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# Health Impact Assessment of Waste Management: Methodological Aspects and Information Sources

Science Report P6-011/1/SR1

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Professor Mike Depledge

Head of Science

# Executive Summary

## Terms of Reference

The Environment Agency Research and Development Project P6-011/1 was initiated to provide an informed approach to ‘health risk management’ in the waste field. This report completes Stage 1 of the Project and presents a literature review of impacts on human health from different waste disposal options and an evaluation of the current state of knowledge regarding health impact assessment.

## Generic findings

The generic lessons derived from this study can be summarised as follows:

- Experience with Health Impact Assessment (HIA) is better developed in local authority departments of environmental health than in planning departments. It is better developed still amongst health professionals. However, the HIA procedure is still evolving and current levels of adoption by other UK agencies is low. The greatest barrier to adoption is the current lack of any statutory requirement for a HIA.
- HIA is more effective if applied to different alternatives before decisions are made and it would be sensible if HIA were applied at the strategic level before it is applied to individual projects.
- Most epidemiological investigations reported in this area have been based on spatial patterns of morbidity or mortality and confounding factors, such as deprivation, have been insufficiently quantified to permit scientifically robust conclusions on disease causation, let alone providing credible dose-response relationships.
- A large UK study has reported a small excess risk of congenital abnormalities for babies born to mothers residing within 2 km of landfill sites, compared with mothers residing further than 2 km; but a causal link has not been established.
- Newly constructed incinerator plants have to meet stricter controls on emissions than those operating prior to the mid 1990s. Analyses of cancer incidence associated with the older generation of incinerators demonstrates that any potential risk of cancer, due to residency for periods in excess of ten years near to municipal solid waste incinerators, is exceedingly low and probably not measurable.
- Data on emissions and ambient air monitoring in the vicinity of incinerators indicate that modern well-managed waste incinerators will only make a very small contribution to background levels of air pollution.
- Robust epidemiological data concerning health risks associated with exposure to bioaerosols are not available.
- Emissions of bioaerosols from composting, landfill and waste recycling plants are a potential concern.
- Emissions and exposures from landfill and composting operations are much less well characterised than those from incineration.
- A limited number of epidemiological studies have investigated psychological morbidity for residents in the vicinity of hazardous waste sites. There is some evidence that

psychological morbidity is increased but the association may be confounded by factors related to social deprivation.

## **R & D Gaps**

- Research is needed to facilitate a move away from a simplistic “concentric circle” spatial impact approach, employed in previous epidemiological studies, to a much more precise definition of the exposed population. This requires a more sophisticated spatial epidemiological approach linked to dispersion modelling of emissions which incorporates local meteorology and topography.
- Research should seek to generate comparative data on bioaerosol concentrations as well as constituents of bioaerosols in the following situations:
  - a) at distance from contained and uncontained systems
  - b) in proximity to different operations eg. agriculture, sewage treatment
  - c) at different background locations eg near woodlands, crops or urban areas.
- The development of appropriate biomarkers of exposure and effect would represent a significant research advance of considerable operational and regulatory utility.
- The size distribution of particles, particularly ultra fine particles and their trace metal content, may have toxicological significance which is, at present, not understood but has been hypothesised by several authors.

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# 1 Introduction

The inter-relationship between health effects, perceived or real, and the range of waste management processes regulated is an important cause of concern to the public at large, the media and local interest groups today. There have been an increasing number of epidemiological research reports linking a range of ill-health outcomes to waste management installations. These studies include childhood cancers, birth abnormalities and liver cancers associated with incinerators and with landfill facilities.

Nevertheless, there is a paucity of clear evidence to support or refute claims of adverse health effects from managing waste. In the public mind, therefore, there is considerable doubt which inevitably leads to misunderstanding and mistrust of decisions made on waste management options.

The production of waste in England and Wales in the foreseeable future is inevitable, so waste management is a necessity. Given this fact, there is a need for an overview of the current state of knowledge regarding the health impacts from waste management, and a comparative assessment of the risks and benefits of the variety of available waste management options. There is also a need to assess the health impacts of the changes in waste management which will result from implementation of the Government Waste Management Strategy 2000 and the implementation of the Landfill Directive.

It forms the first part of a two stage project commissioned by the Environment Agency (the Agency) to assess the impact of waste disposal practice (both current and projected) on health and to provide advice on future waste disposal options likely to minimise health impacts, at both local site, regional and national levels within the UK.

The second part of this project will build on the review contained in this report and will provide methodological advice on practical ways in which the health impact of waste management options can be assessed. This will include identifying those areas of data and information that will require further research before a meaningful assessment of health impact can be made. This advice will also be placed in the context of current UK and international views on Health Impact Assessment (HIA) as a process.

In order to provide the necessary precursor to the second part of this project, this report addresses methodological aspects and information sources relating to health impacts associated with waste disposal options. It reviews the epidemiological research literature on health effects, including those related to community mental health, which are associated with waste disposal options and highlights the uncertainties involved. This is supplemented by a review of the current understanding of the health effects of key chemical contaminants associated with waste. It identifies the gaps in current data on emissions and exposures and presents a critique of site-specific risk assessments for each of the waste disposal options.

All this needs to be placed in the context of current state of knowledge regarding HIA. The next chapter provides such a review in addition to evaluating current practice both within the UK and at an international level.

## 2 Health Impact Assessment of Projects and Policies

### 2.1 Introduction

This Chapter deals with the process of Health Impact Assessment (HIA). It examines the literature in order to explore what is meant by a HIA and covers, in particular:

- definitions of HIA
- the purpose of HIA
- the conceptual roots of HIA
- definitions of 'Health'
- the determinants of health and the scope of HIA
- issues requiring co-ordinated policy responses
- positivistic and relativistic bases for predictions in HIA
- levels at which HIA takes place
- quality criteria for HIA.

Following on from this, there is a consideration of the claim that HIA can improve the formulation of strategy or policy, and a consideration of how HIA can assist with the implementation of a waste strategy by contributing to decisions on the selection, siting and operation of particular waste facilities.

The Chapter concludes with a review of practice: this describes a survey of local authorities in the UK to ascertain HIA practice, briefly reviews the guidance on Environmental Health Impact Assessment issued in Canada, and finally considers some published HIA reports. The consideration of reports is divided into those related to waste management (of which there are two) and selected HIAs of non waste related projects, in order to illustrate the sorts of approaches used and the sorts of issues covered in the reports.

### 2.2 Methodology

This Chapter attempts to draw together ideas from a wide range of diverse sections of the literature rather than comprehensively to review any one of them. For this reason it is based on the authors' experience and knowledge of HIA rather than a systematic search of any particular area of the literature.

However, a more formal approach was used to carry out the review of practice in UK local authorities as comprehensively and effectively as possible.

1. A questionnaire survey was conducted of local authorities throughout the UK to gauge experience of HIA at both project and plan levels. The results of this survey were used to draw some basic conclusions about current experience, and also to identify examples of HIA documents which were submitted (February 2002).
2. The Canadian Government has produced a comprehensive *Handbook on Health Impact Assessment* which is briefly reviewed in the context of this study and the value of the 'materials for waste projects' (within the handbook) is evaluated.
3. The documents identified in the questionnaire survey were individually analysed to determine the health issues covered, the methodological approaches used, the

stakeholder involvement (and approaches used) and the conclusions reached in each case.

Each stage of this approach is addressed below under the appropriate headings.

### **2.2.1 Questionnaire survey**

A very brief questionnaire (see [Appendix A](#)) was sent to local authorities in the UK to find out about their experience with HIA. The choice of respondent was simply made by selecting those council officers with an email address, thereby allowing rapid responses to be obtained. Furthermore, the 2002 edition of the *Municipal Yearbook* (Hemming Information Services, 2002) was used to make a selection of those council officers separately responsible for 'Environmental Health' and for 'Planning'. There is not always a clear distinction between these two in the administrative structures of local authorities, and no attempt was made to refine the selection obtained using the Yearbook (CD ROM version) search facility.

As this review was seeking experience, the study was not restricted to England and Wales, but also included Scotland and Northern Ireland (which are outside the remit of the Environment Agency).

A total of 304 questionnaires were sent to Environmental Health Officers and 326 to Planning Officers (this is the figure after deleting all those email addresses rejected as being invalid). These two departments were selected because it might be expected that Environmental Health have expertise relating to health issues, whereas Planning have the expertise they need to determine planning applications, some of which might affect human health. Indeed, Planning Departments have experience of Environmental Impact Assessment and might be expected, therefore, to develop equivalent experience of HIA. As such, it is interesting to identify where any expertise in HIA is concentrated – if there is a difference at all.

In total 170 responses were received, thus, the study allowed a picture to be obtained of current experience and indicated where to obtain further information to help with the study. The results by no means cover the whole of the UK and cannot be considered to be comprehensive, but the response is sufficiently good to allow some tentative conclusions to be drawn.

## **2.3 What is meant by a HIA?**

### **2.3.1 Definitions of HIA**

Definitions that have been given of HIA include

'Any combination of procedures or methods by which a proposed policy or program may be judged as to the effects it may have on the health of a population' (Ratner *et al.*, 1997).

'A combination of procedures, methods and tools by which a policy, a program or project may be judged as to its potential effects on the health of a population and the distribution of effects within the population' (WHO European Centre for Health Policy, 1999).

'A methodology which enables the identification, prediction and evaluation of the likely changes in health risk, both positive and negative (single or collective), of a policy, programme plan or development action on a defined population. These changes may be direct and immediate or indirect and delayed.' (BMA Board of Science and Education, 1998).

'Health Impact Assessment is the estimation of the effects of a specified action on the health of a defined population' (Scott-Samuel, 1998).

‘Health Impact Assessment is a method of evaluating the likely effects of policies, initiatives and activities on health at a population level and helping to develop recommendations to maximise health gain and minimise health risks. It offers a framework within which to consider, and influence the broad determinants of health.’ (Scottish Office, 1999).

HIA is ‘a combination of procedures or methods which enable a judgement to be made on the effect(s) – positive or negative – of policies, programmes or other developments on the health of a population or on parts of the population where health are concerned.’ (National Assembly for Wales, 1999).

The two essential features of an HIA are that it is:

- concerned to predict how a contemplated decision would affect the health of a population
- intended to influence and inform that decision.

This definition excludes some activities which others have sometimes described as HIAs. The assessment of health consequences of decisions already implemented, and of events which have occurred, have been described as retrospective or concurrent HIA but would be excluded by the definition offered above. Similarly, exercises involving participation of the community in assessing how aspects of their environment affect their health, which are often described as HIAs, would be excluded by the definition proposed. Advocacy of a particular policy choice is not HIA although it may well be informed by an HIA.

While retrospective and concurrent assessments of how decisions and events affect the health of populations are excluded from the definition of HIA, they are highly relevant to it. Understanding of causal chains and the links between changes in the physical or socio-economic environment and the health experience of populations is mostly based on study of previous events. Retrospective and concurrent studies of health impacts provide the knowledge on which prediction of future events in HIA is based.

Participation is usually viewed as a characteristic of good HIAs and efforts have usually been made to involve the community affected (stakeholders) but some HIAs have not included this feature.

### **2.3.2 The purpose of HIA**

HIA is an activity intended to influence decision making so that policies, projects and programmes in all areas lead to improved population health or at least do not damage population health. The government waste strategy is an example of a policy and consists of a collection of goals, values and broad proposals guiding the approach to this particular issue. The construction of a particular waste disposal facility at a particular site would be an example of a project. There are three ways in which HIA might influence policy, project and programme decisions:

- by ensuring that decision makers always include health consequences among the issues considered
- by helping decision makers identify and assess possible health consequences and optimise overall policy outcomes
- finally by helping those affected by policies to participate in policy formation and contribute to decision making.

This review concentrates on the claim that HIA is a tool to help decision makers gain better insight into outcomes, balance health against other policy considerations, appraise options and improve the trade-offs which are an inherent feature of most decisions.

### 2.3.3 The conceptual roots of HIA

HIA draws on a wide range of disciplines, but the two main conceptual roots lie in:

- Impact Analysis, especially Environmental Impact Assessment
- Policy Appraisal and the promotion of Healthy Public Policy.

The development of Environmental Impact Assessment was encouraged by increased awareness of environmental damage and is now embedded in a legislative framework in many countries. It draws on cost-benefit analysis, ecology, biological sciences, epidemiology, toxicology, risk assessment and, increasingly, sociological disciplines. Currently Environmental Impact Assessments, particularly in the UK, frequently pay inadequate attention to the possible consequences for human health (Arquiaga *et al.*, 1994). However many suggest that extension of Environmental Impact Assessment so that health issues are properly covered would be a logical way to develop HIA (Joffe and Sutcliffe, 1997).

HIA could also be seen as a specialised form of Policy Appraisal which seeks to analyse the content of policies and of the policy making process. Policy Appraisal draws on political science, political economics and social sciences. Until recently, however, discussion of health consequences was largely limited to policies concerned with the provision of medical facilities. The call for Healthy Public Policy was a response to this restricted view and tried to extend the applications of Policy Appraisal to health consequences of all policies.

### 2.3.4 Definitions of 'health'

HIA is concerned with health but the meaning of that term is contested. Frequently the term health is defined as the absence of disease and a great deal of 'health' policy is concerned with the provision of health services for those with disease. Sometimes 'health' policy is extended to cover the prevention of disease through measures such as immunisation, health education and even provision of safe food and water. The definition of health given in the constitution of WHO (1946) noted above, however, suggests a very different view of health, that is 'Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity'. Such a view has been criticised as hopelessly utopian (Siracci, 1997) and so boundless as to be meaningless (Seedhouse, 1997). However, it is arguably a better vision against which to compare policy outcomes than one that focuses solely on disease.

Many HIA practitioners emphasise that they take a broad view of health (Scott-Samuel *et al.*, 1998) and rightly insist that an analysis limited to impacts on death and frequency of medically defined disease is inadequate. A satisfactory assessment considers the impact on all aspects of physical, mental and social well-being, including objectively assessed states, subjective feelings and positive aspects of health (as well as negative ones). Unfortunately epidemiology and related sciences, which could contribute to HIA, are currently limited in their ability to explore outcomes other than death or frequency of objectively assessed disease. This is likely to improve, and considerable progress is being made in developing measures of subjective health such as the Nottingham Health Profile (Hunt *et al.*, 1986), the Euroqol (EuroQol Group, 1990) and the Sickness Impact Profile with its derivatives such as Short Form 36 item (SF36) (Ware and Sherbourne, 1992).

### **2.3.5 The determinants of health and the scope of HIA**

The Black report (Black *et al.*, 1980) and, twenty years later the Acheson enquiry (Acheson, 1998) into health inequalities in the United Kingdom, emphasised the powerful influence that living conditions exercise over health. The overarching importance of general socio-economic, cultural and environmental conditions, housing and working conditions and social and community influences on health have been emphasised (Whitehead, 1995; Hertzman *et al.*, 1994) as has the effect of income distribution (Wilkinson, 1996). The terms atomistic fallacy (Marmot, 1998) and individualistic fallacy (Krieger, 1994) have been coined to describe the futility of attempting to understand the health of individuals in isolation without considering the communal context within which they exist.

The realisation that virtually every area of human activity influences health leads to the conclusion that most public or political decisions have the potential to impact on health for good or ill. Major improvements in population health are more likely to be achieved through interventions in economic, industrial, housing, transport, agriculture, education, law and order and other ‘non health’ areas than in the policy areas with which ministries of health are usually concerned. Milio (1986) argued that public policy should be used and assessed for the way it affected health and WHO include ‘healthy public policy’ as one of the key health promotion actions in the Ottawa Charter (WHO, 1986). HIA provides a framework within which the healthiness, or otherwise, of public policy can be assessed (Kemm, 2001).

