight.[9] Deposition of dioxins was also higher than acceptable in Hoboken (⩾27 *vs* ⩽6·8 pg toxicity equivalents/m^2).[10] Additionally, Hoboken has been polluted by lead since the end of the 19th century.[7,8] In 1997, the lead concentrations in airborne particles ranged from 0·08 to 1·35 μg/m^3, and deposition from 3·3 to 7·2 mg/m^2 (Vlaamse Milieumaatschappij, 1997).

Our control area was the town of Peer and its surroundings. This rural area lies 15–25 km east of the nearest non-ferrous smelters and chemical plants, is not crossed by motorways,[6] and has no large industrial settlements.

Participants

Eligible participants were adolescents (in 1999) who were life-long residents of the control area or the two suburbs. Our study protocol required 100 participants from the two suburbs combined, and 100 controls. In Peer (control area) and in Hoboken (study area), adolescents were enrolled from a large grammar school. Our fieldwork coincided with the school holidays in Wilrijk (study area); we enrolled adolescents from a local examination centre and recruited from only the area (Neerlandwijk) surrounding the main waste incinerator. Most pupils in Peer were girls. We therefore stratified recruitment by sex with the aim of enrolling at least 40% boys from all areas.

The ethics committee of the University of Leuven approved the study. We obtained informed written consent from the parents of participating adolescents.

Procedures

Four trained school doctors recorded medical history, stages of sexual maturation according to Marshall and Tanner,[11,12] and in boys measured testicular volume with Prader's orchidometer.[13] Two doctors examined the teenagers recruited in Peer and two others staged the pupils in Wilrijk and Hoboken.

Nurses used questionnaires to assess lifestyle, use of tobacco and alcohol, food intake, special dietary habits, intake of medicines, and social class of parents.[14] We calculated the amount of animal fat per person from their intake of meat, fish, and dairy products in the year before study, by use of Dutch food composition tables.[15] Regular alcohol intake was defined as a positive answer to the question "do you regularly consume alcohol?", and specification of at least one type of drink containing alcohol in a subsequent question.

To validate our lifestyle questionnaire[14] for teenage smoking habits, we measured participants' urinary concentration of cotinine.[16] About 50 mL of blood and 200 mL of urine were taken from every participant in the morning. Girls were not examined when they were menstruating. Blood samples were spun immediately. Split samples of serum, plasma, whole blood, and urine were stored at 4°C or immediately deep frozen. All tests were done in specialised laboratories that met national and international quality-control standards. Blood samples for cytogenetic tests reached the laboratory within 6 h of withdrawal.

Exposure to heavy metals was estimated from concentrations of lead and cadmium in blood samples, and from urinary excretion of cadmium.[17] We estimated exposure to benzene and toluene (VOCs) from concentrations of their urinary metabolites t,t'-muconic acid[18] and orthocresol,[19] respectively. PAH exposure was estimated by measurement of 1-hydroxypyrene[20] in urine. The dioxin congener 2,3,7,8-tetra-chlorodibenzo-p-dioxin (TCDD)[21,22] is the reference compound for polychlorinated aromatic hydrocarbons (PCAHs), which include dioxins, PCBs, and polychlorinated dibenzofurans. Concentrations are usually expressed in toxicity equivalents relative to toxicity of TCDD. We measured concentrations of congeners 138, 153, and 180 in serum samples as biomarkers of exposure to PCBs.[22] Direct chemical measurement of serum-sample concentrations of dioxins would have required an additional 50 mL of blood. Therefore, we estimated exposure to biologically-active polychlorinated chemicals by the calux assay,[23] which measures in-vitro activation of the aryl hydrocarbon receptor of cultured H4IIE cells by dioxin-like compounds in 2·5 mL of serum.

Cystatin C in serum samples[24] and β_2 microglobulin in alkalinised urine samples[25] were measured to detect early glomerular and tubular renal dysfunction, respectively. DNA damage was assessed from whole-blood samples by comet assay:[26] 50 cells per person were processed and the median proportion of DNA in the tail area was calculated. Chromatid breaks, chromosome breaks, and chromosome aberrations (including gaps) were counted in 100–200 cultured lymphocytes from 100 randomly selected adolescents.[27] Urinary 8-hydroxy-deoxyguanosine[28,29] was measured as a biomarker of the DNA repair response to oxidative stress. Urinary measurements were standardised to 1 mmol of creatinine.

Houses and potential sources of pollution were located by use of the global positioning system, GPS Pathfinder Pro XL (Trimble Navigation Europe; Hampshire, UK). Degrees longitude and latitude (ellipsoid WGS84) were converted into kilometres with the Lambert projection system of Belgium maps. We used SAS/GRAPH mapping software (Cary, NC, USA) and the database of Teleatlas (Gent, Belgium). To protect privacy, we calculated spatial summary statistics for small statistical units, as defined by the National Institute of Statistics (Brussels, Belgium). Mean and maximum daily temperatures, and atmospheric ozone concentrations were obtained from the Royal Meteorological Institute (Brussels, Belgium) and the Vlaamse Milieumaatschappij, respectively. We expressed concentrations of pollutants in molar units,

rather than SI, to allow comparison of the effects of a wide range of pollutants on a similar scale. Conversion factors: cadmium, 1μg=8·897 nmol; lead, 1μg=4·826 nmol; PCB congeners 138 and 153, 1μg=2·771 nmol; PCB congener 180, 1μg=2·530 nmol; t,t'-muconic acid, 1mg=7037 nmol; orthocresol, 1mg=9246 nmol; 1-hydroxypryrene, 1μg=481 pmol. To standarise per mmol creatinine: creatinine, 1g=8·840 mmol; cadmium, 1μg/g=1·006 nmol/mmol; t,t'-muconic acid, 1mg/g=796 nmol/mmol; orthocresol, 1mg/g=1046 nmol/mmol; 1-hydroxypryrene, 1μg/g=518 pmol/mmol.

Statistical analyses

Database management and statistical analyses were done with SAS software (version 6.12). Data that were not normally distributed were log-transformed and described by geometric mean and 95% CI, or by median and IQR.

In the first part of the statistical analysis, we compared unadjusted means and proportions across the three areas with analysis of variance and Fisher's exact test, respectively. We then traced confounders by linear regression for continuous variables or by logistic regression for categorical outcomes. We used stepwise-regression procedures in which we set p=0·05 for the independent variables to enter and to stay in the model. Potentially important covariates were forced into the models irrespective of statistical significance. With allowance for the covariates, we looked for differences across the three areas, by use of analysis of covariance for continuous outcomes and logistic regression for odds ratios. If we found significant geographical differences, we did multiple comparisons between individual areas with Bonferroni's correction of significance levels.

In the final part of our analysis, we calculated dose-effect relations in individuals between biomarkers of exposure and of effect; and dose-response relations between biomarkers of exposure and odds ratio for a disorder, by use of multiple-linear regression and multiple-logistic regression, respectively. Effects sizes and odds ratios with 95% CI were calculated from linear and logistic regression coefficients for a two-fold increase in the biomarker of exposure.

Results

Participants

524 adolescents, born in 1980–83 were eligible. 169 children were excluded: seven because they had not lived all their lives in the study areas, and 162 because the sex quota by area had already been filled. Of 355 invited youngsters, 207 (58%) volunteered to take part. We did not examine seven adolescents: three had recently moved out of the study area, two were unavailable because of illness, and two were away travelling.

The 200 adolescents included 120 girls (60%), none of whom were pregnant. Mean age was slightly but significantly higher in Wilrijk, because these adolescents were examined after the end of the school year. Sex distribution and demographic characteristics did not differ between areas (table 1). In Hoboken, the sample included six descendants of non-European immigrants (one boy and five girls). Exclusion of these children did not alter our results. None of the participants had a part-time job in industry or was grossly obese (BMI >30 kg/m^2).

Background characteristics of the 155 non-participants were similar to participants with respect to: mean age (17·4 *vs* 17·3 years, respectively, p=0·67), sex distribution (105 [68%] *vs* 120 [60%] girls, respectively, p=0·13), and parental social class (low, medium, and high: 44 [28%], 99 [64%], and 12 [8%] *vs* 47 [24%], 129 [65%], and 24 [12%], respectively, p=0·30). Of the non-participants, 97 lived in Peer, 41 in Wilrijk, and 17 in Hoboken. In the suburbs, non-participants and participants lived at similar distances from the lead smelter (1896 *vs* 1993 m, p=0·61) and the largest waste incinerator (1297 *vs* 1376 m, p=0·71).