### **2.3.6 The scope of HIA**

The issues to be covered by HIA are determined at the scoping stage. This should decide what should be covered in the HIA specifically:

- which outcomes
- which determinants (factors influencing health)
- the extent of population to be considered.

The statutory remit of the Environment Agency is limited to a rather narrow set of health determinants, covering emissions to land air and water; it excludes traffic volume, health and safety of workers, economic well-being of the community or social capital. Noise needs to be considered by the Environment Agency for processes (including the treating of waste) regulated under the Pollution Prevention & Control (PPC) regime that carries forward the need to ensure relevant objectives of the Waste Framework Directive are met through the previous Waste Management Licensing regime. Planning enquiries cover a much wider set of determinants, but are still subject to statutory limitations as to what factors they can consider.

### **2.3.7 Issues requiring co-ordinated policy responses**

Governments are concerned with the overall well-being of their populations, but responsibility for each issue is segmented between the many different departments, ministries or branches of government. While this may make for efficient government, it raises problems when the actions of one branch have consequences for the concerns of another. There are many issues, of which health is only one, that are affected by the decisions of numerous different branches of government and require a co-ordinated policy. Environmental sustainability, inequalities within society, social inclusion, well-being of families, law and order and fiscal balance are other examples of issues which cut across the concerns of several different departments. Given the requirement to consider all these issues, policy makers may be concerned that excessive concentration on one issue such as health could detract attention from other equally important issues.

Environmental sustainability is a prerequisite for long lasting health and there is a need to find forms of economic development, which meet ‘the needs of the present without compromising the ability of future generations to meet their own needs’ (World Commission on Environment and Development, 1988). There is concern that depletion of non-renewable resources, loss of biodiversity, overloading of pollution sinks and growth of human population could lead to a situation in which the capacity of the biosphere to support human life and health was exceeded (McMichael, 1993). A programme of action commonly referred to as Agenda 21 (Keating, 1993; Dodds, 1997) was produced at an international conference of heads of government in Rio de Janeiro. National and local governments frequently treat this as a ‘cross cutting’ issue and have set up procedures to ensure that decisions in all areas are congruent with this overall goal. HIA procedures could develop along similar lines.

Health inequalities are another cross cutting issue for many governments. The fact that health inequalities exist and are getting wider in many countries has been noted (Black *et al.*, 1980; Acheson, 1998). Their existence is recognised as inequitable and their reduction has been made another overarching goal for government. It has been suggested that all decisions should be assessed for their impact on health inequalities (Lester *et al.*, 1999), but since HIA has to be concerned with the distribution of health impacts across populations it should provide the information needed to address inequality issues (Douglas and Scott-Samuel, 2001).

Waste disposal might well be taken as an example of how a single policy issue has consequences for numerous areas. Decisions on waste strategy will have implications for the economy, energy use, transportation, regional planning, the environment, biodiversity, the physical environment, relations with European and other countries and many other policy areas as well as human health. The impacts will not be equally distributed but will differentially affect the various sectors of the population.

### **2.3.8 Bases for predictions in HIA**

HIA involves making predictions about the future consequences of decisions. Predictions based on natural science disciplines such as epidemiology and toxicology assess, for each hazard, the number of people exposed to particular levels and the likelihood of an individual experiencing harm when exposed to these levels (dose–response relationship). In theory, this approach could be extended to all determinands of health and all outcomes, but the current level of knowledge means it is usually limited to physico-chemical hazards and the frequency of both death and disease outcomes. Sometimes this approach may be built into complex models, which take into account changing levels of exposure and delays between exposure and outcome.

While immensely powerful, this approach is limited in application. The links between health and its determinants are frequently characterised by complexity. Slight uncertainty in initial conditions is quickly and enormously magnified and the system is unpredictable because the initial conditions can never be specified sufficiently precisely (Kolata, 1986). The approach claims to be value-free and provides no basis for preferring one value over another. Furthermore, it does not provide any basis for handling so called ‘irrational fears’. HIA needs to consider all of these things.

A totally different approach is commonly used in the humanities and social sciences. This emphasises that interpretation of events depends on their context and on the person perceiving them. It pays great attention to the understanding of underlying meanings. The basis for prediction is deemed to be held by the people who would be affected by the policy, and understanding of the situation comes from these people and the unravelling of the meanings that events hold for them. By emphasising the difficulty of handling the underlying

complexity and the differences in perception, it provides a framework in which to discuss values, anxieties and 'irrational' fears. This approach is informed by a world view, which holds that much truth is socially constructed, and that the traditional scientific approach is misleading, since all perception is influenced by the perceiver and all knowledge is subjective. A few take the extreme position in this more subjective approach, holding that all perceptions of the truth are equally valid and that there are no grounds for preferring one version over another, but this extreme offers no basis for prediction.

Neither of these perspectives alone provides a satisfactory basis for HIA. Prediction of the effects such as those of chemical pollutants on mortality or physical disease is probably best understood from the traditional scientific perspective. On the other hand, prediction of outcomes such as anxiety, amenity, social inclusion and quality of life may well be more amenable to approaches of a more subjective nature.

### **2.3.9 Uncertainty and HIA**

Prediction of the future is by its nature an uncertain business. Where predictions are based on epidemiological principles, using concepts such as exposure levels and dose response relationships the degree of uncertainty can be indicated with confidence limits and similar statistical tools. With well-defined models, the sensitivity to different elements can be explored by varying them and observing the effect on model outputs. Use of worst-case assumptions is a common device to reduce uncertainty. However, these apparently precise methods of describing the degree of uncertainty may be misleading. The degree of confidence that the presumed causal relationships are indeed causal, or that important variables have not been left out of the assessment, cannot be quantified and may be much more important than assumed.

While there are numerous problems associated with estimating the uncertainty attached to epidemiologically based predictions, the problems associated with sociologically based predictions are far greater. Here one generally has to resort to crude estimates as to which factors cause large impacts and which cause trivial ones in addition to intuitive assessments as to whether predictions are moderately certain, probable or possible.

### **2.3.10 Levels at which HIA takes place**

Decisions are made at many levels from supranational to local. International bodies such as the World Health Organisation, the World Bank and the European Union, national governments, regional governments, local authorities, health authorities, transport authorities, non-governmental organisations and many other bodies make decisions at their own levels. The ways in which decisions are taken and the capacity to make impact assessments may vary between levels and organisations, so that different approaches to HIA may be appropriate for each context.

Similarly, decisions vary in their scope. Policy decisions produce overall frameworks setting the goals for a particular area, laying out general direction and guiding how issues within that area should be determined. Decisions on programmes set in motion linked activities contributing to a particular goal. Project decisions cover a limited action such as construction of a particular waste disposal facility, a building, a piece of infrastructure, provision of a new service, changing management structures or mounting a communication campaign. All of these may have impacts on health and could benefit from HIA, though different applications may well need different assessment processes.



### **2.3.11 Quality criteria for HIA**

Those practising or commissioning HIA have to be concerned with its quality. Many descriptions of HIA include an audit step which involves reflection on the process. Quality criteria cover three areas (Kemmm, 2000):

- utility – did it assist decision making?
- predictive accuracy – did it correctly predict the health consequences?
- process – did it use appropriate processes? (Scott-Samuel *et al.*, 1998; McIntyre and Pettogrew, 2000).

## **2.4 HIA of policies and waste strategies**

This part of the Chapter examines the claim that HIA can assist in policy and strategy making, as opposed to decision making for individual projects. Because there are very few examples of the application of HIA to waste strategy, as opposed to waste facility projects, the evidence that HIA has contributed to policy making in any area, not just those related to waste disposal, is considered. It examines three aspects:

- the administrative systems in which an attempt has been made to utilise HIA
- the characteristics of the policy making process and how HIA could relate to it
- selected HIAs which claim to have been concerned with influencing policy.

### **2.4.1 Administrative systems in which an attempt has been made to utilise HIA**

#### ***Policy making in British Columbia (Canada)***

The government of British Columbia is often cited as an example of an administration in which HIA contributes to policy making. A tool developed in British Columbia for screening policies for health impact has been published (Population Health Resource Branch, 1994). However, it is not clear which policies were influenced by utilising HIA during their formation, nor are there descriptions of the development of specific policies illustrating the role of HIAs. It appears that the use of HIA in policy making has now fallen out of favour as a result of changing political concerns, change of administration and dispersal of HIA practitioners in the policy making teams (Banken, 2001).

#### ***HIA of the EU Common Agricultural Policy***

In 1996, the Swedish National Institute of Public Health undertook a HIA of various aspects of the EU Common Agriculture Policy covering the regimes for fruit and vegetables, dairy products, tobacco and alcohol (Dahlgren *et al.*, 1996). Following Sweden's accession to the European Union in 1995, the Swedish Ministry of Health and Social Affairs asked the National Institute of Public Health to undertake this study and analyse the public health impact of the European Union's Agriculture Policy. The study was undertaken by a group of six senior members of the Institute. They enlisted the help of seven international experts (three from the UK). They were also assisted by three senior civil servants from Swedish ministries. No European Union civil servants are listed in the contributors and it would appear that they were not involved in the assessment.

The Common Agriculture Policy was established in 1957 but has been subject to numerous reviews and adjustments. In 1992 (four years before the HIA), substantial reforms to the policy were introduced under the Agriculture Commissioner, Ray McSharry. Further reforms

were expected in 1997 and were presumably being considered at the time when the HIA was underway. The timing of this assessment was therefore appropriate to influence policy making. The HIA was commissioned by a ministry of the government of a new member of the EU, who could presumably have used it in policy discussions. It is not known to what extent this occurred. In many ways the document reads like an advocacy document addressing the concerns and agenda of public health rather than a policy-relevant document that addressed the concerns and agenda of policy makers in Brussels. Without detailed knowledge of the processes surrounding the production of the HIA and its presentation to the policy makers in Brussels it is impossible to assess how influential the HIA was. It is hard to avoid the suspicion that it had very little effect on the policy making process.

***HIA and national policy in the Netherlands***

The Netherlands has probably made more progress in using HIA as a tool for policy making than any other country. In 1993, the Ministry of Health in the Netherlands commissioned an expert report which recommended a trial of screening of national policy proposals for health impacts. The policy making process was analysed to determine the contexts and situations in which health considerations could influence it (Putters, 1996). An Intersectoral Policy Office (IPO) was set up with the Netherlands School of Public Health to develop HIAs for the Netherlands Government. The IPO has an annual budget of €340,000 (£220,000) and employs four staff members and a secretariat of two. It is responsible for screening policy development to identify situations for which an HIA would be helpful (i.e. case finding) and then commissioning HIAs.

The screening process involves an attempt to check all parliamentary documents (white papers, reports of committee meetings, budget papers etc) looking first at the title and then a more thorough reading of possibly relevant documents. The volume of documents to be scrutinised is large and efforts are being made to develop a search engine to reduce this workload. Following case findings there are negotiations with civil servants and it may be resolved to commission a Health Impact Screen (equivalent to a rapid appraisal or mini HIA) or an HIA. Subjects to which impact assessments have been applied are listed in Table 2.1.

**Table 2.1      *Subjects of Health Impact assessments in the Netherlands***

<p>Regulatory levy on energy (ecotax),          High speed rail link          Policy to discourage smoking          Alcohol licensing Act          Reduction in dental treatment covered by statutory insurance          The Budget 1997          Tobacco legislation          Political parties manifestos          Housing policy          ICES (Interministerial Economic Structure Strengthening Programme)          Policy related determinants of health          Working conditions Act and occupational health          The 24 hour economy</p>
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New coalition agreement
Social security
National Budget 1999
Fifth National Spatial Planning Policy Document
National Budget 2000
Housing Policy
National Budget 2001

The striking feature of this endeavour, which contrasts strongly with most other HIAs intended to influence policy, is the close link between the public health analytical expertise and the administrative machine it is trying to influence. It appears a far more robust mechanism than any described elsewhere. It is, therefore, disappointing to note that its chief exponents appear unconvinced of its effectiveness. They write ‘At the moment the Ministry is not really convinced that HIA is an applicable and usable tool with regard to the protection and improvement of health’ (Put *et al.*, 2001).

### ***HIA in Wales***

The National Assembly for Wales has been particularly active among UK regional governments in attempting to use HIA in its policy making (National Assembly for Wales, 1999). It has published HIA of the Welsh Home Energy Efficiency scheme (Kemmm *et al.*, 2000) and a ‘preliminary’ HIA of the Objective One programme (Breeze and Kemmm, 2000). Though there is no published evidence to assess the degree to which these activities influenced the relevant policies, the fact that the relevant civil servants were closely involved in the process of undertaking them and appear as authors suggests that their thinking was to some degree shaped by the HIAs. The report of this HIA on the Objective One programme is reviewed in the review of UK practice on page 115.

### ***HIA in England***

The document *Saving Lives: Our Healthier Nation* (DOH 1999) suggested that HIA should be used to assist policy development. Documents on other major policy areas such as transport (DETR, 1998), neighbourhood renewal (Cabinet Office, 1998) and modernising government (Cabinet Office, 1999a) have all endorsed the call for HIA. However, there is little evidence that HIA is currently being used. The government’s Centre for Management and Policy Studies recently published a guide to better policy making (Bullock *et al.*, 2001). It is notable that this document makes little reference to impact assessment and no mention of HIA. The main data collection for this survey took place in 2000 and the authors have made clear (personal communication) that they do not wish to suggest that HIA has no place in policy making. However, it appears that currently HIA is not making much contribution to policy making in England.

Policy makers are already required to apply several impact assessments to their policies (Cabinet Office, 1999b) and there is a fear that HIA could be seen by policymakers as just another bureaucratic hurdle to be overcome. The Department of Health (DH) and Defra have drafted an integrated impact assessment tool in which health is included alongside other impacts that have to be considered. This tool will soon be trialled as an acceptable way of screening policies for health and other impacts.

A guide to HIA published in the West Midlands is unusual in emphasising the link between the assessment and decision making steps of HIA (West Midlands Directors of Public Health Group, 2001) and a recently published guide from the Health Development Agency (HDA) (Taylor and Blair-Stevens, 2002) makes the same point. The present situation in England seems to be a wealth of ‘how to do HIA guides’ and a dearth of reports of completed HIAs.

### ***HIA in the Greater London Assembly***

The Greater London Assembly, assisted by the NHS London Regional Office, has a well-defined process by which all major policy and strategy proposals are subjected to HIA. The regional office is aware of the areas in which policy is being developed and so is able to do some preparatory searching of evidence. However, once the proposal has been published, the time allowed for the HIA is very limited (four to six weeks). A stakeholder group is called to review the policy and usually spends a day on this task, facilitated by public health staff. The staff will also review as much of the literature as can be covered in such a short time. The HIA is then available to the Assembly when they consider the policy proposal. The London Assembly has probably made more progress than any other national or regional authority in using HIA but it could be questioned whether involvement at an earlier stage in policy making might allow more opportunity for the policy to be influenced.

### ***The European Union***

HIA is not a formal requirement of policy preparation in the EU. However DG SANCO has published a screening tool, based on the work of the Netherlands School of Public Health, to help Directorates identify proposals which might be subject to an HIA. DG SANCO has invited other Directorates to use this tool but it is not clear to what extent the tool is being used (European Commission Health & Consumer Protection Directorate-General, 2001).

### ***Strategic Environment Assessment in Europe***

European Union Legislation has been influential in persuading national governments to require environmental impact assessment for many projects. The Espoo convention led to consideration of broader impacts (United Nations Economic Commission for Europe, 1991) and the Strategic Environment Assessments (SEA) Directive (European Parliament and the Council of the European Union, 2003) requires that much more consideration is given to health impacts than is necessary under the project-level Environmental Assessment Directive. The World Health Organisation has already produced advice on how to incorporate HIA into SEA (WHO European Centre for Environment and Health, 2001) although it is too early to analyse its effectiveness as the SEA Directive has only recently been implemented .

### ***Overview of attempts to utilise HIA in policy making***

The administrative contexts in which the application of HIA has been discussed are so diverse that it is premature to talk of best practice. In most cases, there has been little or no analysis of how HIA has influenced the decision making process in these examples. The experiences from the Netherlands and Wales emphasise the need for the HIA process to be owned by the policy makers and closely connected with their concerns. The fact that the British Columbia approach has been discontinued and the Netherlands approach is subject to major restructuring suggests that some administrations did not value them very highly. The methods for risk analysis in these examples have neither been fully described nor apparently been the subject of critical debate. With the exception of London, none of the approaches involved significant participation by anyone other than officials and professional experts.

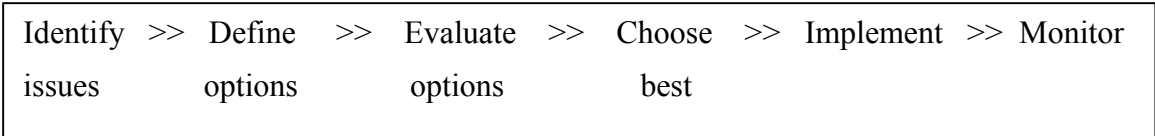
**2.4.2 The characteristics of policy making and how HIA could relate to it**

If HIA is to influence policy, then the HIA process must be designed to fit the stages of the policy making and meet the needs of the policy makers. The following aspects must be considered:

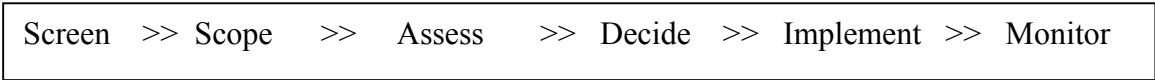
- the policy making process
- timing of HIA
- policy relevance
- proportionality
- relation of assessors and policy makers
- impartiality.

***The policy making process***

Many reviews of policy making suggest it is a linear rational and deductive process



The stages usually suggested for HIA seem designed to fit this model



However, the linear model shown above is only rarely appropriate as it is uncommon for administrations to attempt a ‘blue skies’ approach, in which the policy or strategy making process approximates to the linear model and the conventional approach to HIA is appropriate.

However, much more often, policy making is incremental, consisting of no more than marginal adjustments to existing policies and structures and limited to what is deemed possible on the basis of value judgements and careful negotiations with interested parties (Ham, 1992). In this model of policy making there is no obvious point at which HIAs should be commissioned or presented. Nonetheless, if it is to be useful, HIA must fit into this untidy process.

***Timing of HIA***

One characteristic of incremental policy making is that decisions are often made very rapidly as windows of opportunity arise and, consequently, HIAs may also have to be made with corresponding haste. Assessments, which do not conform to decision-making timetables, will not influence that decision.

***Policy relevance***

The findings of HIA have to be presented in a form that is policy relevant and addresses the concerns of the policy makers (O’Neill and Pederson, 1992). Decision makers will need to

know not only the predicted consequences of different options but also the degree of certainty that attaches to those predictions.

**Proportionality**

Another requirement of HIA is that it should be proportionate to the decision it is intended to influence. HIAs have a cost in time and possibly other resources. It is clearly unreasonable to use £10,000 worth of resources to assess a decision about the use of £20,000, whereas much greater resource expenditure would be justified for an assessment linked to a decision involving many million pounds sterling worth of expenditure.

HIAs vary in complexity and thoroughness (Parry and Stevens, 2001). They have crudely been classed as shown in Table 2.2. For many policy assessments a ‘mini HIA’ is adequate and frequently it is all that the time scale of decision making will allow. On the other hand, major policy areas may merit a full-scale, or ‘maxi HIA’.

**Table 2.2 Levels of HIA**

<p><b>Mini HIA</b> Synonyms: Rapid Appraisal, Health Impact Screening</p>	<p>Desk Top exercise Reliant on information already known Minimum quantification Limited consultation Time – Few hours</p>
<p><b>Standard HIA</b></p>	<p>Limited literature search Mostly reliant on routine data Quantify where possible More participation of stakeholders Time – Several weeks</p>
<p><b>Maxi HIA</b></p>	<p>Extensive literature search Secondary analysis of existing data Collection of new data Extensive quantification Full participation of stakeholders Time – Several months or years</p>

**2.4.3 Relation of assessors and policy makers**

Many descriptions of the HIA procedures intermingle assessment and decision-making steps (Scott-Samuel *et al.*, 1998) but frequently the assessment and the decision makers are different people. Often HIAs seem to have been carried out by groups of enthusiasts, sometimes in an academic setting. It is often unclear whether decision makers had any ownership of the assessment and whether the report has influenced decision making in any way. Frequently, the HIA process has involved joint working between staff of different agencies and it may be that there has been real influence that was difficult to recognise

because it happened through informal channels. It has been remarked that the involvement of decision makers in HIA has made them much more aware of health issues.

In many ways, the ideal solution to the problem of HIA being external to the decision making process would be for the decision makers to own and control the HIA process. This would ensure that it addressed issues relevant to them and occurred at relevant times. The argument against this arrangement is that it would be too easy for HIA to become tokenistic and avoid health issues where these were inconvenient in the achievement of other policy goals.

#### **2.4.4 Openness, impartiality and participation**

Openness is one of the treasured values of HIA practitioners and European policy is increasing people's rights to participate in decision making (United Nations Economic Commission for Europe, 1998). However, openness is not an attribute traditionally favoured by policy makers. Advice to ministers has usually been treated as highly confidential. The complex agendas and dealing, which are an essential feature of much political decision making, could be more difficult if everything were in the public domain. In many situations one may be forced to choose between an HIA which is open and ignored or one which is closed and highly influential.

Similarly, HIA practitioners claim to be impartial in predicting future impacts without favouring any party. It is feared that if HIA were left to policy makers, they would be unduly influenced by the views of their political masters or administrations. However, those who claim to be impartial are never free of value systems or notions of which claims are deserving or undeserving. Epidemiologists, toxicologists and adherents of the natural sciences sometimes mistakenly believe that their evaluations are objective and value free. However, the 'scientific method' implies a particular value set and issues such as which outcomes to consider, how to assess the acceptability of risk and how to trade off risks and benefits are clearly value laden. The goal must thus be an assessment informed by appropriate values rather than a value free assessment and to be partial to appropriate interests (such as the community) rather than impartial. Participation of stakeholders offers one way of determining values and the direction of partiality.

The benefits of a participatory process for HIA include:

- identifying causes of concern
- selecting outcomes for particular consideration
- making value judgements
- gaining access to lay expertise (Popay and Williams, 1996)
- contributing expert knowledge of local condition and life experience
- giving ownership of conclusions.

Even in technical areas, where non experts might be expected to have relatively little to contribute, there are numerous examples of lay views being persistently held in the face of expert opposition and eventually proving to be correct (Ozonoff and Boden, 1987; Brown, 1987; Brown, 1992). Ideally, participation in impact assessment not only leads to better decision making but also directly benefits the community by being a social learning process (Webler *et al.*, 1995; Daniels and Walker, 1996).

There are, however, major theoretical and practical difficulties in participation and involving stakeholders. Stakeholders being defined as all those who would be influenced by the policy are a very large group. Talk of 'public opinion' is misleading since there are many different

publics with different views which change over time. Which publics and which views should influence public meetings, meetings with community leaders, involvement of elected representatives, focus groups, citizen juries and opinion polls all have their limitations and problems. It is always difficult to give adequate voice to the inarticulate and diffident. All methods are time consuming and may be impossible to reconcile with the tight deadlines of the decision making process.

#### **2.4.5 Selected examples of HIA which have been purported to influence policy**

Though HIA claims to be invaluable for policymakers, it is difficult to find reports of cases where policies have clearly been influenced by HIA. Most HIA reports describe HIA of discrete projects rather than HIA of policies or strategies.

#### **2.4.6 Impact Assessments of policies or strategies not meeting the definition of HIA**

Many reports which are described as impact assessments of policies are retrospective and, while of great interest, do not fall within the definition of HIA used in this report. Some reports claiming to be impact assessments are a description of particular groups of health determinants rather than an attempt to predict the consequences of proposed policy options. Examples of this are a report on fruit consumption (Joffe and Robertson, 2001) and a report on general traffic strategies (Wolff and Gillham, 1991). Some reports describe processes designed to raise the interest of communities in health relevant aspects of their social and physical environment rather than assess any particular proposal. Examples of this are the PATH Community HIAs (Mittelmark, 2001; Gillis, 1999) and the HIA of the North Edinburgh Housing Strategy (Scottish Needs Assessment Programme, 2000a). Some reports described as impact assessments are clearly examples of advocacy rather than impartial assessment. Examples of this are the impact assessment of the US embargo on Cuba (Garfield and Santana, 1997; Barry, 2000) and the assessment of the EU Common Agricultural Policy (Dahlgren *et al.*, 1996), described earlier.

#### **2.4.7 HIAs which may have influenced policy making**

The earlier descriptions of use of HIA in Netherlands, in Wales and in London furnish several examples of HIAs, which are likely to have influenced policy.

Transport strategies furnish some examples of reports that meet the definition of HIA used in this report. A report on Edinburgh City Urban Transport Strategy (Scottish Needs Assessment Programme, 2000b) attempted to predict the consequences of three different options and analyses their differential effect on various sectors of the population. The public were not directly involved in the process though the representative of the local health council and a pressure group ('SPOKES') might be considered to have represented their interests. The working party that produced the report consisted not only of staff from the health authority but also staff from the city transport-planning department, so it would have been expected to influence policy makers. Even so, it is unclear to what extent it has been taken into account by policy makers.

A similar HIA of the Merseyside Integrated Transport Strategy ('MERITS') has been reported (Fleeman, 1999). A broad strategy for delivering integrated transport investment in Merseyside had been published and this HIA was attempted to predict its consequences. It produced a number of recommendations for measures to mitigate harmful impacts on those living and working near major routes and to enhance benefits for those reliant on public transport. The steering group for the HIA included representatives of the five local authorities



responsible for developing MERITS and representatives of ‘Merseytravel’. The level of representation from the decision-making authorities is not stated and once again it is unclear as to whether implementation of the policy was affected in anyway by the HIA.

It may be that truly influential HIAs do not produce reports since they are so closely integrated with the policy development process that there is no discrete process to be described.

## **2.5 Health Impact Assessment practice**

### **2.5.1 Health Impact Assessment practice in the UK**

Of the responses received to the 304 questionnaires sent to Environmental Health Officers and 326 to Planning Officers, 68 could be attributed to Environmental Health departments, and 84 were from Planning departments; the remainder could not be allocated to a specific department or were from Minerals and Waste departments (four) or were clearly the result of discussion between the Planning and Environmental Health departments.

Referring to the responses to question 1, 92 respondents claimed to understand what was meant by the term HIA, and of these, 50 were Environmental Health departments and 33 were Planning departments. Another way of viewing this figure is that 74% of Environmental Health departments understand what is meant by HIA, but only 39% of Planning departments understand this. Three out of four Minerals and Waste departments claimed to understand the term HIA. These results must be considered against the background that the understanding of the respondents was not tested, so we are unsure whether they do understand, or just think they understand the term. Also, it was clear from the responses that there was a lot of dialogue between different departments and so a single response may not be representative of just Environmental Health or Planning; indeed, the contact person for both of these is the same person for some Councils.

Further analysis was made of the perceived understanding of the term HIA between those local authorities with responsibilities for waste (Unitary Councils in England, Scotland and Wales, Metropolitan Borough Councils, London Boroughs and County Councils) where 50 out of 80 respondents (63%) claimed to understand the term and those without responsibilities (District Councils in England and Northern Ireland) where 42 out of 90 (47%) claimed to understand the term. Bearing in mind that responses from Environmental Health Departments and Planning Departments are combined in this analysis, it may suggest that the health implications of dealing with waste raises some awareness of potential tools to help in decision-making processes.

Of more interest to this study is the actual experience gained. For example, Question 2 sought to find out how many Councils were sufficiently aware of HIA that they had requested such studies to be carried out. Just twelve authorities responded ‘yes’ to this question: six of those responses were from Planners, four from Environmental Health departments and the origin of the other two were unknown. Bearing in mind the responses to Question 1, it might have been expected that councils with waste responsibilities would be the ones predominantly seeking HIAs; in fact, five of the 12 were English District Councils without such responsibilities.

Twelve respondents answered yes to Question 3 to indicate that HIAs had been submitted as part of EIAs. Of these responses, five were from Environmental Health departments and six from planning departments; six of the responses were from District Councils in England without waste responsibility.

Only two authorities (one English District Council and one Welsh Unitary Authority) answered yes to Question 4 to indicate that they had received a separate HIA with a planning application.

Six authorities answered yes to Question 5 to indicate they had carried out or commissioned HIAs of their own. The respondents included two Environmental Health Departments and three Planning Departments, from three English District Councils, two Metropolitan Borough Councils and one London Borough (the remaining respondent did not indicate which department they represented).

Five respondents answered ‘yes’ to Question 6 to indicate they had carried out or commissioned HIA of plans or programmes. The respondents included four from Planning Departments and one from Environmental Health and came from three District Councils in England, one Metropolitan Borough Council and one County Council.

The conclusions which can be drawn from this study is that experience in HIA is patchy at present, with only half of local authorities even claiming to know what it is, but with more awareness in Environmental Health Departments as opposed to Planning Departments. One explanation for this uneven split may be better access by Environmental Health Departments to the result of studies like that produced by the Small Area Health Statistics Unit (‘SAHSU’) on the possible health risks for populations living around landfill sites (SAHSU, 2001) which was cited by some of the respondents. Where authorities do have an understanding of HIA, actual experience is limited. There is no evidence to suggest that experience is being developed related to waste issues (as potential sources of health impacts).

**2.5.2 Lessons from the Canadian experience**

The Canadian Handbook on HIA can be accessed at:

<http://www.hc-sc.gc.ca/hecs-sesc/ehas/publications.htm>

and comprises a three-volume set:

Volume 1: *The Basics*

Volume 2: *Decision Making in Environmental Health Impact Assessment*

Volume 3: *Roles for the Health Practitioner*

Volume 1 considers an approach to HIA as part of the existing EIA procedure in Canada and introduces some types of health information and health indicators which might be used in EIA, these are all summarised in one table which is reproduced here as Table 2.3:

**Table 2.3 Types of health information and indicators for use in EA**

Group	Physical Health	Socio-cultural Well-Being
Public	Respiratory effects Noise Effects of accidents and malfunctions Rates of communicable and sexually transmitted diseases Cancer incidence Effects on fertility and development, including congenital anomalies	Changes in the quality or way of life Changes in cultural and social patterns Rates of crime Rates of drug and substance abuse Changes in stress levels
Worker	Injuries, effects of accidents and malfunctions	Changes in the quality or way of

Days off work or disability days	life
Long term activity limitations	Necessity for relocation
Respiratory effects	Stress-related conditions
Effects on skin (e.g., irritation, chloracne)	
Effects on fertility	
Cancer incidence	

It is, of course, stressed that the types of information and indicators used in HIA will depend on the type of project (or programme/plan/policy) and its possible effects.