Proportions of current smokers were similar in control and polluted areas (table 1). Geometric mean concentration of urinary cotinine was higher in 50 smokers than in 150 non-smokers, of whom 81 (54%) were passive smokers (309·2 *vs* 22·7 nmol/mmol creatinine, p=0·0001). Pearson's correlation coefficient

Characteristics	Peer (control group) n=100	Wilrijk (study group) n=42	p* between Wilrijk and Peer	Hoboken (study group) n=58	p* between Hoboken and Peer	p* between Wilrijk and Hoboken	p between all 3 areas
Demographics							
Mean (SD) age (years)	17·2 (0·8)	17·8 (0·8)	<0·0001	17·2 (0·8)	0·91	<0·001	<0·001
Mean (SD) height (cm)							
Girls	166 (6)	165 (8)	0·62	165 (6)	0·53	0·99	0·51
Boys	179 (6)	180 (6)	0·66	177 (8)	0·23	0·15	0·31
Mean (SD) body weight (kg)							
Girls	57·7 (8·1)	57·9 (11·9)	0·93	58·7 (9·8)	0·62	0·76	0·62
Boys	66·2 (10·3)	71·4 (15·2)	0·10	66·8 (10·4)	0·86	0·22	0·63
Mean (SD) BMI (kg/m^2)							
Girls	21·0 (2·5)	21·3 (3)	0·72	21·6 (3·1)	0·37	0·72	0·36
Boys	20·5 (2·4)	21·9 (3)	0·07	21·2 (2·7)	0·37	0·44	0·24
Sociodemographics							
Girls	60 (60%)	21 (50%)	0·47	39 (67%)	0·62	0·14	0·22
Girls on oral contraceptives	21 (35%)	11 (52%)	0·27	17 (44%)	0·68	0·88	0·35
Smokers	23 (23%)	14 (33%)	0·39	13 (22%)	0·93	0·35	0·37
Consume alcohol	50 (50%)	22 (52%)	0·01	15 (26%)	0·005	0·80	0·005
Take vocational education	36 (36%)	16 (38%)	0·62	26 (45%)	0·95	0·57	0·51
Social class of parents							
Workers	31 (31%)	5 (12%)		11 (19%)			
Middle class	60 (60%)	29 (69%)		40 (69%)			
Educated professionals	9 (9%)	8 (19%)	0·05	7 (12%)	0·43	0·80	0·08
Serum-sample lipids							
Mean (SD) total cholesterol (mmol/L)	4·21 (0·74)	4·63 (0·86)	0·003	4·30 (0·73)	0·51	0·03	0·01
Mean (SD) triglycerides (mmol/L)	1·07 (0·46)	1·26 (0·50)	0·03	1·06 (0·50)	0·90	0·04	0·07
Mean (SD) total fat (g/L)	5·19 (1·14)	5·36 (1·24)	0·43	4·80 (1·10)	0·04	0·02	0·01

*Bonferroni's method.

Table 1: **Characteristics of participants**

Biomarkers	Peer (control group) n=100	Wilrijk (study group) n=42	p* between Wilrijk and Peer	Hoboken (study group) n=58	p* between Hoboken and Peer	p* between Wilrijk and Hoboken	p between all 3 areas
Blood							
Lead in blood (nmol/L)†	72·0 (65·0–79·0)	87·0 (75·0–101)	0·04	132 (116–149)	<0·0001	<0·0001	<0·0001
Cadmium in blood (nmol/L)†	3·58 (3·19–4·03)	3·66 (3·06–4·39)	0·84	2·62 (2·24–3·05)	0·002	0·006	0·003
Marker PCBs in serum							
(nmol/L)‡	1·19 (1·10–1·28)	1·48 (1·31–1·67)	0·003	1·19 (1·07–1·32)	0·99	0·007	0·008
(pmol/g fat)‡	234 (217–253)	278 (246–314)	0·02	259 (234–287)	0·14	0·31	0·050
Dioxin-like compounds in serum§							
(TEQ ng/L)‡	0·13 (0·11–0·14)	0·16 (0·13–0·20)	0·09	0·21 (0·17–0·25)	<0·0001	0·06	0·0002
(TEQ pg/g fat)‡	24·9 (21·4–29·0)	29·8 (23·4–38·0)	0·20	45·8 (37·5–56·0)	<0·0001	0·01	<0·0001
Urine (standardised to 1 mmol of creatinine)							
Cadmium (nmol)†	0·14 (0·13–0·15)	0·14 (0·12–0·16)	0·81	0·15 (0·13–0·17)	0·30	0·54	0·570
t,t'-muconic acid (nmol)‖	33·3 (28·3–39·2)	50·0 (37·6–66·0)	0·02	45·8 (35·0–60·0)	0·08	0·72	0·020
Orthocresol (nmol)‖	47·6 (39·5–57·5)	120·5 (87·1–167)	<0·0001	61·6 (45·1–84·1)	0·22	0·01	<0·0001
1-hydroxypyrene (pmol)‖	30·8 (25·1–37·8)	38·5 (26·9–55·2)	0·28	36·2 (25·7–51·1)	0·48	0·83	0·460

Data are geometric mean (95% CI). PCB=polychlorinated biphenyls. TEQ=toxicity equivalents. *Bonferroni's method. †Adjusted for sex and smoking. ‡Adjusted for sex, BMI, weeks of breastfeeding, parental social class, and dietary fat intake. Marker PCBs (sum of congenes 138, 153 and 180) were not measured in one resident of Wilrijk and two of Hoboken. Calux assay results were unavailable in one resident of Wilrijk. §Calux assay[23] measures biologically active dioxin-like compounds. ‖Adjusted for sex, smoking, mean daily temperature, and mean atmospheric ozone concentration in the week before samples were obtained.

Table 2: **Biomarkers of exposure**

between urinary cotinine concentration and number of cigarettes smoked per day was 0·45 (p=0·001). Median daily tobacco consumption was 11 cigarettes (IQR 6–16) in 19 male smokers, and six cigarettes (4–9) in 31 smoking girls. Participants who smoked had higher blood concentrations of cadmium (geometric mean 8·65 *vs* 2·38 nmol/L) and lead (104 *vs* 85 nmol/L), and higher urinary concentrations (standardised to 1 mmol of creatinine) of t,t'-muconic acid (56·1 *vs* 35·5 nmol), orthocresol (84·9 *vs* 56·4 nmol), and 1-hydroxypyrene (59·1 *vs* 28·1 pmol). All other exposure and effect biomarkers, which included those for DNA damage, were similar in smokers and non-smokers.

Among 52 boys and 35 girls who drank alcohol, median intake per week was 11·4 g (IQR 4·3–24·7) and 4·3 g (1·1–7·1), respectively. Smoking and consumption of alcohol were significantly associated (p=0·02). In Hoboken, fewer participants reported regular alcohol intake than in the other areas (15/58 [26%] *vs* 72/142 [51%]).

Reported food intake was similar in all areas. Median servings per months were: 30 (IQR 20–30) for meat, three (1–8) for fish, and 30 (20–60) for dairy products. However, in the rural area (n=100), compared with the two suburbs (n=100), more adolescents consumed locally produced meat (33 *vs* 5%; p=0·001), dairy products (47 *vs* 20%; p=0·001), and vegetables or fruit (39 *vs* 24%; p=0·02). 113 adolescents (57%) had been breastfed for a median of 9 weeks (IQR 6–13); their serum-sample PCBs (sum of congeners 138, 153, and 180) increased by 17% (95% CI 9–27%; p<0·001) per 10 weeks of breastfeeding. Adolescents who reported eating fish on more than 3 days per month (median) had a higher urinary concentration of 1-hydroxypyrene (44·0 *vs* 30·6 pmol per mmol creatinine; p=0·02) than those who did not.

Dietary fat intake was similar in all areas (63·3 g per day [IQR 49·3–75·2, p=0·82]). Serum-sample cholesterol was significantly higher in Wilrijk than in the other areas. Mean concentration of total fat in serum-samples was lowest in Hoboken (table 1).

Meteorological conditions

Adolescents from Peer were investigated from May 20, to June 3, and from Sept 16, to Oct 28, those from Wilrijk Aug 10–31, and those from Hoboken from Nov 9, to Dec 2, 1999. In the week before blood and urine samples were obtained, mean daily temperatures were 13·6 (3·4) °C in Peer, 16·8 (5·2) °C in Wilrijk, and 5·3 (4·5) °C in Hoboken; and mean ozone concentrations in air measured from 10·00 to 18·00 h were 58·1 (23·0) μg/m^3, 52·6 (13·5) μg/m^3, and

Characteristics	Peer (control group) n=100	Wilrijk (study group) n=42	p* between Wilrijk and Peer	Hoboken (study group) n=58	p* between Hoboken and Peer	p* between Wilrijk and Hoboken	p between all 3 areas
Renal function†							
Cystatin-C in serum (mg/L)	0·65 (0·08)	0·63 (0·08)	0·13	0·71 (0·08)	<0·0001	<0·0001	<0·0001
β_2 microglobulin in urine (μg/mmol creatinine)	5·22 (4·59–5·94)	5·30 (4·34–6·48)	0·90	9·09 (7·67–10·8)	<0·0001	<0·0001	<0·0001
Cytogenetic‡							
8-hydroxy-deoxyguanosine (μg/mmol creatinine)	0·44 (0·40–0·48)	0·57 (0·49–0·66)	0·004	0·49 (0·42–0·56)	0·31	0·19	0·01
Comet assay (percentage DNA in the tail)	1·02 (0·44)	1·70 (0·49)	<0·0001	1·01 (0·42)	0·98	<0·0001	<0·0001
Chromatid breaks§	31 (62%)	19 (68%)	0·30	12 (55%)	0·81	0·22	0·28
Chromosome breaks§	23 (46%)	12 (43%)	0·17	11 (50%)	0·08	0·21	0·17
Chromosome aberrations§	36 (72%)	20 (71%)	0·61	18 (82%)	0·74	0·58	0·62
Sexual development							
Left plus right testicular volume (mL)‖	47·3 (6·50)	42·8 (6·70)	0·02	42·1 (6·30)	0·004	0·72	0·005
Boys with genital stage G3–G4¶	3 (8%)	8 (38%)	0·003	0	0·96	0·001	0·003
Girls with breast stage B3–B4¶	6 (10%)	7 (33%)	0·03	8 (21%)	0·10	0·08	0·04
Adolescents with stages G3–G4 or B3–B4¶	9 (9%)	15 (36%)	<0·0001	8 (14%)	0·27	0·01	0·0002

Data are mean (SD), geometric mean (95% CI), or number of participants (% of those examined). *Bonferroni's method. †Adjusted for sex and smoking; β_2 microglobulin was also adjusted for initial urinary pH. ‡Adjusted for sex, smoking, mean atmospheric ozone concentration, and mean daily temperature in the week before blood samples were obtained. §Lymphocytes of 50, 28, and 22 randomly selected adolescents from Peer, Wilrijk, and Hoboken, respectively, were cultured; number of participants who had one or more lymphocytes with a specified chromosomal abnormality are shown. ‖Adjusted for age (no data from one boy of Peer). ¶Two boys from Peer were not staged. Adjusted for age, BMI, parental social class, and use of oral contraceptives (girls).