Volume 2 is of particular interest because it deals with decision making and credibility issues. There is a chapter titled '*Credibility and Communication*' which includes, as an example, an indication of the typical questions asked by the public when faced with the development of an incinerator project – this information is reproduced below:

*Typical questions raised by the public when faced with the development of incinerator projects*

1. What are the specific risks compared to the benefits of the project? Is the risk to each group worth the benefit gained? What are the benefits and risks of alternative solutions? What are the benefits and risks of taking no action?
2. How did you calculate the risk? Is there one standard way of doing it, or are there several? Is there a prevailing consensus on the basic facts in the scientific community? Or are there distinguished dissidents?
3. Did you base your calculation on data from facilities already in operation, or is the database theoretical?
4. If you based the data on already-operating facilities, were they very similar to the proposed project? If not, how would their differences alter the analyses?
5. Does the design of the facility make the risk as low as it possibly can be? Can the facility be updated later if new ways are found to lower the risk?
6. Who in the community bears the burden of risk? Are older, younger, and sick people more at risk?
7. What is the chance of a serious accident? If one occurred, what would be the worst possible impact? How often do accidents happen in currently operating facilities? Will their likelihood increase over time? What is their magnitude? Would the effects of an accident be irreversible? What provisions have been made to handle accidents?
8. Will risks be identifiable? Who will monitor the performance of the plant? Can the risk be reduced?
9. Can the public influence how the facility is designed and operated?
10. Does approving the project mean foreclosing future, potentially less risky, options?

(Konheim, 1988)

This Volume also contains an appendix dealing with examples of health risk by economic sector, one of which is waste management and covers landfilling and incineration. The information presented here is potentially a good model to follow and includes matrices for both sanitary landfill and incineration. The format of the matrix is identical for both types of

development, whereby one axis deals with the ‘stressor’ or ‘exposure’ and includes: technological disaster; gaseous or air emissions; liquid emissions or discharge into water; solid emissions or discharge into the soil; nuisances; indirect impacts or other exposure. The other axis covers different issues for each of the stressors and includes: type of stressor; environmental impact; area of influence; control measures; standards or recommendations; effect on health; population at risk; probability of occurrence; environmental/biological indicator (monitoring); information/references. Each cell in the matrix has been completed with helpful advice or with relevant standards or facts.

The value of the advice in Volume 3 would depend very much on the model developed for HIA in the UK as it specifically looks at the potential roles of health practitioners in the process – but this is written for the Canadian - and not the UK - situation.

### **2.5.3 Lessons from UK experience**

The questionnaire survey has indicated that experience is limited in the UK, although some examples of HIA having been carried out were identified. These examples have been followed through and attempts made to track down the documents with a view to analysing their content. Appendix B sets out a brief analysis of the documents obtained which are:

- Wrexham Resource Recovery Centre
- Integrated Recycling Facility at Sydalit
- Shortstown Development, Bedfordshire Health
- Change of use of the former Psychiatric Hospital, Maida Vale
- Community Safety Projects, Huyton Area
- Alconbury HIA
- National Botanic Garden of Wales
- The health potential of the Objective 1 Programmes for West Wales and the Valleys

## **2.6 Conclusions**

Based on the survey of local authorities, it is clear that experience with HIA is better developed in Environmental Health departments than in Planning departments, despite the fact that it is the Planning departments who will be dealing with the development proposals potentially affecting human health. Of all the responses received, only one (Lancashire County Council) referred to WISARD (a software tool for waste management planning), and it does appear that they have used the tool to consider health as part of environmental impacts in their Waste Strategy.

A number of HIA documents have been analysed for their relevance to this study. The Merseyside Guidelines for Health Impact Assessment (which can be downloaded from <http://www.hiagateway.org.uk/Resources/toolkits/toolkits.asp>) have played a significant part in much of the experience gained in the UK and have provided the procedural basis of many studies. This has a clear implication for stakeholder involvement in that a ‘key informant’ approach is recommended, with ‘brainstorming’, then this is used to identify key issues as a precursor to prioritisation. Brainstorming tends to be focused by reference to a table of key areas influencing health, and it is possible that this list could be amended to incorporate advice from the Canadian HIA guidance.

Whilst the Merseyside guidelines have been helpful in encouraging stakeholder involvement and open participatory processes, they may be criticised for paying insufficient attention to epidemiological methods. HIAs applied to waste management frequently need to resolve conflicting views on likely consequences of proposals, and the Merseyside guidelines offer little guidance on this point. However, where the Merseyside Guidelines have not been used, stakeholder involvement seems to have been limited to project teams and consultants only and the comprehensiveness of the factors considered is questionable.

Prioritisation of health issues for further studies is an important step in HIA, but still leaves open the question of techniques to be used to predict meaningful outcomes for these issues. Of particular relevance are air quality impacts, which might be associated with incinerator projects or landfill projects. Several sources have been mentioned which could form the basis for prediction:

- Committee on the Medical Effects of Air Pollutants (COMEAP)
- United States Environmental Protection Agency Human Health Risk Assessment protocol for hazardous waste combustion facilities
- WHO three cities approach (Dora *et al.*, 1999).

Thus, HIA is clearly an evolving procedure with clear evidence of low levels of take-up by other relevant agencies, such as local authorities. The potential divergence in interpretation of the goals and methods of HIA, which derive from the integration of scientific principles (and their associated uncertainty) within a socio-political and socio-economic context, may confound, or possibly determine, the main health outcomes.

Despite this, there is a growing requirement for the application of HIA as part of the planning process and for policy development and evaluation. A consideration of HIA must be informed by an understanding of the resource implications. There is a significant opportunity for this project to influence the broader implementation of HIA to result in practical environment and health enhancement.

The evidence of HIA applied at a Policy level is too patchy to draw conclusions on best practice. This is because in administrative systems where progress has been made, tools have been suggested or statements made about the role of HIA, but experience is limited and does not allow an assessment of best practice.

Evidence from project-based HIA in the UK leads to a number of recommendations for best practice:

- stakeholder involvement is essential if HIA is to focus on the issues of concern to the public - if this does not happen, then it will be difficult to gain public acceptability
- there needs to be a clear justification of all the issues to be studied in a HIA
- HIA is more effective if applied to different alternatives before decisions are made; the implications for HIA of Waste Management in the UK are that HIA must apply to the strategy level before it applies to individual projects; furthermore, when it is applied to individual projects, different locations must be given equal consideration
- the evidence base is often weak for a HIA, but confidence in the assessment can be enhanced by the application of quantified techniques (such as COMEAP) where they can be applied. Such evidence needs to be placed in context by involving the public in the identification of key health determinants

- For reference, the Health Development Agency has produced its own generic guidance on HIA (Taylor and Blair-Stevens, 2002) which supports a flexible approach which can be adapted to local circumstances. The Health Development Agency supports the website referred to above (Section 2.6 para 2) the HIA Gateway, which holds copies of HIA reports and provides useful advice, including new approaches for carrying out HIA (<http://www.hiagateway.org.uk/>), and a similar website is being developed by WHO (<http://www.who.int/hia>).

### 3 Landfill

This and the following three Chapters (Chapters 4 to 6) investigate particular waste treatment methods; Chapter 7 looks at potential implications on community mental health arising from concern over the treatment of waste. These Chapters were produced using a common approach to the identification of relevant research literature described in [Appendix C](#). The Chapters are supplemented by a review of the health effects of sixteen key chemical contaminants associated with waste disposal and this is detailed in [Appendix D](#).

Landfilling for disposal of municipal solid waste will continue to be the major waste disposal process for many years, despite the increasing role of recycling, composting and incineration. The major components of municipal waste are paper and card (26%), garden waste and putrescibles (21%), kitchen waste (8%), plastic (7%), glass (6%), metals (6%), and potentially hazardous waste (0.5%) (Freeman, 1989; Eduljee, 1998). The EC Hazardous Waste Directive requires that non-hazardous and hazardous wastes are not subject to co-disposal after 2004.

The bulk of landfilled special waste (i.e. those materials or substances on the Hazardous Waste List) consists of inorganic thermal process waste (27%), construction and demolition waste and asbestos (26%), oil mixtures (19%), inorganic chemical process wastes (9%), and organic chemical processes wastes (9%) (Welsh Assembly Government, 2002).

Within the landfill a complex sequence of chemical and biological processes produces liquid and gaseous emissions from the parent waste. In older landfills, contaminants may be leached from the solid waste by water producing contamination of surface and groundwater. Modern landfill designs incorporate leachate containment, using geomembranes and low porosity materials such as bentonite. Leachate chemistry is highly variable but organic compounds detected within it include organic acids, aromatic compounds, chlorinated aromatic compounds, halogenated aliphatic compounds, pesticides, polyaromatic hydrocarbons, polychlorinated biphenyls and organophosphates (Brown and Donnelly, 1988; Assmuth T, 1992). The class of organic compounds found at highest concentration in leachates is generally volatile fatty acids produced during decomposition of proteins and carbohydrates, but benzene, toluene, ethylbenzene, xylene (all often termed 'BTEX' compounds) are commonly found at lower concentrations (Schulz and Kjeldson, 1986). Heavy metals including zinc, copper, cadmium, lead, nickel, chromium and mercury are also contaminants of leachate.

Much organic waste is converted to gaseous products, termed 'landfill gas' (LFG) which contains approximately 40-60% methane, 30% carbon dioxide (CO<sub>2</sub>) and trace gases such as hydrogen sulphide (H<sub>2</sub>S). Many trace concentrations of toxic volatile organic compounds (VOCs) are also observed in landfill gas, including halogenated aliphatics, heterocyclic compounds, aromatics and ketones (Wood and Porter, 1987; Brosseau *et al.*, 1994; El-Fadel *et al.*, 1997). The Environment Agency has funded an R&D project 'Investigation of the Composition and Emissions of Trace Components in Landfill Gas' (Parker *et al.*, 2002). This reviewed UK data on trace components of landfill gas in addition to gas sampling at one landfill site. A priority list of substances was obtained by scaling the published toxicological properties of trace components against the average concentration reported in the literature, augmented by the empirical data acquisition at the single site.

The US Agency for Toxic Substance and Disease Registry (ATSDR) have characterised emissions from hazardous waste sites and developed a health research programme to study environmental exposures to hazardous substances (Johnson, 1997; De Rosa *et al.*, 1998; Amler and Lybarger, 1993). It is known that approximately 100 trace chemicals can be

present in leachate or landfill gas at such sites (De Rosa *et al.*, 1996). People in the vicinity of hazardous waste sites may also be exposed to chemical mixtures (Johnson and De Rosa, 1995, Hansen *et al.*, 1998, Etkina and Etkina 1995).

Solid wastes and landfill leachates have been reported to contain approximately 10 faecal coliform colony forming units per millilitre (cfu ml<sup>-1</sup>) and 1,000 faecal streptococci cfu ml<sup>-1</sup> with *Clostridium perfringens* the predominant pathogen although *Clostridium* spp., *Salmonella* spp. and *Listeria* spp. were also found (Donnelly and Scarpino, 1984). Outdoor air-borne bacterial densities observed at five sanitary landfills in Finland during the summer and autumn ranged from 50-17,000 colony forming units per cubic metre (cfu m<sup>-3</sup>) and mesophilic fungal concentrations ranged from 500 to 5,600 cfu m<sup>-3</sup> (Rahkonen *et al.*, 1987). These levels are comparable to those measured in the vicinity of composting facilities as detailed in section 5.2.2. below. Conditions within landfills are very heterogeneous and the size and composition of the microbial population is not known with any certainty. Microbial communities which develop on the solid material in landfills may not be the same as communities developing within the liquid leachate (Archer *et al.*, 1995). Landfill leachate may also affect the composition of extant microbiota in aquifers (Ludvigsen *et al.*, 1999).

Each landfill site is unique with respect to age, quantity and type of waste contained, local meteorology, hydrogeology and engineering control of leachate and landfill gas (Reinhard, 1993). Toxic pollutant emissions can be minimised through optimisation of biodegradation, leachate and gas collection and treatment.

### 3.1 Emissions and exposure

Trace gas concentrations in LFG have been derived from various data sources (e.g. AERC 2001) although other data sources are also available (Environment Agency, 2002a) and may be used instead. A software tool ('GasSim') (Environment Agency 2002c) developed for the Environment Agency (Gregory *et al.*, 1999) takes the emissions output and simulates atmospheric dispersion to assess the potential exposure of residents in proximity to the landfill. The Environment Agency has also initiated monitoring of air pollutants at the site boundaries of two landfills (Environment Agency 2003a).

The reporting of annual emissions from most landfills is necessary under the Pollution Prevention and Control (England and Wales) Regulations 2000. There is, therefore, a requirement to measure, or estimate, annual mass emissions to air of Pollution Inventory (PI) substances from landfills. In order to facilitate this, a reduced utility version of GasSim ('GasSim Lite') has been made available by Golder Associates on behalf of the Environment Agency, which estimates annual mass emissions to air of PI substances from landfills (Golder Associates, 2002). GasSim Lite determines the generation of LFG based on the mass of waste deposited and its composition for an individual site using the same input characteristics as the full GasSim model. The emission model takes this output and uses it to calculate LFG emission of bulk gases to the environment after allowing for LFG collection, flaring, energy recovery and biological methane oxidation.

A number of potential exposure pathways may contribute to the exposure of people to contaminants from landfills.

Airborne exposure may lead to inhalation of LFG or emissions from LFG flares and/or particulate matter. Air quality guidelines have been published by the World Health Organisation (WHO) and the UK Expert Panel on Air Quality Standards (EPAQS) for a number of substances including, sulphur dioxide, nitrogen dioxide, carbon monoxide,



benzene, and PM<sub>10</sub> and by WHO for hydrogen sulphide and toluene. Other substances such as mercaptans, xylene and ethylbenzene have no associated air quality guidelines as yet. Respiratory health may also be affected by inhalation of particles that penetrate the respiratory system beyond the larynx i.e. particles less than 10 microns in diameter (PM<sub>10</sub>) (Parkes, 1982). The adverse respiratory effects of these particles may be enhanced by contaminants adsorbed onto the particle surface. A proportion of these PM<sub>10</sub> particles is cleared from the lung by the mucociliary escalator and is subsequently swallowed, and a proportion of larger particles is directly ingested. Chemical contaminants and heavy metals may be adsorbed or adhered to such particles and may therefore produce an ingested dose.

Water-related human exposures may occur through direct ingestion, dermal contact or by consumption of produce irrigated (and/or manufactured with) contaminated water (Assmuth, 1996). If public water supplies (i.e. those supplied by a water undertaker regulated by the Drinking Water Inspectorate (DWI) under Water Quality Regulations (HM Government, 2000)) were to be affected by leachate chemicals, this would be quickly identified by routine monitoring. Private water supplies (PWSs), particularly small domestic supplies, are monitored much less frequently by Local Authorities (LAs) under the requirements of the Private Water Supply Regulations (1991). LA Environmental Health professionals would be responsible for deciding if a landfill caused a health risk to consumers of PWS and, if necessary, initiating supplementary water quality analyses.

Soils may also be contaminated through atmospheric deposition of landfill emissions, or pollution transport by surface water. Exposure of children to contaminated soil is of particular relevance since young children are known to ingest greater quantities of soil and dust particles than adults through inadvertent ingestion of dust adhering to their hands. Risks associated with dermal exposure to contaminated soil are not well characterised but relative loads on non-hand skin have been determined in field measurements (Holmes *et al.*, 1999) and about 37% of total skin surface is estimated to be exposed during young children's warm weather outdoor play (Wong *et al.*, 2000). In a non-random sample of 64 children between one and four years of age who resided in an academic community in Massachusetts, mean soil ingestion estimates were 45mg day<sup>-1</sup> for 50% of children (Stanek and Calabrese, 1995).