Table 3: **Biomarkers of effect**

Biomarkers of effect	Related biomarker of exposure	Effect type	Effect size* (95% CI)	p
Renal effects				
Cystatin-C in serum	Lead in blood	% increase	3·6 (1·5 to 5·7)	<0·0001
β_2 microglobulin in urine	Lead in blood	% increase	16·0 (2·7 to 31)	0·02
Cytogenetic effects				
8-hydroxy-deoxyguanosine in urine	Orthocresol in urine	% increase	6·8 (2·3 to 11·5)	0·003
Comet assay (percentage DNA in the tail)	t,t'-muconic acid in urine	% increase	4·3 (−0·70 to 9·3)	0·09
	Orthocresol in urine	% increase	5·3 (1·1 to 9·5)	0·01
	1-hydroxypyrene in urine	% increase	7·0 (3·1 to 10·9)	0·0005
Chromatid breaks	t,t'-muconic acid in urine	Odds ratio	1·74 (1·13 to 2·66)	0·01
	1-hydroxypyrene in urine	Odds ratio	1·58 (1·10 to 2·26)	0·01
Chromosome aberrations	1-hydroxypyrene in urine	Odds ratio	1·56 (1·07 to 2·27)	0·02
Effects on sexual development				
Genital stage G3–G4 in boys	Sum of marker PCBs in serum	Odds ratio	3·80 (0·94 to 8·00)	0·06
Breast stage B3–B4 in girls	Dioxin-like compounds in serum†	Odds ratio	2·26 (1·15 to 4·46)	0·02

For number of participants and factors for which the relations were adjusted, see table 3. *Effect sizes were calculated for a two-fold increase in the biomarker of exposure. †Calux assay.[23]

Table 4: **Dose-effect relations**

15·1 (3·5) μg/m^3, respectively (p<0·001 compared with Hoboken).

Orthocresol and 1-hydroxypyrene concentrations in urine-samples and comet assay results were significantly (p<0·0001) correlated with mean temperature and atmospheric ozone concentration. In single regression analysis, *r* for mean temperature and atmospheric ozone concentration were, respectively, 0·56 and 0·40 for orthocresol, 0·29 and 0·31 for 1-hydroxypyrene, and 0·53 and 0·45 for the comet assay.

Regional differences in biomarkers of exposure

Table 2 shows concentrations of biomarkers of exposure adjusted for various factors. Before and after these adjustments, blood lead concentration was higher in Hoboken than in the control area and in Wilrijk, whereas the opposite was noted for blood cadmium concentrations (table 2). Urinary cadmium concentrations were similar in all areas. Marker PCBs in serum samples were significantly higher in Wilrijk than in Peer. Exposure to dioxin-like compounds was highest in Hoboken. Urinary concentration of t,t'-muconic acid was significantly increased in Wilrijk compared with the control area. Urinary concentration of orthocresol was significantly higher in Wilrijk than Peer and Hoboken (table 2).

Regional differences in biomarkers of effect

Table 3 shows biomarkers of effects adjusted for various factors. Before and after these adjustments, cystatin-C in serum samples and urinary β_2 microglobulin were significantly higher in Hoboken than the other areas (table 3). Urinary concentrations of 8-hydroxy-deoxyguanosine and comet assay results were higher in Wilrijk than Peer. Among 100 randomly selected adolescents, median percentage of cultured lymphocytes with chromatid breaks, chromosome breaks, or chromosome aberrations was 1 (IQR 0–1), 0 (0–1), and 1 (0–2), respectively. The number of adolescents who had one or more cultured lymphocytes with these cytogenetic characteristics was similar in all areas (table 3).

Measurements of sexual development and testicular volume, done by the two school doctors in Peer, did not differ significantly (p values ranged from 0·21 to 0·85). In a separate validation study of the school doctors who had examined the teenagers in Peer and Wilrijk, each examined on the same day ten boys and 12 girls in random order. Mean (SD) age of the teenagers was 16·6 (0·6) years. With the physician who had worked in Wilrijk as a reference, κ coefficients[30] for staging sexual maturity were 0·64 (95% CI 0·27–1·00, p=0·009) and 0·58 (0·23–0·94, p=0·01), and mean differences in estimated testicular volume were −3·0 (4·8) mL (p=0·08) and 0 (3·3) mL (p>0·99), respectively. κ coefficients between 0·40 and 0·75 represent good agreement beyond chance.[30] Although

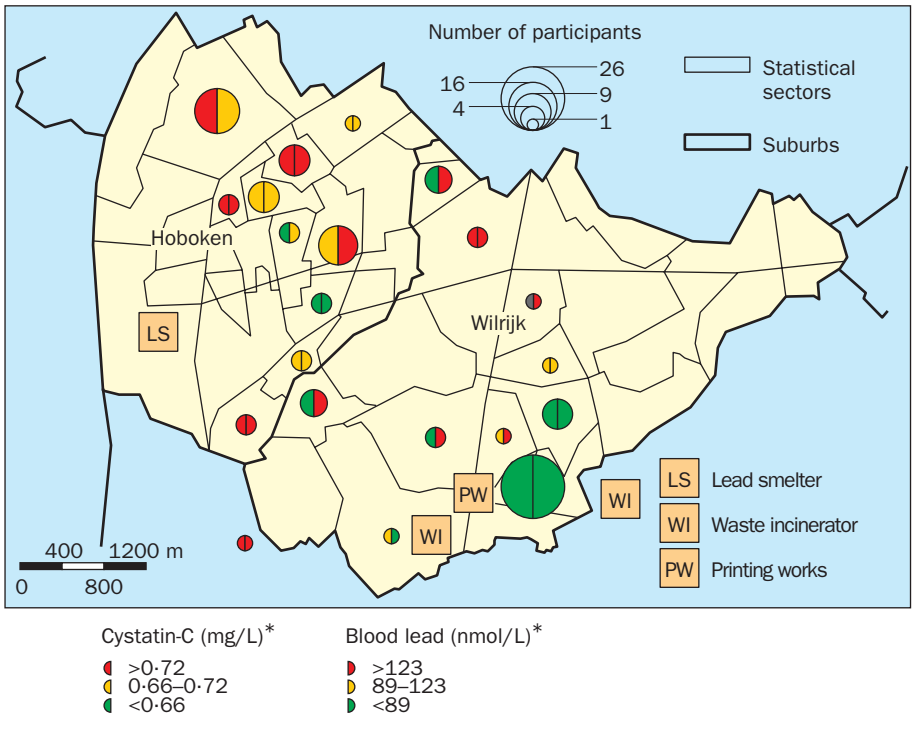

Figure 1: **Location of study-group participants and heavy industry, and concentrations of lead in blood and cystatin-C in serum in Hoboken and Wilrijk**

*Shades from green to red represent increasing levels of biomarkers. The right half of each circle represents the biomarker of exposure and the left half the biomarker of effect.

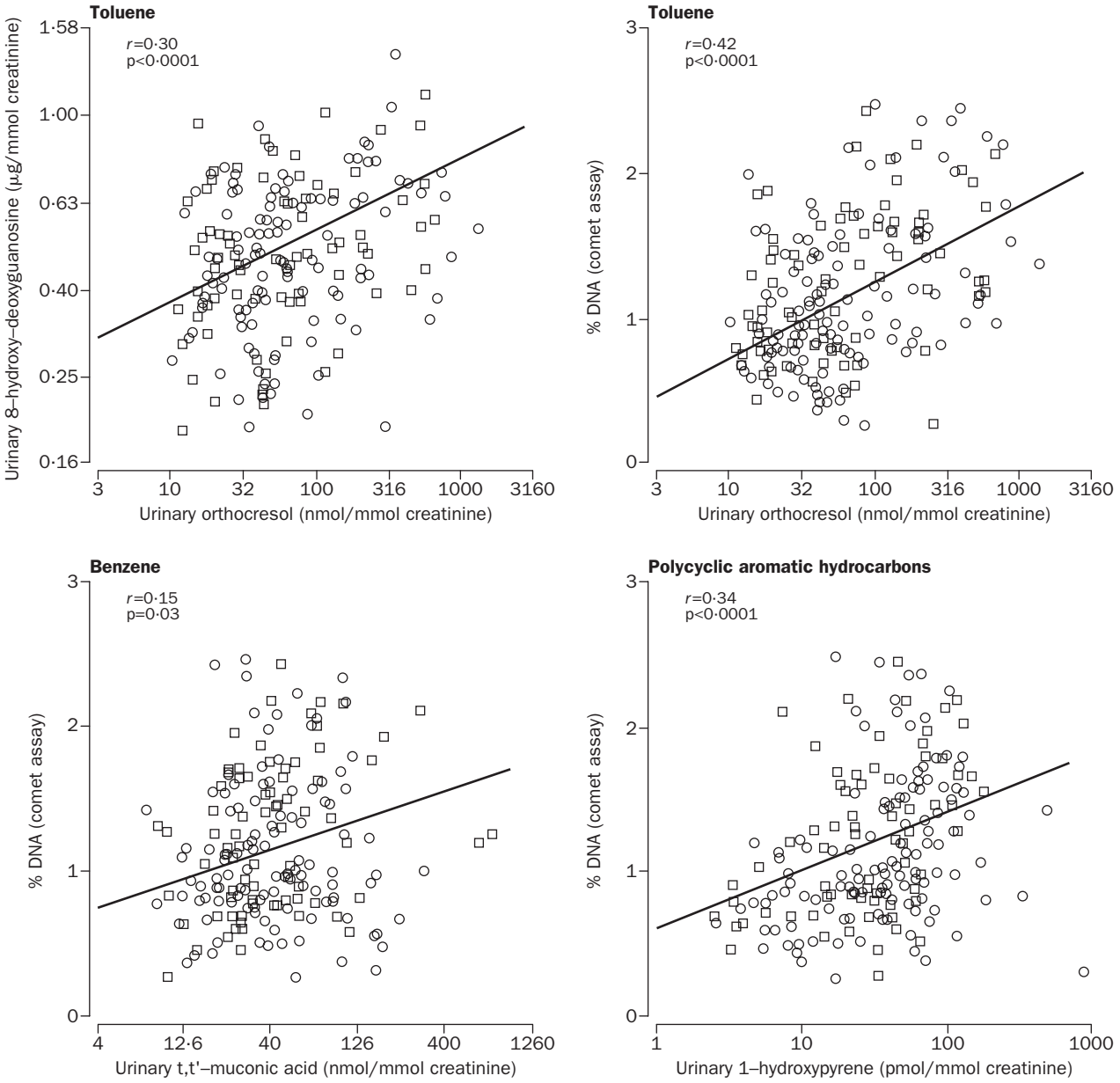

Figure 2: **Unadjusted dose-effect relations in 200 adolescents between two biomarkers of DNA damage,* and urinary biomarkers of exposure to benzene,† toluene,‡ and polycyclic aromatic hydrocarbons§**

Circles and squares indicate girls and boys, respectively. *Urinary 8-hydroxy-deoxyguanosine and % DNA in the tail area in the comet assay. †t,t'-muconic acid. ‡Orthocresol. §1-hydroxypyrene.

participants in Wilrijk were slightly older than those in Peer and Hoboken, more boys and girls had not reached the adult stage of genital or breast development and were rated G3-G4[12] or B3-B4,[11] respectively (table 3). Testicular volume was significantly lower in Hoboken and Wilrijk than in Peer (table 3).

Dose-effect and dose-response curves in individuals

Adjustments applied to calculate dose-effect and dose-response curves in individuals were the same as those used in table 3. Before and after adjustment, cystatin-C and β_2 microglobulin values rose with increasing blood concentration of lead (table 4) but not cadmium ($p>0·27$). Figure 1 shows the association between cystatin-C in serum-samples and the blood-sample lead concentration among adolescents living around the lead smelter in the two suburbs.

Before (figure 2) and after adjustment (table 4), concentrations of 8-hydroxy-deoxyguanosine were significantly correlated with those of orthocresol in urine. Comet assay results were also positively correlated with urinary concentrations of orthocresol and 1-hydroxypyrene. Relative risk of chromatid breaks (logistic regression) rose with higher urinary concentrations of t,t'-muconic acid or 1-hydroxypyrene. Probability of chromosome aberrations rose with increasing 1-hydroxypyrene concentration in urine (table 4). Odds of not having reached adult breast development in girls was positively correlated with estimated concentration of dioxin-like compounds in serum-samples. In boys, probability of less than adult genital development increased with higher serum-sample concentrations of marker PCBs (table 4).

Discussion

In adolescents, biomarkers were sensitive enough to detect significant geographical gradients in common environmental pollutants, in their metabolites, and in their biological effects. Across individual teenagers, dose-effect and dose-response curves were established, which were prespecified in our protocol on the basis of experimental data,[21,22,31] hypotheses,[31–33] or observations mostly made at high levels of occupational[1,18,20,29,34] or accidental[35] exposure to pollutants. We also showed spatial associations between biomarkers and probable sources of present or past pollution.

Our results are unlikely to be confounded by selection bias; participants and non-participants had similar sociodemographic characteristics such as sex, age, and parental social class. Self-selection of more exposed participants than less exposed participants did not occur in the polluted suburbs; participants and non-participants lived at similar distances from the lead smelter and the largest waste incinerator.

By contrast with traditional methods of environmental surveillance, biomonitoring does not require measurement of chemicals in external media. Nevertheless, we also assessed the effect of external factors such as atmospheric conditions and lifestyle on our results. Atmospheric ozone concentration and urinary concentration of metabolites of VOCs and PAHs varied seasonally. Diet affects non-occupational exposure to heavy metals,[3] PCBs,[4,5] dioxins[4] and PAHs.[36] We confirmed the effect of breastfeeding on serum concentrations of PCBs[5] and that of fish intake on urinary excretion of 1-hydroxypyrene.[36] Furthermore, we deliberately included smokers, because 10–20% of older teenagers smoke; cigarette smoke contains many xenobiotics and might increase the harmful effects of various environmental pollutants.[5,36] Cigarette and alcohol consumption were significantly associated.[14] Smokers compared with non-smokers had increased blood concentrations of cadmium and lead, and excreted greater quantities of metabolites of VOCs and PAHs in their urine. None of our adjustments for various factors removed the between-area difference in biomarkers, or the dose-effect or dose-response relations across individuals.

Lead and cadmium accumulate in the human body with age.[1,37] Gastrointestinal absorption and inhalation of contaminated particulate, such as the cadmium-loaded particles in tobacco smoke, are the main routes of exposure. Blood lead concentration was highest in those who lived near the lead smelter.[7,8] All teenagers but one who lived in Hoboken, had a blood lead concentration below 100 μg/L (483 nmol/L). Experts have proposed that in environmentally exposed adults, blood lead concentration should be lower than 250 μg/L (1208 nmol/L).[1]

Cadmium is stored in the kidney from birth. Therefore, its urinary excretion shows life-time exposure.[1,37] The young ages of our participants might be why concentrations of urinary cadmium did not differ between the groups. By contrast, blood cadmium concentration shows recent exposure.[1,37] Cadmium in fertilisers,[38] unidentified point sources, or both, might have caused the blood cadmium concentrations to be higher in the rural control area than in Hoboken. Incineration of waste is an important source of cadmium emissions, because litter frequently includes different types of cadmium-containing products, such as plastics, batteries, or metal scrap.[38] Thus, the incinerators in Wilrijk probably raised the blood cadmium concentrations above those of Hoboken.

In adults[39] and children,[40] environmental lead exposure can affect glomerular[39] and tubular[40] renal function. Serum concentration of cystatin-C is a reliable index of glomerular function.[24] Unlike serum creatinine, this biomarker is independent of sex, age, height, and body composition.[24] In 184 children whose ages ranged from 0·2 to 18·0 years, and of whom 54% had renal impairment, serum cystatin-C averaged 1·75 mg/L.[24] We showed that at a young age glomerular function was independently and negatively correlated with blood lead concentration.

β_2 microglobulin is a circulating microprotein which can pass through the glomerular filter, but is then almost completely reabsorbed in the renal tubules.[1,37] The independent and positive correlation between urinary β_2 microglobulin and blood lead concentration indicates slight some renal tubular dysfunction, and corroborates observations in 13-year-old Czech children living near a lead smelter.[40]

VOCs[1,41] and PAHs[1,36] are common environmental pollutants. Benzene is a constituent of gasoline. Benzene[1,41] and PAHs[1,36] are formed by incomplete combustion of organic matter and fossil fuels (petroleum products, coal, and to a lesser extent wood). They are present in tobacco smoke and car exhaust fumes.[1,36,41] VOCs also originate from organic solvents used in the chemical industry, printing works, or at home. Absorption of VOCs and PAHs occurs mainly through inhalation, and to a lesser extent, through skin contact.[1,41] PAHs present in toast, barbecued food, or contaminated food are gastrointestinally absorbed.[36] Intakes of different food types did not differ between areas, which is probably why urinary concentration of 1-hydroxypyrene also did not vary.

Environmental exposure to toluene was highest in Wilrijk, and benzene exposure in both suburbs combined was higher than in Peer. Traffic or local effluents from point sources (eg, the printing works in Wilrijk) might have caused these findings. Across five studies in Europe, median urinary concentration of 1-hydroxypyrene, standardised to 1 mmol of creatinine, ranged from 80 to 270 pmol in non-smokers and from 170 to 510 pmol in smokers.[42] Urinary excretions of t,t'-muconic acid, orthocresol, and 1-hydroxypyrene that we recorded were far below the reference values for the general population of 398 000 pmol, 314 000 pmol, and 1036 pmol per mmol creatinine, respectively.[1] VOCs and PAHs are potent carcinogens.[1,36,41] 8-hydroxy-deoxyguanosine is formed in response to a specific form of DNA damage induced by reactive oxygen species[43] and is also mutagenic.[43] In workers occupationally exposed to asbestos, rubber, or azo-dye, urinary concentration of 8-hydroxy-deoxyguanosine was 30–80% higher than in controls.[29] In concordance with the biomarkers of exposure to VOCs, concentration of 8-hydroxy-deoxyguanosine in urine-samples and results of the comet assay[26] were highest in Wilrijk.

Furthermore, we also noted an independent and positive relation between urinary excretion of 8-hydroxy-deoxyguanosine and orthocresol. Comet assay[26] results were positively correlated with urinary concentration of orthocresol or 1-hydroxypyrene. Results of logistic regressions also showed an increased risk of chromatid breaks with high urinary concentrations of t,t'-muconic acid and 1-hydroxypyrene, and accorded with the greater risk of chromosome aberrations with high 1-hydroxypyrene concentration in urine. Thus, three independent

measurements of cytogenetic damage, two of which were unrelated to atmospheric conditions (8-hydroxydeoxyguanosine in urine and chromosome abnormalities in cultured lymphocytes), were positively correlated with urinary marker metabolites of VOCs or PAHs.