The transport of landfill gas through the geosphere may be modelled to determine contamination in soils at distance from the landfill site (Gregory *et al.*, 1999). The Department for Environment Food and Rural Affairs (Defra) has produced the Contaminated Land Report (CLR) series of documents to assist with the assessment of health risks arising from contamination in soils (Defra, 2002a, 2002b, 2002c; Defra and Environment Agency, 2001).

### **3.2 Potential health effects**

In the USA, results of public health assessments conducted at 167 waste sites during 1993 to 1995 showed that about 1.5 million people had been exposed to site-specific contaminants.

At 10% or more of the sites that had completed exposure pathways, 56 substances of interest were identified. Of these, 19 are suspected human carcinogens and 9 are associated with reproductive or endocrine-disrupting effects (Hansen *et al.*, 1998), but some health effects are known to be synergistic and the potential importance of additive effects of chemical mixtures is highlighted both by in-vitro experimentation and evidence from animals. However, the scientific basis for identifying synergism (and associated health effects) has yet to be established. For example, mixtures of organochlorine compounds produced enhanced oestrogenic activity in human breast cells (Soto *et al.*, 1994) and combinations of PCB

compounds showed elevated adverse effects on the developmental stage of reptilian eggs (Bergson *et al.*, 1994). It is notable that the Agency for Toxic Substances and Disease Registry (ATSDR) also has a programme investigating chemical mixtures and mechanism-based interaction and synergy (Johnson *et al.*, 1995).

This body of evidence has led to concern about health effects associated with exposure to substances from landfill sites (Smith *et al.*, 1995; Zejda, 1998; Rushbrook, 1994; Carpenter, 1994; Wyes, 1997). The ATSDR has sought further to research the area by developing a Priority List for Hazardous Substances (Johnson, 1997). It has also used information from public health assessments and toxicological profiles to develop a list of seven Priority Health Conditions (ATSDR, 1992): these are birth defects and reproductive disorders, cancers (selected sites), immune function disorders, kidney dysfunction, liver dysfunction, lung and respiratory diseases and neurotoxic disorders.

The particular vulnerability of children and pregnant women is a principal consideration when considering health effects. Children have disproportionately high exposures to environmental toxicants because, per unit body weight, they drink more water, eat more food and breathe more air than adults. Their ability to metabolise, detoxify and excrete toxicants is also different from that of adults (Landrigan *et al.*, 1999). The foetus is also known to be at elevated risk from exposure to toxicants at certain key stages: in the period three to seven weeks post conception, toxic exposures can produce major structural defects such as cardiac abnormalities and neural tube defects (Kipen, 1996). A different spectrum of disorders is more characteristic of toxic effects during later periods in pregnancy: these include low birth weight and functional disorders (Bellinger *et al.*, 1987) and transplacental carcinogenic effects (Herbst *et al.*, 1971).

The Department of Health has commissioned a review of the potential teratogenicity of substances emanating from landfill sites (Sullivan *et al.*, 2001). This provides an overview of the potential for developmental toxicity for thirty three chemicals, but covers only the hazardous properties of the chemicals and does not include risk assessments, for which exposure data caused by landfill site emissions would be required.

### **3.3 Epidemiological evidence of health effects**

The epidemiological evidence of health effects associated with exposure to substances from landfill sites has been the subject of a number of recent reviews (Vrijheid, 2000; Johnson, 1997; Johnson, 1999; Applied Environmental Research Centre Ltd., 2000; South West Public Health Observatory, 2002).

Most studies of landfill sites have focused on hazardous waste sites rather than household or domestic waste sites and many of these have studied sites with relatively high emissions. Public concern in the vicinity of landfills has prompted a number of single-site studies (Kharrazi *et al.*, 1997; Berry and Bove, 1997; Najem *et al.*, 1994; Kilburn, 1999; Williams and Jalaludin, 1998). These are prone to respondents' recall bias and are limited in statistical power due to the size of population residing in the vicinity of a single site. A number of ecological (geographic comparison) studies have also been undertaken which have examined rates of adverse health outcomes in counties containing waste sites and compared these to state or national rates (Harmon and Coe, 1993; Schwartz *et al.*, 1998). Retrospective case-control studies have also been undertaken (Polednak and Janerich, 1989; Marshall *et al.*, 1997). In these studies, exposures of people with disease (i.e. cases) are compared to exposures of people without disease (i.e. controls).

To increase statistical power, several multi-site studies have been undertaken where sites have been selected independently of community concerns or reported disease clusters. A collaborative European study ('EUROHAZCON') examined the association of non-chromosomal congenital anomalies with 21 hazardous waste landfill sites. In this study, a 'proximate' zone of 3 km radius from the site (within which it was assumed that most exposure to chemical contaminants would occur) was compared to a zone of radius 3-7 km from the site. A 33% increase in the risk of non-chromosomal anomalies for residents living within 3 km of the sites was reported (Dolk *et al.*, 1998). Recently, a similar analysis for chromosomal anomalies suggested a comparable level of risk to that found for non-chromosomal anomalies (Vrijheid *et al.*, 2002).

In Great Britain, risks of adverse birth outcomes in populations living within 2 km of 9,565 landfill sites which were each operational at some time between 1982 and 1997 were compared with those in a reference population who resided more than 2 km from all known landfill sites. For all congenital anomalies combined, the relative risk for residence near landfill sites adjusted for confounders (e.g. social deprivation) was 1.01 (Elliot *et al.*, 2001). The precision of the estimate of the relative risk is high as a consequence of the large sample size involved. The confidence interval (CI) of estimates is important and defines (at a specified level of probability) the range in which the estimate of the relative risk will lie. For all congenital anomalies combined, 99% confidence interval for relative risk is in the range 1.005 to 1.023.

Somewhat higher relative risks were found for specific anomalies: neural tube defects 1.05 (99% CI 1.01 – 1.10); abdominal wall defects 1.08 (99% CI 1.01 to 1.15); and hypospadias and epispadias 1.07 (99% CI 1.04 – 1.10). The authors also compared different types of sites i.e. 7,803 sites for non-special waste and 774 sites for special waste. For special waste sites, relative risks increased when compared to non-special waste sites: all anomalies 1.07 (99% CI 1.04 to 1.09) compared to 1.02 (99% CI 1.01 to 1.03); neural tube defects 1.07 (99% CI 0.95 to 1.20) compared to 1.06 (99% CI 1.01 to 1.12); hypospadias and epispadias 1.11 (99% CI 1.03 to 1.21) compared to 1.07 (99% CI 1.04 to 1.11). For abdominal wall defects and for cardiovascular defects, the comparison could not be made as results were not statistically significant. It is important to realise that although one can say that the relative risk exceeds unity with a very high degree of statistical confidence, this does not prove a causal link.

Differential identification of congenital malformations by hospital has been shown to be an important confounding factor (James, 2003) and misclassification of exposure may also be a factor (McNamee and Dolk, 2001).

In the US, a 12% increase in congenital malformations was reported for women residing within 1 mile of 590 hazardous waste sites in New York State (Geschwind *et al.*, 1992). A follow-up study, with improved study design, found no association between potential exposures from hazardous waste sites and risks of musculoskeletal and central nervous system birth defects (Marshall *et al.*, 1997). In a larger US study of women living within 1 mile of 1,281 sites over the entire United States, no increase in congenital malformations was observed (Sosniak *et al.*, 1994). The lack of information on alternative pollution sources hampers the interpretation of all such multi-site studies.

Most multi-site investigations have concentrated upon congenital malformations, but increased bladder cancers and leukaemias have been reported in women residing in areas likely to be exposed to landfill gas (Lewis *et al.*, 1998). Renal disease was investigated in people residing within 1 mile of 37 sites in New York State, but the evidence for increased incidence of kidney disease did not achieve statistical significance (Hall *et al.*, 1996).

There have also been a large number of health surveys which have relied upon residents reporting symptoms through questionnaires (Vrijheid, 2000). Increased incidence of reported symptoms in exposed areas may be subject to reporting bias, but nevertheless it indicates the impact that perceived risks may have on health.

### 3.4 Critical appraisal of risk assessments

Reviews of a number of site-specific risk assessments of landfill facilities have been undertaken. In general these correctly identify the risk to human health presented by specific toxicants and also particular exposure pathways. For example, air dispersion modelling is sufficiently developed to predict exposure at specific locations, or over defined areas, and, groundwater quality can also be modelled well with existing tools. Risk assessments, in general, correctly employ the most conservative (i.e. maximum exposure) values for estimated toxicant concentrations, but some aspects are frequently overlooked:

- One hundred per cent absorption of contaminants adhering to PM<sub>10</sub> particles by inhalation is commonly assumed, but the potential significance of larger particulates is frequently overlooked. For example, it is known that a proportion of airborne lead, which is trapped in the upper respiratory tract, is removed to the throat by ciliary action and may be swallowed and ingested. Therefore, for heavy metals and carcinogens, it is appropriate to estimate the amount ingested via this route.
- For dioxin, calculation of 'Toxic Equivalent' should be based on the new WHO 'Toxicity Equivalency Factor' scheme. Risk assessment procedures typically consider indirect routes such as deposition on vegetation and subsequent ingestion. A more sophisticated approach needs to be developed for indirect exposure. For example, the bioaccumulation of dioxins in the tissues of a lamb eating a representative amount of vegetation for a representative period could be calculated. The doses arising from human consumption of lamb could then be estimated.
- Perhaps the most susceptible group to dioxins are breast fed babies since, during breast feeding, a very significant proportion of a mother's lifetime dose of dioxin is transferred to the baby. Although most of the mothers' burden of dioxin will have accumulated from dietary intake, the extra accumulated burden from chronic (i.e. many years') exposure to environmental dioxin arising from landfill activities should also be estimated.
- Some risk assessments propose limits at the site perimeter for exposure of the public based on Occupational Exposure Standards (OES). This is inappropriate as OES values need to be adjusted, first to take account of residents' exposure for twenty four hours and seven days a week and second, to accommodate susceptible groups in the general population which are not represented in an occupational setting.

Risk assessments should focus more on susceptible groups in the population such as children. For example, the WHO guideline (WHO, 1998a) for maximum daily intake of dioxins from non-dietary sources equals 0.1 to 0.4  $\mu\text{g kg}^{-1}$  body weight. The United States Environmental protection agency (USEPA, 1999) suggests a value of 10  $\text{m}^3$  per day as the typical respiration rate for a child and 20  $\text{m}^3$  for an adult. For inhalation exposure, this suggests twice the risk for an adult compared to a child, but the relative weights involved result in a higher maximum daily intake for children per unit weight.

## 4 Incineration

### 4.1 Introduction

Modern, well-managed incinerators can be an effective means of reducing and disposing of waste materials so that any potential health risk is minimised. However, the by-products of the combustion process may contain hazardous or toxic pollutants and emissions will add to background pollution levels. As a result, there is often considerable public concern over the possible health effects of living near to incinerators processing hazardous, clinical or municipal waste. This Chapter presents a review of epidemiological studies of the public health effects of waste incinerators.

There is no doubt that air pollution (from all sources) can have an adverse effect on the health of susceptible people (i.e. young children, the elderly and particularly those with pre-existing respiratory disease). Recent work in the UK by the Committee on the Medical Effects of Air Pollutants (COMEAP) has demonstrated that exposure to air pollution can bring forward death in patients with severe existing disease, although the degree of life shortening is typically of the order of a few weeks, at most, per individual. However, there is currently little convincing evidence that ambient levels of air pollution can cause acute adverse health effects in healthy people. Particulate matter has also been shown to be associated with chronic (long-term) effects.

Many of the epidemiological studies of possible health outcomes, in populations living close to incinerators, have not given clear indications of the presence or absence of an effect. Of necessity, many of the studies examining possible health effects are retrospective and employ routinely collected data such as cancer registrations, in addition to birth and death records. Whilst such observational studies can provide evidence of an association between a health outcome and an environmental pollutant, they cannot, by themselves, demonstrate a cause and effect relationship. The interpretation of these findings is also crucially dependent on well-known limitations, including possible sources of bias and confounding, together with the ever-present difficulty in obtaining reliable and accurate population exposure data.

Direct measurements of exposure from incinerators are seldom made and often the distance from the incinerator site is used as a proxy for exposure, a technique that can be very unreliable. Many studies use concentric circles to identify 'at risk' populations, a technique that does not take into account the influence of meteorological conditions or process characteristics (e.g. stack height, efflux velocity and plume temperature). Furthermore, the zones of influence used, which can be up to 7.5 km from the site, introduce considerable possibilities for confounding co-exposures from other industries.

Another problem is that most studies are, by their very nature, *post hoc* since they were prompted by complaints of apparent 'clusters' of ill health in areas around incinerators. As a result, unintentional bias (such as in the reporting of health outcomes) can be built into the study, which can weaken the result. In addition, many studies analyse a small number of cases which reduces the statistical power to detect an association between exposure and ill-health.

Another factor that needs to be taken into account is that the majority of the studies, and any associated environmental data, originate from incineration facilities whose emission profile was significantly different from today's modern incinerators. Up until the mid-1990s, incinerators in the UK were fitted with rather rudimentary emission controls and therefore emitted quite significant amounts of air pollutants. Newly constructed incinerator plants have to meet much stricter controls on emissions and are significantly cleaner. Where available, we have included details of the period of operation.

The methodology used to identify the relevant literature in the field detailed in [Appendix C](#) led to the identification of 23 epidemiological studies which were reviewed. The search methodology was supplemented by a literature review by staff in the Division of Environmental Health and Risk Management (University of Birmingham).

#### **4.1.1 Method**

Each study was assessed using a critical appraisal according to study type, sample size, exposure definition and measurement, outcome and control for confounding factors. A total of 23 epidemiological studies were evaluated (those references are denoted by \* in the reference list). In addition, four review papers were considered. Most weight was given to those studies that took into account potential confounding factors, had a valid means of estimating exposure from the incinerator (since none had measured values of exposures) and had sufficient statistical power to produce results with a small confidence interval (i.e. had studied a sufficiently large number of people to distinguish a genuine effect from random variations in the prevalence of disease within small population groups).

In 2000, the Department of Health Committee on Carcinogenicity agreed that there were a number of factors that should be considered in deriving conclusions from studies of municipal solid waste incinerators (Committee on Carcinogenicity, 2000):

- accuracy of health statistics
- accuracy of cancer diagnosis
- potential confounding factors for individual cancers
- variables particular to incineration, such as type of waste burnt, geographical and meteorological conditions, and controls placed on the emission of pollutants.

These factors form the basis of this literature review.

## **4.2 Potential pathways and exposure routes**

The general public can be exposed to pollutants associated with incinerators through a number of routes, with direct inhalation and indirect entry via the food chain of particular importance. For many pollutants including some of the trace metals, and carcinogenic organic compounds (such as dioxins and furans), the major route of exposure is through the food chain.

People in the UK with existing respiratory and cardiovascular diseases may have their illness exacerbated by acute exposure to air pollutants. Children and the elderly are also particularly vulnerable to air pollution. Exposure via the food chain may be a potential problem if locally grown or reared produce is important to the diet of local people. Groups such as local allotment owners and farmers may need particular consideration.

## **4.3 Possible health effects associated with the process**

### **4.3.1 Cancer**

#### ***Introduction***

Studies in the UK have principally focused on the possible effects of living near to the older generation of incinerators, although all incinerators can emit known or suspected human carcinogens, albeit in small quantities.