However, our cytogenetic findings must be interpreted carefully. None of the adolescents had abnormally raised numbers of cultured lymphocytes with chromatid breaks or chromosome abnormalities. The prognostic value of cytogenetic markers in adolescents is unknown. Nonetheless, in a pooled analysis of 3541 Nordic and Italian people (age >15 years), chromosome aberrations in peripheral lymphocytes were a biomarker of the cancer risk, reflecting either early biological effects of genotoxic carcinogens or individual cancer susceptibility.[34]

Dioxins and PCBs are byproducts of many chemical and thermic reactions that contain organic substances and chlorine. They contaminate emissions of waste incinerators and smelt furnaces. PCBs were first produced commercially in the 1920s, although it was not until the 1950s that industrial applications of PCBs increased substantially.[5] They were used as hydraulic or transformer fluids, as plasticisers in paint, and in carbonless copying paper.[5] PCBs have entered the environment and contaminated the food chain, most notably fish.[4,5] PCAHs are common in the environment, although usually present in very small amounts. However, they are lipophilic substances and become biologically magnified in the food chain from soil and sediment to fish or animal feed, to dairy and meat products, and eventually to man.[44] Breastfeeding, as we noted, is an important source of PCB intake.[21,45] Human milk also contains traces of dioxins.[1,45] Absorption of PCAHs occurs via all possible routes, which include inhalation and skin contact.

The calux assay is sensitive to compounds that activate the aryl hydrocarbon receptor, such as dioxins, and coplanar and mono-ortho PCBs.[21,22] We also measured di-ortho PCB congeners 138, 153, and 180, which frequently make up 40–60% of total PCB in human tissue.[22] These di-ortho PCBs have little (congener 138) or no activity mediated via the aryl hydrocarbon receptor.[22] Serum concentrations of dioxin-like compounds and PCBs were highest around the lead smelter and the waste incinerators, respectively, irrespective of whether concentrations were expressed in volumetric units or per g serum fat. At the time of our study the main waste incinerator in Wilrijk was not working. In middle-aged Belgian and Dutch women whose serum was analysed with the calux assay, the median concentrations of dioxin-like compounds were 37·4 and 100·1 pg of toxicity equivalents/g fat, respectively.[46] Marker PCB concentrations in our adolescents were lower than those in cord-blood samples from Düsseldorf, Germany.[31] In 1995, median serum PCB concentrations in the general population of the USA were between 2 and 7 μg/L (about 6–21 nmol/L).[5] These large between-study differences might not only show gradients in environmental exposure, but also differences in participants' diets and lifestyles, and investigators' preparation of biological matrices, handling and cleaning-up of biological samples, and analytical methods. Furthermore, in the more-developed world, exposure to PCBs has fallen since 1971.[5]

PCBs and dioxins accumulate in fat tissue and are endocrine disruptors.[21,22,31,32] PCBs bind to oestrogen receptors and have oestrogenic and antiandrogenic effects.[21,22,31,32] Dioxins and dioxin-like compounds mainly disturb endocrine or cellular function by binding to the aryl hydrocarbon receptor and inducing enzymes involved in the synthesis, intracellular bioactivation, or degradation of hormones.[31,32] In Wilrijk, compared with the other areas, a larger proportion of the adolescents had not yet matured into the adult stages of genital or breast development. Age at which adult genital characteristics are attained varies greatly between individuals. Normative data for Belgium are not available. However, around 1970, British boys reached the adult stage of genital development at a mean (SD) age of 14·9 (1·1) years,[12] and British girls reached the adult stage of breast development at 15·3 (1·8) years.[11] In boys, the probability of slowed genital development rose with higher serum concentrations of marker PCBs. In girls, the probability of slowed breast development was positively correlated with serum concentrations of dioxin-like compounds.

We also noted that testicular volume in boys was lower in the suburbs than in the rural control area. Testicular volume is dependent on the number of Sertoli cells.[33] Follicle stimulating hormone (FSH) causes the multiplication of Sertoli cells during fetal, neonatal, and prepubertal life. FSH secretion is under negative feed-back control of oestrogens produced by Sertoli cells. Multiplication of Sertoli cells stops before puberty. Thus, the main determinants of testicular volume (the number of Sertoli cells) is fixed before puberty.[33] Testicular volume was unrelated to serum concentrations of dioxins and PCBs. Because the two waste incinerators and the lead smelter were in full operation at the time of the boys' birth (1980–83), the smaller testes in the suburbs might have been caused by exposure to xeno-oestrogens in fetal, neonatal, or prepubertal life. Furthermore, xeno-oestrogens might decrease the male to female sex ratio and human fertility because of their sex-linked effects on fetal survival,[44,47,48] and sperm quality.[33] In 1997, a Flemish government report[49] showed that the percentage of medically assisted conceptions was higher around the waste incinerators in Wilrijk than in Flanders, for singleton (5·6 *vs* 3·4%, respectively) and multiple (59·0 *vs* 33·4%, respectively) births. Although prognostic extrapolations are difficult to make from our findings, we note that the number of Sertoli cells and testicular volume correlate with sperm density, and with the total and percentage motile sperm per ejaculate.[50,51]

Young people are very vulnerable to many noxious agents,[52,53] and their protection is an important public health challenge. Feasibility of large-scale and long-term implementation of systematic biomonitoring in adolescents need to be assessed. Because we identified significant effects on sexual development, examination of younger people (aged 14–16 years) might be advisable. Environmental biomonitoring should be part of a health strategy, which could include screening for important cardiovascular risk factors, such as obesity, hypertension, and hypercholesterolaemia, and provide health education. Finally, our findings suggest that present environmental standards are insufficient to avoid measurable biological effects, which might cause disorders in adult life.

Contributors
J A Staessen and H A Roels developed the concept of environmental biomonitoring in adolescents, wrote the initial protocol, and drafted the manual of operations with the help of D Vanderschueren and G Schoeters. J A Staessen and V Nelen organised fieldwork. K Hoppenbrouwers trained the school doctors. G Koppen,

G Schoeters, H A Roels, and L Verschaeve supervised and managed the toxicological measurements. E Den Hond constructed and maintained the database and did the statistical analysis with T Nawrot and L Thijs. E Van Hecke did the spatial analysis and mapped the data. J A Staessen, E Den Hond, T Nawrot, and H A Roels wrote the paper. All authors read and commented on the paper.

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Health Effects of Waste Incineration: A Review of Epidemiologic Studies

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ABSTRACT

There is an increasing trend toward using incineration to solve the problem of waste management; thus, there are concerns about the potential health impact of waste incineration. A critical review of epidemiologic studies will enhance understanding of the potential health effects of waste incineration and will provide important information regarding what needs to be investigated further. This study reviews the epidemiologic research on the potential health impact of waste incineration. Previous studies are discussed and presented according to their study population, incinerator workers or community residents, and health end points. Several studies showed significant associations between waste incineration and lower male-to-female ratio, twinning, lung cancer, laryngeal cancer, ischemic heart disease, urinary mutagens and promutagens, or blood levels of certain organic compounds and heavy metals. Other studies found no significant effects on respiratory symptoms, pulmonary function, twinning, cleft lip and palate, lung cancer, laryngeal cancer, or esophageal cancer. In conclusion, these epidemiologic studies consistently observed higher body levels of some organic chemicals and heavy metals, and no effects on respiratory symptoms or pulmonary function. The findings for cancer and reproductive outcomes were inconsistent. More hypothesis-testing epidemiologic studies are needed to investigate the potential health effects of waste incineration on incinerator workers and community residents.

IMPLICATIONS

For lawmakers and policy-makers, this manuscript provides well-summarized information regarding the potential health effects of waste incinerator emissions on both community residents and incinerator workers. This information will enhance their understanding of what adverse effects have been observed in various populations or workers and what needs further clarification. For environmental epidemiologists or other related researchers, this manuscript aids in determining what is currently known and unknown about the potential health effects of waste incinerators, and what types of studies are needed to investigate the health impact of waste incineration. Based on this information, they can design future studies that address this issue.

INTRODUCTION

Disposal of large quantities of municipal waste has become a serious problem in many U.S. cities because of inadequate landfill space.[1] There is an increasing trend toward using incineration as an alternative means of solving the problem of waste management.[1] The U.S. Environmental Protection Agency predicted that 315 municipal waste incinerators would be in operation by the year 2000.[2] It was estimated that 352 incinerators are used to destroy hazardous waste in the United States.[3] Incinerator emissions are complex, depending on the composition of waste, design of incinerators, combustion condition, and the downstream pollution control equipment.[4,5] Hazardous or municipal waste incineration may emit HCl, SO_x, PM, NO_2, metals, incomplete combustion byproducts, dioxins, and furans.[4,6] There has been substantial local opposition to the construction of waste incinerators because of concern about the potential health and environmental impact.[7]

The purpose of this study is to review previous epidemiologic studies of health effects of waste incineration. These studies comprise two populations—incinerator workers and community residents—and are presented according to their health end points. The

criteria for selection of studies are described as follows: A keyword search mapped to the subject headings for incinerator and incineration, respectively, with an "explode" option was used to identify all incinerator-related studies indexed in the Medline database from 1985 to early 1999 using the Ovid platform (Ovid Technologies, Inc.). We then examined the titles and abstracts and chose all epidemiologic studies of health effects or human body chemical levels in either community residents or incinerator workers. Risk assessment and case reports were not included in the review. Furthermore, we checked the references cited by the reviewed articles to identify as many relevant studies as possible, and only one study was selected from this method.

EPIDEMIOLOGIC STUDIES OF COMMUNITY RESIDENTS

Eleven epidemiologic studies conducted on residents of communities with a waste incinerator are reviewed and listed in Tables 1–3.

Effects on Reproductive Health

Four studies examined reproductive effects (Table 1).[8-11] Lloyd et al.[8] investigated the frequency of twinning in areas exposed to airborne pollution from a municipal waste incinerator and a chemical waste incinerator (the type of waste burned was not specified), and in neighboring control areas. Wind direction was taken into account to differentiate areas potentially exposed to the emissions. The frequency of twinning was increased, particularly after 1979, in the areas at most risk of being exposed to air pollution from the incinerators. The authors speculated that polychlorinated hydrocarbons, some of which have estrogenic properties and have been burned in the chemical incinerator between 1975 and 1977, might be related to increased twinning. In a register study based on information from the central register, Jansson and Voog[9] found no increase in incidence of cleft lip and palate malformations after the start of waste incineration in 18 Swedish boroughs with municipal waste incinerators. In the case study of six children with cleft lip/palate, the subjects lived more than 15 km from an incinerator, whereas the highest dioxin levels were found ~1 km from the incinerator. Although meteorological dispersal calculation was applied to assess the subjects' exposure to dioxin from the incinerator in the case study, borough of residence was used as a surrogate for exposure measurement in the register study.

Table 1. Epidemiologic studies of the reproductive effects of waste incineration on community residents.

Study	Study Subjects/ Type of Incinerator	Exposure Assessment	Outcomes	Results
Lloyd et al., 1988	Residents in areas exposed to a municipal and a chemical WI Control: residents in neighboring areas	By postcode sectors and wind direction	Frequency of twinning in Scotland, 1976–1979 and 1980–1983	Frequency of twinning increased in areas at most risk from the emissions
Jansson and Voog, 1989	Case study (CS): 6 children with cleft lip and palate Register study (RS): 18 boroughs with municipal WIs	CS: meteorological dispersal calculation of dioxin exposure RS: before and after start of WIs	Incidence of cleft lip and palate malformations in Sweden, 1973–1986	CS: highest levels of dioxin within 1 km of the WI, decreased as distance increased CS and RS: no increased risk of cleft lip and palate in studied areas after start of incineration
Williams et al., 1992	Residents in at-risk areas near two WIs Residents in a comparison area	By wind speed/ direction, local topography, soil levels of pollutants	Male/female sex ratio of births in Scotland, 1975–1979 and 1980–1983	No differences between the at-risk and comparison areas A significant excess of female births in the district at most risk
Rydhstroem, 1998	Residents in all municipalities or in 14 municipalities near 14 refuse WIs in Sweden	With or without WI; before and after start of WI	Incidence of twinning, 1973–1990	No clustering of twinning in area/time RR (before vs. after) significantly increased in one but decreased in one of the municipalities with WI

Note: WI is waste incinerator and RR is relative risk.

By using geographical information and 3-dimensional mapping techniques, Williams et al.[10] observed significantly low male-to-female sex ratios of births in the district identified as being at most risk of exposure to air pollution from two incinerators in central Scotland. There was no significant difference in the sex ratios of births between all at-risk areas and the comparison area. Past exposure to air pollution from the incinerators was estimated by wind direction/strength, local topography, anecdotal evidence of residents in the nearby communities, and concentrations of pollutants in the soils. However, the types of incinerators and waste burned have not been clearly stated in the study, and comparisons with results from other studies are difficult to make. Rydhstroem[11] observed no clustering of twin deliveries in 14 municipalities near refuse incinerators in Sweden from 1973 to 1990. The data source was the Swedish Medical Birth Registry, which had records for all pregnancies with a gestational age greater than or equal to 28 weeks or less if the newborn was alive at birth. The relative risk (before vs. after start of the incinerator and adjusted for year of delivery and maternal age) was nonsignificant for 12 municipalities, but significantly increased in one and decreased in another municipality.

Effect on Cancer Risk

Four studies investigated the cancer risk of incinerator emissions (Table 2).[12-15] Elliott et al.[12] used the postcoded database of the Small Area Health Statistics Unit to analyze the incidence of cancers of the larynx and lung near 10 incinerators of waste solvents and oils because of reports of clusters of laryngeal cancer cases near one incinerator site. No excess in incidence of laryngeal or lung cancer was found, nor was there evidence of increasing cancer risk with closer proximity to the incinerators. More recently, Elliott et al.[13] examined cancer incidence of people living within 7.5 km of 72 municipal solid waste incinerators in Great Britain in a two-stage study. The results showed a statistically significant decline in risk for all cancers and for stomach, colorectal, and lung cancer as the distance from incinerators increased. However, the authors cautioned that residual confounding near the incinerators and misdiagnosis might have contributed to these findings. Limitations of this study may include lack of exposure data and mixing of the potential effects of old and new incinerators. Moreover, the type of pollution control equipment used in the incinerator, which affected the emissions, was not taken into account.

In a case-control study, Biggeri et al.[14] investigated

Table 2. Epidemiologic studies of cancer risk of waste incineration on community residents.

Study	Study Subjects/ Type of Incinerator	Exposure Assessment	Outcomes	Results
Elliott et al., 1992	Residents near 10 incinerators of waste solvents and oils in Great Britain	All study areas Distance from the incinerator (<3, 3–10 km)	Incidence of cancers of larynx and lung	No excess in incidence of cancers of larynx and lung compared to national rates No significant increase in cancer risk with closer proximity to the incinerators
Elliott et al., 1996	People living within 7.5 km of 72 municipal solid WIs in Great Britain	Distance from the incinerator (0.5, 1, 2, 3, 4.6, 5.7, 6.7, 7.5 km)	Incidence of all and selected cancers	Both stages of the study: risk of all cancers, and of stomach, colorectal, liver, and lung cancer decreased as distance increased; residual confounding
Biggeri et al., 1996	755 male lung cancer cases and 755 matched (date of death, sex, age) controls in Trieste, Italy 4 sources: shipyard, iron foundry, city center, a WI	Spatial models based on distance from each of the four sources	Lung cancer deaths	Lung cancer risk significantly related to the incinerator: excessive RR = 6.7 in the source, adjusting for age, smoking, air particulate, and occupational carcinogens
Michelozzi et al., 1998	Residents within 10 km of a waste disposal site, a municipal WI, and an oil refinery plant in Rome	Distance from the sources (0–3, 3–8, 8–10 km)	Deaths by cancer of liver, larynx, lung, kidney, and lymphatic and hematopoietic systems in 1987–1993	No associations between cancer deaths and distance, except for laryngeal cancer in men—with a significant decline as distance increased

Note: WI is waste incinerator and RR is relative risk.

the effects of air pollution from four sources (including shipyard, iron foundry, city center, and an incinerator) on lung cancer deaths. The incinerator was significantly related to lung cancer (excessive relative risk = 6.7 in the source) after adjusting for age, smoking habits, exposure to occupational carcinogens, and air particulate levels in the spatial models. This study used distance between each subject's residence and each of the four sources of pollution as a continuous variable, instead of a categorical variable, in the spatial modeling. Michelozzi et al.[15] used the small area techniques to study mortality from cancer of the liver, larynx, lung, kidney, and lymphatic and haematopoietic systems among residents within 10 km of three major sources (a waste disposal site, a municipal waste incinerator, and an oil refinery plant) of air pollution. There was no significant decline in cancer mortality with increased distance from the sources, except for laryngeal cancer in men. Note that the study did not differentiate the effect of incinerator emissions from the other two major sources of air pollution.

Effect on Respiratory Health

Two studies examined the respiratory effects of incinerator emissions (Table 3).[16,17] In a cross-sectional study, Gray et al.[16] compared the frequency of respiratory symptoms between 713 children in two study areas close to two sewage treatment facilities with high-temperature sludge-burning incinerators in coastal Sydney and 626 children in a control region. The levels of several air pollutants were similar in the study and control regions during the study period. Baseline forced expiratory volume in 1 sec (FEV_1) and prevalence of current asthma, atopy, symptom frequency, or any category of severity of asthma illness was not significantly different between the control and study regions, except for past asthma, which was lower in the study areas. One advantage of this study was that both subjective methods, such as a questionnaire for respiratory symptom, and objective methods, such as the pulmonary function test and the histamine bronchial challenge test, were used to measure outcomes. However, the comparisons between two regions were made without taking into account other important factors, such as parental cigarette smoking, socioeconomic status, and indoor air quality.

Shy et al.[17] conducted a 3-year air quality and epidemiologic study of three incinerator communities (with a hazardous, biomedical, and municipal waste incinerator, respectively) and three matched control communities. Results from their first-year study showed (1) no consistent differences in prevalence of respiratory

Table 3. Epidemiologic studies of respiratory effects of waste incineration on community residents.

Study	Study Subjects/ Type of Incinerator	Exposure Assessment	Outcomes	Results
Respiratory Health				
Gray et al., 1994	713 children in 2 regions near 2 sludge burning WIs in Sydney Controls: 626 children in a region with no WI	Air monitoring Region of residence	Prevalence of respiratory illness, airway hyper-responsiveness, atopy; FEV_1	No significant differences in baseline FEV_1 and prevalence of current asthma, atopy, symptom frequency, or severity of asthma illness between study and control regions
Shy et al., 1995	Selected normal and sensitive residents of 3 WIs (biomedical, hazardous, and municipal) and 3 matched comparison communities in North Carolina, 1992–1994	Air monitoring; wind sector analysis; chemical mass balance receptor modeling; dispersion modeling	Prevalence of acute/ chronic respiratory diseases/symptoms; PEFR, FEV_1; cell counts, albumin, etc., in nasal lavage	No consistent differences in prevalence of respiratory symptoms between matched communities, adjusting for age, sex, race, education, respiratory disease risk factors No differences in nasal lavage analysis Among sensitives: significant difference in PEFR between two matched pairs of communities
Body Levels of Chemical				
Kurttio et al., 1998	113 residents aged 7–64 years within 5 km of a hazardous WI, 11 workers of the WI, a reference group of 55 people in Finland	Distance (1.5–2, 2.5–3.7, 5 km)	Hg in hair, 1984 and 1994	Changes in hair Hg levels increased with decreased distance from the WI during 10-year period, adjusting for age, sex, fish consumption, and water source

Note: WI is waste incinerator, FEV1 is forced expiratory volume in 1 sec, PEFR is peak expiratory flow rate, and RR is relative risk.

symptoms between matched incinerator and comparison communities after adjustment for age, sex, race, education, and respiratory disease risk factors in the logistic models; (2) no differences for mean peak expiratory flow (PEFR) and FEV_1 among normal subjects of matched communities; and (3) significant differences in PEFR among sensitive subjects in two pairs of matched communities. This study applied several methods to assess exposures, including air monitoring, wind sector analysis and chemical mass balance receptor modeling, and dispersion modeling. Moreover, the effects of incinerator emissions were assessed in both normal and sensitive subjects.