Most concern has focused on the effects of exposure to dioxins and furans and polycyclic aromatic hydrocarbons (PAHs), substances that are known or suspected carcinogens. It has been hypothesised that exposure to dioxins and furans (either directly via inhalation or indirectly via the food-chain) are major causes of cancer in communities around incinerators. Whilst older incinerators were often significant sources of dioxins and furans in the local environment, modern incinerators are significantly cleaner. A recent study around a modern incinerator in Spain could find no difference in the levels of exposure (based on blood samples) in residents living near to the incinerator and those living further away (Gonzalez *et al.*, 2000).

### ***Stomach, colorectal and liver cancers***

Several epidemiological studies have suggested a possible association between incinerator emissions and stomach, colorectal and liver cancers. In the UK, a possible distance-related link with the old generation of incinerators has been reported (Elliot *et al.*, 1996). This large study examined cancer incidence in over 14 million people living near to 72 municipal solid waste incinerators between 1974 and 1986 (England), 1974 and 1984 (Wales), and 1975 and 1987 (Scotland). Age standardised observed/expected (O/E) ratios were calculated for radii up to 7.5 km from each incinerator and five and ten-year lag times for the onset of the cancers were assumed. After applying control factors for social deprivation, a significant decline in risk with distance from the incinerators was found for all cancers combined, and particularly for stomach, colorectal and liver cancer. However, incomplete control for socio-economic confounders may have been responsible for these results and once the authors took into account a number of *post hoc* estimates, such as examination of the data before the incinerators were built and estimation of the likely impact of ethnicity, only liver cancer showed a significant association with distance from the incinerators. In this case, it is likely that misclassification of secondary tumours (i.e. tumours due to migration of cancer from another site within the body) as primary liver cancer may have caused or contributed to the result.

Given the uncertainties that surround this reported excess of liver cancers, particularly the possibility that misclassification of tumours contributed to the outcome, the data were re-examined (Elliot *et al.*, 2000). Although this study could not completely discount the possibility of an association between residential proximity to municipal solid waste incinerators and incidence of liver cancer, confounding from socio-economic deprivation (primary liver cancers in Great Britain are strongly related to social-economic deprivation) further weakens this outcome, making identifying a causal link impossible. Even if such a causal link could be proved, this excess relates to historical exposure patterns around older incinerators and not current or future incinerators.

In both of these studies, direct measurement of the pollutants was not available and the distance from the incinerator site was used as a proxy for exposure, which is far from ideal.

The waste solvent and oil incinerator at Charnock Richard, Lancashire, which operated between 1972 and 1980, has been much studied. Gatrell and Lovett (1992) reported a possible excess of cancer of the stomach and larynx after examining cancer registrations between 1974 and 1983. However, little weight can be given to this study as it did not adequately control for confounders (e.g. socio-economic deprivation). The authors themselves cautioned against inferring a causal link between cancer incidence and residence near the incinerator.

## **Cancer of the larynx and lung**

The possibility of a cancer cluster, particularly of cancer of the larynx, near to the Charnock Richard incinerator prompted a more detailed study which included the other nine UK incinerators licensed to burn waste solvents and oils (Elliot *et al.*, 1992). Cancer registration data were used to identify cases of cancer of the larynx and lung within 3 km of the sites, and between 3 and 10 km. Expected values were calculated using regionally adjusted national cancer rates to enable age standardised O/E ratios to be derived. Cancer of the lung was also included in the study because it shares many of the same epidemiological characteristics as cancer of the larynx, including a strong social class gradient, a strong association with cigarette smoking and with similar occupational risks. However, despite initial reports of a cluster, neither cancer of the larynx nor of the lung showed a statistically significant relationship with distance from the site, once socio-economic status was taken into consideration. Correction for confounding by socio-economic status was achieved by stratifying the analysis using the Carstairs index of material deprivation, based on the 1981 census. The study concluded that the apparent cluster of cases of cancer of the larynx at Charnock Richard, Lancashire, was unlikely to be due to the incinerator.

A small area study of mortality among residents of Malagrotta, a suburb of Rome, Italy, found no association between proximity to industrial sites and mortality from a range of cancers, including laryngeal cancer for the period 1987 to 1993 (Michelozzi *et al.*, 1998). This area contained a number of industrial point sources, including a waste incinerator that closed in 1985 because of a failure to comply with pollution control standards. Despite no evidence linking mortality from laryngeal cancer, or any other cancer, with specific sources in the area, there was a marked decline in mortality from laryngeal cancer with distance from the industrial sites. However, the actual dispersion of pollution from these sites was not evaluated and no direct link with the incinerator can be made.

Using a case-control study, Biggeri *et al.* (1996) reported that lung cancer was significantly related to a waste incinerator in the city of Trieste, Italy, after adjusting for confounding factors such as smoking. The study used distance from the source as a measure of exposure and consequently it is difficult to isolate emissions from the incinerator from other sources of air pollution in the area, which included a shipyard and iron foundry. Both these factors weaken the strength of the outcome and the most reliable conclusion that can be drawn from the study is that the results provide further evidence that air pollution is a moderate contributory factor for lung cancer. Nonetheless the strongest association appeared to be with the incinerator.

## **Childhood cancers**

Several studies by Knox have examined a possible association between childhood cancers and industrial emissions including those from incinerators (Knox and Gilman, 1995; 1998; Knox, 2000). These studies employ spatial analysis of postcodes of those diagnosed with childhood cancer but limitations with the methodologies used mean that the results of these studies are far from certain. No direct measure of exposure is included in the analysis, with exposure estimates being entirely reliant on using distance from the source as a proxy for exposure. The standardisation technique employed in the earlier studies does not attempt to account for the potential effect of deprivation, which would be a major potential confounding factor (Knox and Gilman, 1995; 1998). Both of the early studies have been heavily criticised on the basis of their lack of proper control for population density and the extreme implausibility of some of the findings, which have been interpreted as linking childhood cancer with a wide range of combustion sources including major highways, but only at considerable distance from the road.



The most recent study by Knox (2000) differs in that it is based upon an analysis of the birth and death addresses of children diagnosed with cancer. This showed a greater incidence of cancer in children born close to incinerators and moving away than in those who moved closer to an incinerator. As its basis, the study assumes that migration of children who subsequently develop cancer should be essentially random. The choice of non-combustion industrial markers as a test of this is somewhat curious and includes cathedrals, mail order firms and biscuit makers, industries that are not necessarily located in the same sort of areas as incinerators. It is not clear whether hospital incinerators are included in the analysis; since their results are not presented separately. A careful reading of the full paper reveals that the effect observed by Knox was due to just ten of the incineration plants studied, all but one of which had been open before 1945 and therefore may have produced substantial historical contamination. All were closely associated with other potential sources of environmental hazards. It can, therefore, be taken as reassuring in relation to the relatively lower levels of emissions effects from more modern combustion plants. The work is nonetheless liable to criticism on the grounds that there is no information provided on the net migration of total population inwards or outwards from the vicinity of such plants and therefore, again, no control for temporal changes in population densities. The overall conclusion of the study was that children who died from cancer tended to have moved away from incinerator sites.

### ***Soft-tissue sarcomas and non-Hodgkin's lymphomas***

Viel *et al.* (2000) examined the spatial distribution of soft-tissue sarcomas and non-Hodgkin's lymphomas around a French municipal waste incinerator with 'high' dioxin emissions from 1980 to 1995. The study found localised case clusters of soft-tissue sarcoma and non-Hodgkin's lymphoma in the vicinity of the incinerator, which were more pronounced at the end of the study period. Again, caution is advised before attributing these clusters to emissions from the incinerator, since the study did not take into consideration socio-economic status as a contributing factor and there were other uncertainties, due to low spatial resolution of clusters. Furthermore, these findings are not consistent with the large epidemiological study by Elliot and colleagues in the UK, which did not find any association between both soft-tissue sarcoma and non-Hodgkin's lymphomas with distance from municipal solid waste incinerators (Elliot *et al.*, 1996).

### ***Conclusion***

Despite reports of cancer clusters, no consistent or convincing evidence of a link with incineration has been published. In the UK, the large epidemiological studies by Elliot and colleagues at the Small Area Health Statistics Unit (SASHU) examined an aggregate population of 14 million people living within 7.5 km of 72 municipal solid waste incinerators. This included all incineration plants irrespective of age up to 1987. Despite the consequent inclusion of incinerators with emissions of potential carcinogens orders of magnitude higher than would occur from modern incinerators, both studies were unable to demonstrate an excess of cancers once socio-economic confounding factors had been taken into account (Elliot *et al.*, 1992; 1996; 2000). As a result, the Department of Health's Committee on Carcinogenicity published a statement in March 2000 evaluating the evidence linking cancer with proximity to municipal solid waste incinerators in the UK (Committee on Carcinogenicity, 2000). The Committee specifically examined the results of these studies and concluded that, '*any potential risk of cancer due to residency (for periods in excess of ten years) near to municipal solid waste incinerators was exceedingly low and probably not measurable by the most modern techniques*'. The Committee agreed that an excess of all cancers, stomach, lung and colorectal cancers was due to socio-economic confounding and was not associated with emissions from incinerators. The Committee agreed that, at the

present time, there was no need for any further epidemiological investigations of cancer incidence near municipal solid waste incinerators.

It has been hypothesised that exposure to dioxins and furans (either directly via inhalation or indirectly via the food-chain) is responsible for some cancers in communities around incinerators. However, if this were true, then epidemiological studies on the older generation of incinerators (that emitted significantly greater amounts of dioxins than newer facilities) would have identified an effect. To date, they have been unable to do so. Given that the emissions of dioxins and furans from modern incinerators are orders of magnitude lower than from older incinerators, it can be said, with some confidence, that impacts on cancer rates in local people will not be significant.

There is also no convincing evidence of a causal link between incineration and childhood cancers and most current evidence suggests that infectious biological agents (unconnected with incineration) may be important risk factors in clusters of childhood cancers.

### **Respiratory function**

Several studies have examined possible adverse effects on respiratory health among people living near incinerators. The most credible studies are those which have examined the respiratory health of six communities in North Carolina, USA, three of which are exposed to emissions from biomedical, municipal or hazardous waste incinerators (Shy *et al.*, 1995; Lee and Shy, 1999; Mohan *et al.*, 2000; Hu *et al.*, 2001).

The early study by Shy *et al.* (1995) used objective measures of exposure and respiratory function in a study of three incinerators burning clinical, municipal or hazardous liquid wastes. Indicators of air quality (particles > 10 µm in diameter [PM<sub>10</sub>], particles > 2.5 µm in diameter [PM<sub>2.5</sub>], hydrogen chloride [HCl], nitric acid [HNO<sub>3</sub>] and sulphur dioxide [SO<sub>2</sub>]) were monitored in the areas around these facilities and compared to three matched comparison areas over a 35-day period. The study involved a descriptive investigation of >2,500 households (*c.* 7,000 individuals). With the exception of self-reported sinus trouble, chronic cough and wheezing in the area around the hazardous waste incinerator, there were no significant differences between the study and control areas. Aggregating the data for the control and incinerator areas failed to show any excess of acute or chronic respiratory symptoms in the incinerator areas. However, some statistically significant differences in the prevalence of respiratory symptoms were reported in a sub-set of individuals from each community who had provided lung function data together with behaviour and health diaries, although nasal lavage (wash out) did not reveal any effects from living in the vicinity of an incinerator.

The study reported no significant difference of particulate air pollution or overall respiratory health in communities residing near to three waste incinerators from 1992 to 1994. The study found no significant difference in the concentration of PM<sub>10</sub> in the incinerator communities relative to comparison communities, and later analysis of lung function could not confirm any relationship between PM<sub>10</sub> levels in the communities and lung function (Lee and Shy, 1999). Despite some statistically significant differences in the prevalence of respiratory symptoms being reported, no consistent differences over the different years of the study were found and there were no differences reported between the different types of waste incinerators studied (biomedical, hazardous and municipal).

There have been two further published studies on the respiratory health of these communities (Mohan *et al.*, 2000; Hu *et al.*, 2001). In the first, over 4,200 respondents were interviewed over the telephone about their respiratory health, smoking and other risk factors (Mohan *et al.*, 2000). Respondents were also asked to provide a subjective assessment of the air quality in

their area. The study examined differences in symptom prevalence between each of the study communities and their respective control community, and a combined control group. Results indicated a higher prevalence of self-reported respiratory symptoms in one community near the hazardous waste incinerator, compared with its control community. After controlling for perceived air quality, and when compared with the combined control group, only respiratory symptoms of a long duration remained statistically significant, but this result should be treated with caution because of the lack of actual exposure data and the use of self-reported symptoms. As a result, the study is of limited value in evaluating the effect of incinerator exposures.

The second study examined whether chronic pulmonary effects were related to emissions from the three waste incinerators (Hu *et al.*, 2001). A total of 1,018 subjects underwent a spirometric test once a year between 1992 and 1994. The study attempted to assess exposure by three methods; living in an incinerator community, distance from the incinerator and the use of an incinerator exposure index, which was a function of the distance and direction of the subjects' residence to the incinerator, the number of days the subject spent downwind and the average time spent outdoors. Overall, the test results showed no consistent statistically significant association between pulmonary function and exposure. However, two significant associations were identified, with exposure to the hazardous waste incinerator in 1994 and the municipal waste incinerator in 1993 being linked with poor forced vital capacity. These associations were present when exposure was estimated based on the distance from the incinerator and also from the incinerator exposure index. However, in neither case can a causal link between pulmonary effects and incinerator emissions be proven. No ambient air monitoring was undertaken prior to the annual spirometric tests and the dispersion of emissions was not considered in the exposure estimate. Furthermore, the time-activity patterns which form the basis of the incinerator exposure index were derived from self-administered questionnaires, which may be subject to bias. It is also worth noting that ambient air monitoring after the annual tests demonstrated that emissions from incinerators did not make a significant contribution to particulate air pollution in these communities and that other sources of pollution would be of more importance.

Gray *et al.* (1994) examined asthma severity and morbidity among children living in two areas of Sydney, Australia containing high temperature sewage sludge burning incinerators. A total of 713 children aged between eight and twelve years were studied in the two regions close to the incinerators, together with a further 626 children in a control region which did not contain a sludge burning incinerator. Respiratory illness was measured by questionnaire, airway hyper-responsiveness and atopy. The study found no statistically significant differences in the prevalence of current asthma (as defined by air hyper-responsiveness and recent wheeze), atopy, symptom frequency between the control and two study regions. Furthermore, air monitoring data did not demonstrate any major differences in air quality in the study and control areas. The study concluded that releases from high temperature sewage sludge incinerators appeared to have no adverse effect on the prevalence or severity of childhood asthma.

A small study on a wire reclamation incinerator in Taiwan reported a high incidence of pulmonary effects in children exposed to pollution from the incinerator when compared with a non-exposed control population (Hsue *et al.*, 1991). Whilst, air monitoring confirmed that air pollution was worse in the exposed area, it was unclear whether other industrial sources were present and the study could not confirm whether this pollution was in fact directly related to emissions from the incinerator. The study also did not find any significant difference in the prevalence of cough and wheeze, which tended to contradict the findings of the pulmonary

measurements. As a result, the findings of this study do not appear to support the view that emissions from the wire reclamation incinerator caused pulmonary problems in local children.

To conclude, the majority of available studies are old and typically examine respiratory health around the older generation of incinerators. Most are based upon self-reported symptoms and therefore may be subject to bias. Overall, there is little evidence to suggest that waste incinerators are associated with increased prevalence of respiratory symptoms in the surrounding population. This is consistent with the data from risk assessments, emissions and ambient air monitoring in the vicinity of incinerators, which indicate that modern, well-managed waste incinerators will only make a very small contribution to background levels of air pollution. In many cases, air monitoring data do not demonstrate that emissions from the incinerators are a major contribution to ambient air pollution.

### **Reproductive problems**

It has been suggested that populations living near waste incinerators are at a higher risk of giving birth to twins, possibly due to exposure to dioxins, furans and polychlorinated biphenyls (PCBs) in incinerator emissions, since these are endocrine disrupting chemicals. Lloyd *et al.* (1988) reported that, during 1980–1983, the incidences of twinning in two areas near to a chemical waste incinerator in Scotland were significantly higher at 20 and 16 per 1,000 when compared with rates in control areas of between 3 and 13 per 1,000. The authors hypothesise that the increased incidence of human twinning rates was consistent with anecdotal evidence that polychlorinated hydrocarbons and PCB-related compounds were burnt regularly in the late 1970s. However, the study does not specifically link twinning in the exposed human and animal populations to exposure to polychlorinated hydrocarbons and it acknowledges that it would ‘*be premature to attribute causality to this association between air pollution from incinerators and twinning*’. Furthermore, although maternal age was taken into account, several other possible confounding factors were not. No social or personal risk characteristics were included in the study and no data relating to possible hereditary causes of twinning were examined. The study also suffered from a lack of direct exposure data.

The strength of these findings has been debated in the medical literature with Jones (1989), in particular, presenting a sound argument questioning the basis of this study and correctly citing the lack of evidence of any increased environmental (soil) pollution around the incinerator.

A study of twinning in Sweden failed to find any evidence of spatial clustering of twinning rates in areas near incinerators (Rydhstroem, 1998). In this study, twinning rates before and after the introduction of incinerators was examined in 14 areas between 1973 and 1990. In the majority of the study areas, no evidence of an increased incidence of twinning was reported, once the incinerators became operational. Little weight should be given to the report of a statistically significant increase in incidence in one particular area because it was not consistent with the majority of the study areas and because another area containing a similar incinerator reported a statistically significant decline.

It has been suggested that airborne pollutants associated with incinerators, particularly dioxins and furans, may be associated with fluctuations in the sex ratio of births. Current statistics indicate that, in the UK, the proportion of males has decreased over the last fifty years. Sex ratio is thought to be affected by a wide range of biological and environmental factors including race, birth order, parental age, parental hormone levels, timing of conception, ovulation induction, environmental pollutants and socio-economic status. Undoubtedly human sex ratios at birth are partially controlled by the hormone levels of both parents at the time of conception and therefore, in theory, a mechanism exists by which disruption of the hormonal system may be detected as a change in sex ratios. Such disruption may have been the cause of

the apparent change in sex ratio in populations exposed to high levels of dioxin (TCDD) during and after the industrial accident in Seveso, Italy in 1976 (Mocarelli *et al.*, 1996). In the 74 births reported from nine months to seven years after the accident in a highly exposed population, there was a statistically significant excess of female offspring (26 males vs. 48 females; a male proportion of 0.351, with a probability of this arising by chance of less than 0.1% - i.e.  $p < 0.001$ ). Furthermore, in nine families where both parents were known to have been highly exposed, no males were born during this period (no males vs. 12 females). However, in the years after this period (1985–1994) the sex ratio returned to expected levels (60 males vs. 64 females).

In the UK, Williams *et al.* (1992) reported a significant excess of female births around two incinerator plants in Central Scotland (the same study area as in Lloyd *et al.*, 1988). However, the strength of this outcome is particularly weak as the study lacks a direct measure of exposure to environmental air pollution and inadequately considers several possible confounding factors. There is also the possibility that some births may have been misclassified with regard to antenatal exposure. Almost certainly, other factors were responsible for this excess and no link with environmental pollution can be made.

A recent study has reported a significant association between renal function, cytogenetic measurements, and sexual development in adolescents exposed to environmental pollution, including pollutants from waste incinerators which operated between 1971 and 1980 (Staessen *et al.*, 2001). However, the views of the authors that this study points to possible delayed development are difficult to accept. The potential for adverse effects was determined by the presence of biomarkers of exposure and of effect, whereas the presence of a biomarker does not necessarily imply that an individual will suffer a clinical health effect. Very often they simply identify alterations in tissues and body fluids that do not result in measurable health effects. In fact the study does not relate markers of effect with clinical health effects and, because of this, cannot provide evidence of a relationship between a specific industry and clinical health effects. The fact that individuals living near industries such as incinerators had higher levels of pollutants in their blood or urine is not surprising and the study does not take into account the importance of other routes of exposure (e.g. the food chain) or full control for all potential confounders. Furthermore, the industries included in the report are out-dated and would be expected to be more polluting than their more modern equivalents.

Evidence has begun to emerge that congenital malformations may be associated with environmental pollution. Whilst most studies have focused on hazardous landfill sites, there has been speculation that increased rates of congenital malformations are linked with exposure to dioxins and furans. Such views are consistent with the findings of animal studies which have demonstrated that high exposure to dioxin is strongly associated with congenital malformations, including cleft-lip and palate malformations. In Sweden, a case study examined the incidence of cleft-lip and palate malformations near an incinerator following reports of a cluster (Jansson and Voog, 1989). The authors interviewed the parents of six children born with cleft-lip/palate but found no common explanatory factor other than the possibility of a hereditary link in three of six cases. The authors also conducted a study of cleft lip and palate registrations in the borough both before and after the start of refuse incineration, but could find no increase in the total incidence of these malformations.

Recently ten Tusscher *et al.* (2000) reported on a possible association between incidence of cleft lip and palate in the period 1961-69 and the open incineration of chemical waste in Zeeburg in the Netherlands.

#### 4.4 Critical appraisal of risk assessments

There are only a small number of epidemiological studies on populations around incinerators and the results of these are typically inconsistent and inconclusive. Based on current epidemiological evidence, it is difficult to establish causality, particularly once confounding factors such as socio-economic variables, exposure to other emissions, population variables and spatial/temporal issues are taken into account. In reality, most data on the possible health effects of incinerator emissions are derived from risk assessments, which are routinely used to evaluate both direct and indirect carcinogenic and non-carcinogenic risks. Whilst such assessments can help public health professionals identify chemicals of concern, they struggle to evaluate the level of risk at the concentrations actually emitted into the environment.

In the absence of good epidemiological data, there is a reliance on toxicity data for individual substances released into the environment. The effect of any toxic substance depends on factors such as duration of exposure, concentration of the substance in the environment, biological uptake, and personal susceptibility factors eg. age, sex, and all these factors have to be considered in any estimate of impact of incinerator emissions. In reality, almost all toxicological data are derived from either, studies where exposure levels greatly exceed those typical of incinerator releases, or from animal studies which are not directly applicable to humans.

The quality of any risk estimate is also strongly dependent on the appropriateness of the toxicity data employed in the risk assessment process. Toxicity data employed in the assessment should be up to date, robust and relevant to both the receptor and exposure pathway. In many cases, the assumptions adopted in deriving standards tend to be highly conservative and are likely to over-estimate the actual level of risk.

Risk assessments typically cite a cancer lifetime risk range of  $10^{-4}$  to  $10^{-6}$  as an acceptable level of risk. This is not wholly appropriate in the UK since there is no official UK view on what constitutes an acceptable risk of cancer and other health outcomes. The appropriateness of Quantitative Risk Assessment methods in such circumstances has not been entirely accepted in the UK.

Most risk assessments have attempted to estimate 'reasonable worst case' rather than 'theoretical worst-case maximum' risks from the combustion sources, together with areas where emissions would likely be deposited (as determined by air dispersion modelling). Human health risks are then evaluated using various risk assessment protocols. Although most risk assessors would agree that worst-case maximum risk estimates are not entirely appropriate, it is important that any assumptions relating to potential health effects err on the side of caution, to compensate for the many sources of uncertainty in these risk assessments.

Methodologies for assessing the risks from direct inhalation are well established but understanding of the risks from indirect and less obvious exposure pathways is poor. As a result, procedures for estimating risk from such pathways are more complex and the results of such assessments tend to be less certain and more open to criticism. That said, exposure from indirect pathways such as via the food chain can be very important and any risk assessment must consider all relevant potential source–pathway–receptor relationships. Where pathways and receptors have been omitted, evidence should be provided to support this decision.

Published risk assessments of the health impacts of incineration have been strongly criticised by environmental pressure groups. Although the results of risk assessments are, because of the assumptions made, inherently conservative, it has been suggested that compared with the results of epidemiological studies, many risk assessments have failed to predict or have underestimated real or potentially serious health impacts. As already discussed, there is little

convincing evidence of such impacts and this criticism is often not justified. The other main criticism levelled at risk assessments is that they only consider direct routes of exposure, particularly exposure via inhalation. Whilst many earlier risk assessments were guilty of this approach, more modern risk assessment procedures typically consider indirect routes such as ingestion of soil and vegetation. However, it is worth noting that such procedures are still in their infancy and the ability of risk assessments to predict accurately the degree and consequence of exposure, from indirect routes, is still uncertain.

Although it is important that the chosen risk assessment procedure is appropriate for the site in question, and the potential routes of exposure, it is also important to ensure that the resulting risk estimates are applied appropriately. Despite claims to the contrary, such estimates are not necessarily authoritative and will often be highly tentative. Risk assessments are not simply, as is often implied, a straightforward task involving plugging data into various equations and algorithms. There will be many site-specific conditions associated with a particular study and, in many cases, the chosen risk assessment processes may not fully address these; additional analyses may therefore be required. The use of site specific data should be encouraged, where available, as there is often an over reliance on the use of generalised data, which may not be reflective of actual or expected site specific conditions.

Finally, a robust risk assessment should address the issue of background exposures, since for some contaminants it is necessary to account for existing body burdens and intakes from other sources. This is often forgotten or poorly considered, despite the fact that it can place the risk from incinerator emissions in a more appropriate context.

#### **4.4.1 Application of the COMEAP methodology to calculate deaths brought forward and hospital admissions**

COMEAP have developed a methodology derived from the results of time series epidemiological studies which allowed calculation of the public health impact of exposure to the classical air pollutants, in terms of the numbers of deaths brought forward and the number of hospital admissions for respiratory disease (COMEAP, 1998). Whilst this methodology was applied to the urban population of Great Britain, it is however, possible to apply it to smaller areas and to calculate incremental impacts from developments such as new incinerators. COMEAP has issued a statement expressing some reservations about this development, but nonetheless recognising that it is broadly acceptable to estimate health impacts in this way, provided certain caveats are applied. These generally relate to the fact that the exposure–response coefficients used in the COMEAP report derive from studies of whole urban populations, where the air pollution climate may differ from that around a new industrial installation. The report also refers to the COMEAP report on the chronic (long-term) effects of particle exposure. As yet, however, it is not possible to use the results of that study in estimating the public health impact of an industrial development of this type.

In their report entitled *The Regulatory and Environmental Impact Assessment of the Proposed Waste Incineration Directive* which has become known as WID REIA, the consultants ENTEC applied a COMEAP type methodology to hypothetical incinerators, deriving health impact coefficients in terms of deaths and hospital admissions per tonne of pollutant emitted (ENTEAC, undated). Such results are specific to the precise scenarios in terms of, for example, stack height, plume rise and population density used by ENTEC and are therefore not directly applicable to other emissions scenarios. Despite this fact, they have been widely applied, especially by pressure groups, to the estimation of deaths and respiratory hospital admissions relating to proposed industrial developments.

In the original ENTEC report, by far the largest impacts were attributed to nitrogen dioxide, through its indirect effect on the production of ozone. In a subsequent retraction, ENTEC admitted to a large error in their coefficient and recommended an alternative coefficient for nitrogen dioxide. However, whilst the level of detail of their calculations presented in the REIA report is extremely limited, it does appear likely that the COMEAP methodology has been applied incorrectly and therefore the results are in error. Furthermore, the coefficients in the report relating to deaths per tonne of pollutant, and hospital admissions per tonne of pollutant, appear to be erroneous, since the coefficient for sulphur dioxide exceeds that for particulate matter both for deaths and hospital admissions, whilst in the COMEAP report the coefficients for PM<sub>10</sub> are consistently larger than those for sulphur dioxide.

The calculation of deaths brought forward and respiratory hospital admissions brought forward in the WID REIA report appear to be subject to a number of errors and it is recommended that they be disregarded. A crude calculation using the population reported to live within 2 km of a currently proposed incinerator, and assuming exposure to the maximum annual mean sulphur dioxide concentration, leads to an incremental number of deaths due to sulphur dioxide exposure of 0.57 over 25 years. This calculation is highly approximate as it would be reasonable to include exposure of those living at somewhat greater distances, whilst the use of the annual mean concentration for the point of maximum ground-level concentration will tend to have over-estimated the public health impact. Nonetheless, this gives an approximate order of magnitude for impact effects.

In response to such use and concern that the methodology was being misused, members of COMEAP discussed the applicability of using these time-series coefficients to areas affected by emissions of air pollutants from industrial sources (COMEAP, 2000). They agreed that coefficients reported in time-series epidemiological studies linking concentrations of air pollutants and measures of ill-health, could be used to estimate the effects of air pollutants emitted by industrial processes on the health of people living in areas affected by such emissions, provided that the uncertainties of this approach were acknowledged. However, whilst it was accepted that such an approach might provide useful estimates of effects on health, COMEAP agreed that the extent of these uncertainties could not, at present, be established. COMEAP members identified a number of factors and assumptions that would contribute to the uncertainty of the estimates. These were:

- (i) in applying the above approach it is assumed that the spatial distribution of concentrations of the air pollutants considered is the same in the area under study as in those areas, usually cities or large towns, in which the studies which generated the coefficients were undertaken;
- (ii) it is also assumed that the temporal pattern of pollutant concentrations in the area under study is similar to that in the areas in which the studies generating the coefficients were undertaken.

Both (i) and (ii) above are more likely to be met as the size and nature of the areas studied approach those used in the original coefficient-generating work (the reference areas).

- (iii) it should be recognised that a difference in the pattern of socio-economic conditions between the areas to be studied and the reference areas could lead to inaccuracy in the predicted level of effects;
- (iv) in the same way, a difference in the pattern of personal exposures between the areas to be studied and the reference areas will affect the accuracy of the predictions of effects.

It will be seen from (iii) and (iv) that some comparisons of the study areas and the reference areas should be undertaken before making calculations of effects. For instance, it might well



be unwise to use coefficients derived from studies in towns undertaken in deprived urban areas to predict effects in comparatively affluent rural areas.

It is important to appreciate that there are also a number of assumptions regarding the toxicological properties of the air pollutants considered under the COMEAP methodology. These include:

- (i) linearity of the relationship between ambient concentrations and effects. This assumption is well supported for key pollutants at common ambient concentrations;
- (ii) in the case of particles it is assumed that the toxicity of the ambient aerosol represented by a measure of the mass concentration of a specified fraction of the aerosol eg PM<sub>10</sub>, in the study area is similar to that in the reference area; most epidemiological studies have been conducted in urban areas where transport-generated particles make a significant contribution to the ambient aerosol; application of coefficients from such studies to areas in which transport-generated particles make only a small contribution will include an element of uncertainty;
- (iii) the annual average concentration of pollutants is frequently used as a basis for calculations; its use implicitly assumes that the effects of the pollutants are not characterised by a threshold of effect;
- (iv) if coefficients for several pollutants are applied, and the calculated effects summed, it is assumed that the pollutants act independently and that the coefficients have been derived from studies in which this was tested and found to be supported by the evidence; adding the effects attributed to particles and ozone is likely to be valid; the case for adding the effects attributed to particles and sulphur dioxide is less strong but probably acceptable; it would clearly be wrong to add the effects attributed to PM<sub>10</sub> to those attributed to PM<sub>2.5</sub>; whether effects attributed to particles should be added to those that might be attributed to nitrogen dioxide or carbon monoxide seems dubious and this was not done in the COMEAP quantification report (COMEAP, 1998).