Body Levels of Heavy Metal

Kurttio et al.[18] compared the hair total Hg concentrations of five groups of subjects, including 45 residents within 1.5–2 km, 38 residents within 2.5–3.7 km, and 30 residents living 5 km from a hazardous waste incinerator; 11 workers of the incinerator; and a reference group of 55 people, before and 10 years after operation of the incinerator in Finland (see Table 3). Hair total concentrations of Hg were similar among groups in 1984, but differed among groups in 1994 (Kruskall-Wallis test, $p = 0.07$). Median of Hg in hair was highest in the incinerator workers, but information of multiple comparisons was not provided. Changes in hair Hg levels during a 10-year period increased slightly with decreased distance from the incinerator, after adjustment for age, sex, fish consumption, and water source. The increases in hair Hg from 1984 to 1994 were 0.35, 0.16, 0.13, 0.03, and 0.02 mg/kg, respectively, for the five exposure groups.

EPIDEMIOLOGIC STUDIES OF INCINERATOR WORKERS

Eleven epidemiologic studies conducted on incinerator workers were reviewed (Tables 4 and 5).

Effect on Mortality

Three studies investigated the potential effect of incineration on cause-specific mortality (Table 4).[19-21] The study by Gustavsson[19] suggested excessive death from lung cancer [standardized mortality ratio (SMR) = 355, 95% confidence interval (95% CI) = 162–675] and ischemic heart disease (SMR = 138, 95% CI = 95–193) among 176 male workers employed for at least 1 year at a municipal waste incinerator, compared with the national rates. Among those employed for more than 30 years or followed up for more than 40 years, there was a significant increase in death from ischemic heart disease (SMR = 167 and 186, respectively). Nonetheless, the information for tobacco smoking, a habit known to be related to both diseases, was collected by interviewing workers' colleagues or employers and was not controlled for in the study. The results could have been biased away from the null if cigarette smoking was more prevalent among these workers than among the reference population. In another study,[20] mortality from esophageal cancer was nonsignificantly higher among the same cohort of 176 workers (SMR = 150, 95% CI = 4–834), but the SMR was very unstable, with only one esophageal cancer death. These two studies were limited by small sample size and lack of exposure data. The exposures were estimated by duration of employment and years from the first employment.

Rapiti et al.[21] studied mortality of a cohort of 532 males employed at two municipal incineration plants between 1962 and 1992. The workers had similar all-cause and all-cancer mortality and lower lung cancer risk (SMR = 55, 95% CI = 15–142), but significantly higher gastric cancer mortality compared with the regional population. The SMR of gastric cancer was 421 (95% CI = 144–964) for those with more than 10 years since first employment and 461 (95% CI = 126–1190) for those with more than 10 years of employment. Air concentrations for chemicals were available only from a 1978 survey, in which the organic dust levels ranged from 0.10 to 8.6 mg/mm^3. Factors that may have affected the results are small number of deaths (total = 31) during the 1965–1992 follow-up, misclassification of exposure, and the healthy worker effect.

Frequency of Urinary Mutagens and Promutagens

Two studies examined the frequency of urinary mutagens and promutagens (Table 4).[22,23] Scarlett et al.[22] observed a significantly higher prevalence of urinary mutagens (11.5 vs. 1.6%) and promutagens (18.3 vs. 3.3%) in 104 municipal incinerator workers as compared with 61 water treatment workers. Incinerator workers were 9.7 (95% CI = 1.2–76.7) times more likely to have urinary mutagens than controls, adjusting for age, smoking and fried meat consumption immediately preceding urine sampling, use of wood stoves at home, and alcohol consumption.

Ma et al.[23] compared prevalence of urinary mutagen and promutagen in 37 workers from four municipal solid waste incinerators and a control group of 35 workers from eight water treatment plants. Urine samples were collected on three separate occasions about 1 week apart. Urinary mutagens/promutagens were significantly more prevalent in incinerator workers than in the controls (21.6 vs. 5.7%) for the first sample, but no differences were found in the second or third samples. Intra-individual repeatability of demonstrating urinary mutagens was poor in these incinerator workers. The only potential confounder evaluated in this study was smoking within 24 hr before urine sampling. These two studies observed a higher prevalence

of urinary mutagens and promutagens in incinerator workers compared with controls. However, the relationship between exposure levels and urinary mutagens and promutagens and their consequent health effects in incinerator workers were not investigated in these studies and were not well understood.

Effect on Lung and Renal Function

One study explored the possible effects on lung function and renal and hepatic function (Table 4). In a cross-sectional survey, Bresnitz et al.[24] found no significant difference in symptoms reported by 86 municipal waste incinerator workers in the high exposure group compared to the low exposure group as defined by their job title. Forced vital capacity (FVC), FEV_1, FEV_1/FVC, and forced expiratory flow were similar for both groups after adjusting for smoking status. Mean blood or urinary levels of Hg, Pb, As, and Cd were comparable between the two groups and were within the normal ranges. However, ~31% of the study cohort had significant proteinuria, and the prevalence of hypertension was higher than that of the U.S. population. This study also included a 5-day environmental monitoring of polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) expressed as 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD equivalents), particulates, silica, and metals. The calculated concentrations of TCDD equivalents were 0–47.0 ng/m^2 for the five surface wipe samples, with one sample exceeding the National Research Council (NRC) guideline (25 ng/m^2); and 0–24.2 pg/m^3 for the six area air samples, with one above the NRC guideline value of 10.0 pg/m^3. The air concentrations of respirable dust and silica, respectively, were below the Occupational Safety and Health Administration (OSHA) permissible exposure limit (PEL) of 5.0 mg/m^3, and the maximal 8-hr time-weighted-average concentrations of metals/minerals, except Pb and P, in area and personal breathing zone were under the standards of the American Conference of

Table 4. Epidemiologic studies of the health effects of waste incineration on incinerator workers.

Study	Study Subjects/ Type of Incinerator	Exposure Assessment	Outcomes	Results
Mortality				
Gustavsson et al., 1989, 1993	176 workers employed 1+ years at a municipal WI, 1920–1985	WI workers vs. general population	Deaths, 1951–1985 Esophageal cancer deaths	Lung cancer death: SMR = 355 (162, 675); Ischemic heart disease: SMR = 138 (95, 193); SMR = 167 for workers employed for 30+ years Esophageal cancer: SMR = 150 (4, 834)
Rapiti et al., 1997	532 males employed at 2 municipal WIs in Rome, 1962–1992	WI workers vs. regional population	All- and cause-specific mortality, 1965–1992	All-cause mortality: SMR = 71 Lung cancer: SMR = 55 (15–142) Gastric cancer: SMR = 279 (94–635); SMR = 461 (126–1190) with 10+ years of employment
Urinary Mutagen/Promutagen				
Scarlett et al., 1990	104 workers at 7 municipal WIs 61 controls at 11 water plants	By type of plants (WI vs. water)	Urinary mutagen/ promutagen	Significantly higher prevalence of urinary mutagen (18.3 vs. 3.3%) and promutagen levels (11.5 vs. 1.6%) in WI workers
Ma et al., 1992	37 workers at 4 municipal WIs 35 controls at 8 water treatment plants	By type of plants (WI vs. water); job activities	Urinary mutagen/ promutagen	Significantly higher prevalence of mutagen/promutagen in WI workers than in controls (21.6 vs. 5.7%) for the first urine samples only
Lung Function				
Bresnitz et al., 1992	86 workers at a municipal WI	By job history (high vs. low exposure groups)	Lung function; renal and hepatic function	Lung function: nonsignificant difference between two groups High prevalence of hypertension and related proteinuria in this cohort

Note: WI is waste incinerator.

Governmental Industrial Hygienists (ACGIH) and OSHA. Furthermore, the exposures to metals varied among incinerator workers of different job titles, and two workers had exposures to Cd or Ni above the ACGIH threshold limit values (TLVs) and the National Institute for Occupational Safety and Health recommended exposure limit (NIOSH REL), respectively.

The major strengths of this study are monitoring air concentrations of chemicals, measuring blood/urine levels (the internal dose) of these chemicals, and assessing the subclinical effects of occupational exposures. Interpretation of the results should consider that the study was conducted on a convenient sample, monitored exposure for only a short period of time, and used a cross-sectional design. Further assessment of the association between body levels of chemicals, instead of high versus low exposure groups, and morbidity will be of great value.

Body Levels of Chemicals

Five studies assessed body levels of certain chemicals in incinerator workers (Table 5),[18,25-28] including the Finnish study[18] of hair Hg levels mentioned earlier. Angerer et al.[25] compared body levels of hydroxypyrene and several precursors of PCDDs and PCDFs among 53 workers at a municipal waste incinerator and 431 controls. Incinerator workers were found to have significantly higher urinary hydroxypyrene and 2,4,5-trichlorophenol, but lower 4-monochlorophenol and 2,3,4,6-/2,3,5,6-tetrachlorophenol than the controls. Although smoking was controlled as a confounder, other factors, such as age and sex, which distributed differently between workers and controls, were not evaluated in the analysis and might bias the results. Malkin et al.[26] examined blood Pb and erythrocyte protoporphyrin (EP) levels in 56 municipal incinerator workers and a control group of 25 boiler workers. The results showed that incinerator workers had a statistically significant higher mean blood Pb level (11.0 vs. 7.4 μg/dL), but a significantly lower mean EP (21.0 vs. 27.9 μg/dL) than the controls.

Schecter et al.[27] compared the blood levels of certain PCDDs and PCDFs between three groups: 10 workers from an older municipal waste incinerator without adequate pollution controls, 11 workers from a newer municipal waste incinerator with modern pollution controls, and 25 controls from the general population. The results showed significant increases in mean blood levels of certain and total PCDDs and PCDFs in workers from the older incinerator, but not the newer incinerator, compared with the controls. In addition, several PCDD and PCDF congeners were identified in the slag and fly ash from the older incinerator. Wrbitzky et al.[28] compared the blood/urine levels of selected metals and organic compounds among three groups of workers (waste incinerator, periphery, and management) employed in an industrial waste incineration plant. There were significantly higher blood levels of toluene, Pb, and Cd, and urine levels of As and tetrachlorophenols in waste incinerator workers than in the other two groups. The differences between groups were very small, although certain parameters of the workers exceeded the background levels of the general population.

Table 5. Epidemiologic studies of the effects of waste incineration on body levels of chemicals in incinerator workers.

Study	Study Subjects/ Type of Incinerator	Exposure Assessment	Outcomes	Results
Angerer et al., 1992	53 workers at a municipal WI 431 controls	By type of plants (WI vs. non-WI)	Blood/urine levels of selected organic compounds	Significantly higher HCB, 2,4/2,5-DCP, 2,4,5-tetrachlorophenols, and hydroxypyrene in WI workers
Malkin et al., 1992	56 workers at 3 municipal WIs 25 controls at heating plants	By type of plants (WI vs. heating); job activities	Blood Pb and EP levels	Significant higher blood Pb (11.0 vs. 7.4 μg/dL) and lower EP (21.0 vs. 27.9 μg/dL) in WI workers
Schecter et al., 1995	10 workers at an older municipal WI 11 workers at a newer municipal WI 25 controls	By type of plants (old vs. new WI vs. non-WI)	Blood levels of PCDDs and PCDFs	Significantly higher PCDDs and PCDFs in workers of old WI compared with controls; no difference between workers of newer WI and controls
Wrbitzky et al., 1995	122 workers at an industrial WI plant	By job titles (WI vs. periphery vs. management)	Blood/urine levels of metals and organic compounds	Blood toluene: WI workers > both groups Blood Pb and Cd, urine tetrachlorophenols and As: WI workers > either group

Note: WI is waste incinerator and EP is erythrocyte protoporphyrin.

Overall, these five studies observed higher body levels of certain organic compounds[25,27] and some heavy metals[18,26,28] in incinerator workers than in the controls. However, the relationship between exposure levels and body concentrations of these chemicals and the consequent health effects of higher body levels of these chemicals in incinerator workers are not clear. Some PCDDs and PCDFs have been shown to be carcinogenic to certain animal species.[6]

DISCUSSION

This study extensively reviewed epidemiologic studies of the health effects of waste incineration on incinerator workers and residents of communities near incinerators. The studies of health effects of waste incineration among community residents showed some similar and some inconsistent results. First, the results for reproductive effects were conflicting. Higher frequency of twinning was found in the areas at most risk in one study,[8] but not observed in municipalities with incinerators in another study.[11] Waste incineration was associated with significantly lower male-to-female ratios of births in the areas at most risk,[10] but not with cleft lip and palate malformations.[9] Second, the findings for cancer risk were inconsistent. Three studies observed a significantly positive relation with lung cancer incidence,[13] mortality,[14] or laryngeal cancer deaths.[15] Yet, two studies found no excess in lung cancer incidence[12] and deaths[15] or laryngeal cancer incidence.[12] Third, prevalence of several respiratory symptoms was not significantly related to living in an area with a waste incinerator in both studies reviewed.[16,17] Finally, one study found a significant but small increase in changes of hair Hg levels with decreased distance from the incinerator during a 10-year period.[18]

The studies of incinerator workers consistently showed higher frequency of urinary mutagens and promutagens[22,23] and increased blood levels of certain organic compounds[25,27] and some heavy metals.[18,26,28] As for cancer risk, significantly excessive deaths from gastric cancer were observed in one occupational cohort[21] and a nonsignificant increase in esophageal cancer mortality was found among another group of incinerator workers.[20] The findings for lung cancer mortality were conflicting—significantly increased in one study,[19] but decreased in another study.[21] Regarding other health end points, working in an incinerator was associated with excessive deaths from ischemic heart disease[19] and higher prevalence of hypertension.[24] There was no evidence of adverse effects on lung function.[24]

Note that only a few epidemiologic studies have investigated the respiratory, cancer, cardiovascular, and reproductive effects of waste incineration products. Also, there are difficulties in comparing the consistency or discrepancy among these studies. First, the exposure sources are not similar for workers and residents of communities with incinerators. Incinerator workers are exposed to chemicals in fly ash and slag, while community residents are exposed to stack emissions. The chemical constituents and concentrations may not be the same. For the study of both residents near and workers of a hazardous waste incinerator,[18] hair Hg levels were higher in incinerator workers than in the reference group or residents within 5 km from the incinerator.

Second, these studies evaluated the health effects of different types of incinerators or different incinerators burning the same type of waste. The emissions, and hence exposures, may vary from incinerator to incinerator. Effects of municipal waste incinerators on community residents were assessed in six of the reviewed studies,[8,9,11,13,15,17] while effects of hazardous or chemical waste incinerators were investigated in four studies.[8,12,17,18] Types of incinerator were not clearly specified in two studies.[10,14] In the occupational studies, municipal waste incinerator was the main exposure of interest,[19-27] except in one study that investigated workers at an industrial waste incinerator.[28] Furthermore, the exposure was nonspecific incinerator emissions, and constituents of waste burned in the incinerator were not clearly described in many studies of residents.[8-11,13-15] Moreover, types of pollution control measures, such as the electrostatic precipitator, used in incinerators could affect the levels of pollutants in the incinerator emissions and hence the exposures among community residents, but they were not clearly presented except in two of the studies.[17,18]

Third, several studies[8-13,15] examined the potential effects of incinerator emissions on community residents using an ecological-type approach. Efforts were made to better assess exposure to incinerator emissions for each location, but not for each individual living in that location. Information regarding other potential confounding factors was not obtained for individuals; thus, these factors could not be taken into account in assessing the exposure-outcome associations and might have biased the observed incinerator-outcome association. Nonetheless, these studies help to generate hypotheses regarding possible effects of incinerator emissions.

Fourth, the occupational exposures in incinerator workers were assessed by job title or duration of employment,[19-28] instead of by actual concentrations of chemicals. Measurement error in exposure assessment could bias the exposure-outcome association. Furthermore, occupational exposure levels could have changed during a long period of time when the incinerating conditions changed, such as in the study by Rapiti et al.[21] For the study of health effects of long-term occupational exposure among

incinerator workers, the assessment of individual worker's exposure to a mixture or specific chemicals during a long time period could be a challenge, especially when there was no historical monitoring of air concentrations of chemicals or detailed information about job history and changes in operations of the incinerators. Misclassification of exposure could bias the exposure-health outcome association in either direction.

Fifth, levels of occupational exposures may differ from incinerator to incinerator and make it difficult to compare the results of these occupational studies. For example, three studies that assessed internal exposures to metals in incinerator workers had different findings. In the study by Bresnitz et al.,[24] air concentrations of Hg, Pb, As, and Cd generally met the standards, except for the maximum 8-hr time-weighted average concentrations of Pb (above the ACGIH standard). The exposures to metals varied among workers of different job titles, and two workers had been exposed to Cd or Ni above the ACGIH TLVs and NIOSH REL, respectively.[24] Moreover, the mean blood levels of Hg, Pb, and Zn protoporphyrin and mean urinary Hg, Pb, and Cd, respectively, were comparable between the high and low exposure groups, except that blood As level was higher in the low exposure group.[24] Note that this study compared incinerator workers of different job activities, instead of using a control group. Malkin et al.[26] found significantly higher blood Pb levels and lower EP in incinerator workers compared with controls. In the Wrbitzky et al.[28] study, blood levels of Pb and Cd, but not Cr and Hg, were significantly higher in incinerator workers than in periphery workers. Health effects were not evaluated in this study.

Sixth, different health effects were investigated in previous studies, and sometimes only one or two studies evaluated the same end points. Selection of the health outcome of interest was explained and justified by the respective authors of the studies reviewed. Comparisons of study results are difficult to make when taking into account the differences in exposures, outcomes, design, or potential bias in each study. Finally, these prior studies investigated the body levels or health effects of either specific chemicals or mixtures of incinerator emissions. Sometimes a specific chemical or a group of chemicals were speculated to be associated with the observed outcomes of interest, based on biological plausibility; however, the estimation of exposure levels for that particular chemical was not available for evaluating the dose-response relationship.

There is an increasing trend toward using incineration to manage waste; therefore, more people will be at risk of exposure to incinerator emissions. It is important to investigate the health effects of waste incinerators currently in operation. More descriptive studies, which use existing disease registration data, can be conducted to compare the incidence of cancers, cardiovascular diseases, reproductive outcomes, and hospital visits of respiratory diseases in areas with and without an incinerator, and also for communities before and after construction of incinerators. Furthermore, studies are required to delineate the relationships between exposures to heavy metals and organic compounds and both internal dose and potential health end points. Moreover, the emissions from waste incineration are complex; municipal waste incineration may emit acid gases, some criteria pollutants, metals (such as As, Cd, Pb, etc.), dioxins, furans, and many other organics. Other than airborne pollutants and Pb, of which the exposure levels and health effects have been studied,[16,17,26] and some organic emission products that have been speculated to be associated with reproductive outcomes,[8,10] the health effects of incomplete combustion byproducts, such as dioxins and furans, and other metals emitted from incinerators, such as Hg, Cd, and Be, have not been extensively investigated. Finally, more hypothesis-testing epidemiologic studies, such as case-control studies and cohort studies, are needed to assess the associations between waste incineration and the risk of cancers, cardiovascular diseases, respiratory health, and reproductive outcomes among incinerator workers and community residents.

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