Finally, COMEAP recommended that the following be noted:

- (i) estimates of effects made as described above exclude the possible effects of long-term exposure to air pollutants; evidence to show that such effects may well be important has accumulated recently; the interpretation of such evidence is still under consideration;
- (ii) coefficients are available for only a small group of air pollutants; no quantitative estimate of effect can be made for other pollutants;
- (iii) if estimates of effect are made for very small areas it is likely that only small numbers of, for example, deaths or hospital admissions will be generated; it would be unwise to put too much weight on small differences between already small numbers; for example, 2 extra deaths as compared with 1 extra death.

## 5 Composting

### 5.1 Introduction

#### Composting in the United Kingdom

Composting is a complex aerobic microbiological process by which the organic fraction of municipal solid waste (MSW) and other organic wastes are converted into compost products. There are many different technologies available from the simple aerated static pile ('windrow'), which is the dominant method used in the UK, to more sophisticated contained systems.

It has been estimated that in 1998, approximately 911,000 tonnes of organic waste were composted within the UK at recognised composting sites. Of these, 11 were community sites, 9 were on-farm sites, 9 were on-site facilities and the remainder were centralised sites. Of these, 53 used open-air windrows, 4 were covered windrows and 2 were in-vessel units. Three of the centralised sites had a throughput > 50,000 tons per annum (tpa) and 34 had a throughput of < 5000 tpa. The survey also showed that 69% of the waste composted in 1998, at all types of site, was MSW; the remaining 31% of the total waste composted consisted of 52% from industrial processes, 34% from commercial processes and 14% green waste from landscaping. This MSW comprised approximately 58% household waste from civic amenity sites, 8% from kerbside collections and 34% was green waste from local authority parks.

The UK currently landfills 27 million tonnes a year of municipal waste and 60% of this is biodegradable. Under the 1999 EU Landfill Directive, by the year 2010, the amount of biodegradable municipal solid waste disposed of in landfills in England and Wales must be reduced to 11.9 million tonnes, or 75% of the amount produced in 1995.

#### The composting process

Organic wastes contain lipids, carbohydrates, proteins and lignin. A diverse range of bacteria, actinomycetes and fungi act upon these substrates in the presence of air and water and decompose them. Some of the lipids and carbohydrates are broken down to produce carbon dioxide and water and heat is produced in the process. Some of the proteins are broken down and ammonia released, whilst the lignin and cellulose remain largely unchanged. Over the composting period different microbial taxa flourish at different stages, with some taxa only surviving in large numbers for a short period of the composting process. Many organic wastes contain sufficient numbers of the required types of microorganisms to initiate and sustain the composting process (Palmisano and Berlaz, 1996).

A pre-composting phase involves shredding the wastes and adding water. In the next major phase, temperature rises and thermophilic organisms proliferate. This phase may last between 3 days and a number of weeks, during which the temperature may rise to between 45°C and 75°C. This is when much of the decomposition of the waste occurs. The initial rapid increase of temperature involves a transition from a mesophilic to a thermophilic microflora and there is an increase in the number of species and biomass of thermotolerant bacteria, actinomycetes and fungi. This stage continues until heat production is less than heat dissipation, due to the exhaustion of substrates that can be metabolised.

The next stage (mesophilic) has a lower temperature range of 45°C – 50°C and may last from a few days to a number of weeks. Finally the maturation, or curing stage, takes place at lower

temperatures (between ambient and 45°C). Further chemical reactions occur during this stage (e.g. conversion of ammonium to nitrate). The length of this phase can be varied and depends upon the type of compost required.

### **Micro-organisms present in compost**

At different times during the process a great variety of thermophilic and mesophilic bacteria, actinomycetes and fungi are present in the compost material, as well as organic constituents of microbial and plant origin (Beffa *et al.* 1996a; Beffa *et al.* 1996b; Millner *et al.*, 1994; Millner, 1995; Gilbert and Ward, 1998; van der Werf, 1996; Epstein, 1994; Fischer *et al.*, 1999a). The micro-organisms present are mostly bacterial strains at the beginning of the composting process but these are largely replaced by fungi and actinomycetes by the end (Strauch, 1987).

The following mould genera are found in and on plant material: *Alternaria* spp., *Cladosporium* spp., *Didymella* spp., *Aspergillus* spp., *Penicillium* spp. and *Rhizopus* spp. (Dutkiewicz, 1997). Yeasts such as *Candida* spp., *Rhodotorula* spp. and *Endomycopsis* spp. can also be associated with plant matter. Fungal species most frequently isolated in composting plants are *Aspergillus candidus*, *Aspergillus fumigatus*, *Aspergillus versicolor*, *Emmericella nidulans*, *Paecilomyces variotii*, *Penicillium brevicompactum*, *P. clavigerum*, *P. crustosum*, *P. cyclopium*, *P. expansum*, *P. glabrum*, *P. verruculosum* and *Tritirachium oryzae*.

The mould *Aspergillus fumigatus* is of particular concern since it is a known opportunistic pathogen (Lewis *et al.*, 1994). It decomposes almost all components of organic waste and due to its thermotolerance (Kozakiewicz and Smith, 1994) it finds ideal proliferation conditions in compost at the thermophilic stage (Beffa *et al.*, 1998).

Fungal cell walls contain the polymer  $\beta$ -1, 3-glucan, which is a polysaccharide, composed of glucose units joined by  $\beta$ -1, 3-linkages. In the lung,  $\beta$ -1, 3-glucans depress macrophages (see below) (Rylander, 1993).  $\beta$ -1, 3-glucans from fungi may cause a delayed effect, appearing 3-7 days after exposure and even at low concentrations ( $<1 \text{ ng m}^{-3}$ ) (Rylander *et al.*, 1992).

Mycotoxins are toxic metabolites of fungi that may be present in mycelium, may be excreted into the substrate or found in spores (Sorensen *et al.*, 1987). Mycotoxins may be cytotoxic, immunotoxic or mutagenic (Ciegler *et al.*, 1981) and some mycotoxins have been reported as toxic to alveolar macrophages (Sorensen *et al.*, 1985).

Among microorganisms occurring in organic dusts, species of Gram-negative bacteria *Pantoea agglomerans*, *Pseudomonas* spp., *Klebsiella* spp. and *Alcaligenes faecalis* are commonly found. Gram-negative bacteria produce endotoxins which, when inhaled, cause inflammation in the respiratory tract and toxic pneumonitis by activation of alveolar macrophages with the release of cytokines (Lacey and Dutkiewicz, 1994; Burrell, 1995). Bacterial endotoxin is the chemically complex portion of the outer layer of cell walls of Gram-negative bacteria. In the alveoli living or dead bacterial cells can be engulfed by macrophage cells which process them and release endotoxins (Duncan *et al.*, 1986). Endotoxins can easily be released in large quantities in the form of discoid particles 30-50 nm in diameter (Dutkiewicz *et al.*, 1992).

The filamentous Gram-positive actinomycetes only occur in dusts from plant materials and have spores about 1  $\mu\text{m}$  in diameter. The most common thermophilic species observed are *Saccharopolyspora rectivirgula*, *Thermoactinomyces vularis* and *Thermonospora* spp. (Lacey and Crook, 1988). Mesophilic actinomycetes developing in soil and vegetable materials are commonly of the genera *Streptomyces* spp., *Rhodococcus* spp. and *Agromyces*

spp. (Lacey and Dutkiewicz, 1994). *Bacillus subtilis* and other aerobic spores may also be found in relatively small concentrations (Dutkiewicz, 1997).

Faecal contamination of raw material is highest when it incorporates large quantities of urban waste water sludge, or farm wastes, and is lower for household wastes. However, household wastes may contain human and domestic animal faeces and surveys of soiled disposable nappies in municipal solid wastes have shown that they contribute approximately 1% by weight, and about one third are soiled with faeces. Such faecal material may be contaminated with potentially pathogenic bacteria (e.g. *Salmonellae*), protozoa (e.g. *Cryptosporidium parvum*, *Giardia lamblia*) or worms (e.g. *Toxocara* spp.). A number of techniques may be used to detect pathogens in compost (Farrell 1993, Pfaller *et al.* 1994, Blanc *et al.* 1997). A number of enteric viruses may also be present in human faeces including *Hepatitis A*, *Poliovirus* and *Coxsackievirus* (Pahren and Clark, 1987).

There is a theoretical possibility that raw food wastes could contain viruses or prions. Viruses may not be inactivated and prions would not be inactivated during the composting process. There is also a risk of contaminating clean areas with contaminated waste from other parts of the compost during turning operations (Pereira *et al.*, 1987).

The ability to control temperatures over a long period of time and throughout the entire mass of material is very important to the efficiency of the composting process. Temperature control is also important in ensuring that human and animal pathogens are killed (Epstein, 2001). Theoretically, a temperature of 55°C, held for three days, is effective (EPA 1994, Epstein and Epstein, 1989) but thermophilic species and certain endospores (e.g. *Bacillus subtilis*) may survive. Therefore adequate temperature control throughout is necessary if kill of pathogens is to be guaranteed. It is generally assumed that to obtain efficient decomposition of the compost, temperatures should not exceed 55° - 60°C.

### **Organic compounds and metals associated with composting**

A wide range of volatile organic compounds (alkanes, alcohols, ketones, aldehydes, esters, ethers, terpenes and terpene derivatives) are produced by fungi during composting (Fischer *et al.*, 1999b). The predominant volatile compounds produced by a range of fungi are 1-octan-3-ol and 2-octan-1-ol, which are associated with the 'earthy' odour frequently reported from composting operations (Lacey and Dutkiewicz, 1994).

Other (non-fungal) interactions in composting may produce offensive odours arising from compounds such as hydrogen sulphide, dimethyl sulphide, ammonia and propanoic acid (Smalley, 1998).

The humus part of composts contains ligands that may have metallic elements bound to them (De Wit *et al.*, 1993). When this compost is applied to soils, these may be released and then become available for uptake by plants. Toxic metals found in compost include zinc, nickel, arsenic, hexavalent chromium, mercury, lead, copper and cadmium. Zinc, copper, lead, mercury, cadmium and chromium are derived from batteries, glassware, plastics and ferrous materials. Zinc and nickel can remain when paper and cardboard are broken down (Lineres, 1993).

Metal contaminants concentrate during composting following the reduction in volume of composted material (Canarutto *et al.*, 1991), whereas insecticides and herbicides are degraded (Lemmon and Pylpiw, 1992). A review of the concentrations of metals in MSW found concentration ranges of 7-9 ppm for arsenic, 0.3-12 ppm for cadmium, 8-403 ppm for chromium, 1-23 ppm for mercury, 1-1,220 ppm for nickel, 11-1,312 ppm for lead and 75-

2,427 ppm for zinc (Deportes *et al.*, 1995). For poorly degradable organic compounds the concentration ranges reported were 0.1 to 7 ppm for PCDDs/ PCDFs, 0.5-5 ppm for PCBs, 1-250 ppm for PAHs and up to 9.1 ppm for volatile solvents.

Composting promotes chemical decontamination but the degree of degradation by type of chemical is uncertain. A degradation of 6% of chlorinated pesticides and 45% of PCBs has been reported (Vogtmann and Fricke, 1992). Also, metals become less bioavailable with increasing compost maturity (Ciavatta *et al.*, 1993) and this limits their subsequent uptake by plants.

## 5.2 Potential exposure routes

Bioaerosols are formed by particles of biological origin in air, during the composting process. These include viruses, bacteria, actinomycetes, fungal spores, fragments of insects, mites and plant cells, proteins from plants and animals, endotoxins from Gram-negative bacteria and mycotoxins and glucans from fungi. In general, the most significant exposure pathway for bioaerosols is via air and inhalation.

Different containment systems are required to prevent leachate from contaminating surface water and ground water, and to reduce atmospheric dispersion. (Rynk, 2000, Hochstin, 1998 and Edwards, 1998). If composting is carried out within a container, rather than with aerated static pile or windrow, then greater control may be achieved over the composting microorganisms so that they function under optimum conditions for composting. Consequently elimination of pathogens is more likely and also bioaerosol and odour dispersion is prevented.

Published studies (Danneberg *et al.*, 1997; Reionthaler *et al.*, 1999) suggest emissions ie concentrations in air, of the order of  $1 \times 10^5$  cfu m<sup>-3</sup> (colony forming units per cubic metre) total bacteria, and total fungi in the range  $3 \times 10^3$  to  $10 \times 10^6$  cfu m<sup>-3</sup> with *Aspergillus fumigatus* in the range  $2 \times 10^3$  to  $7 \times 10^4$  cfu m<sup>-3</sup>. Data from monitoring at three UK sites (Wheeler *et al.*, 1999) report emissions of bacteria in the range  $3 \times 10^3$  to  $3 \times 10^6$  cfu m<sup>-3</sup> and fungi and yeast in the range  $0.5 \times 10^3$  to  $7 \times 10^4$  cfu m<sup>-3</sup> for green and source separated waste. Bacteria emissions as high as  $2.5 \times 10^7$  cfu m<sup>-3</sup> were reported at a site composting refuse for fuel production. Research supported by the Swiss National Foundation (biosafety research) and several compost industries has investigated different aspects of composting. This has demonstrated that up to  $10^6$  *Aspergillus fumigatus* cfu m<sup>-3</sup> could be measured at sites and in the vicinity of turning machines, and concentrations in air were measured at up to  $10^7$  cfu m<sup>-3</sup>. This research also demonstrated that the management of the composting process markedly influences the proliferation of *Aspergillus fumigatus*. More frequent turning has been associated with lower levels (Beffa *et al.*, 1998).

The Health and Safety Laboratory and the Composting Association have reviewed occupational and environmental exposure to bioaerosols (Swan *et al.*, 2003). They report that workers at compost sites are regularly exposed to bioaerosols between 10 and 1,000 times greater in concentration than may be expected normally in ambient air.

Workers collecting and recycling waste are exposed mostly to moulds (e.g. *Aspergillus* spp. and *Penicillium* spp.) with collection of green waste resulting in much higher exposures (Breum *et al.*, 1996a, Nielsen *et al.*, 1997). The concentrations of actinomycetes associated with composting usually are greater than those of fungi (Lacey and Crook, 1988).

### 5.2.1 Background levels of fungal spores in outside air

Bioaerosol concentrations existing in the ambient environment ie at background levels, will vary with geographical location and with season. The species components within the total concentration will also vary according to geography and season.

Any consideration of the type and concentration of fungal spores in outside air requires an understanding of the methods used to collect data, and their shortcomings. Most published data derive from surveys based on the use of either culture plate techniques or spore sampling techniques.

One particular culture plate techniques rely on airborne spores landing on the agar surface of a culture plate and germinating to produce a mould growth which can then be identified to species level. The shortcomings of this technique are that simply allowing the spores to settle on the surface of the agar by gravity will favour the larger spored moulds, which have a higher deposition velocity. A volumetric apparatus is therefore preferred, such as an Andersen sampler (Andersen, 1958), in which the air is drawn onto the culture plate causing the spores to be impacted onto the agar surface. To achieve a high level of sampling efficiency, air is drawn through the sampler at a rate of 28.3 litres min<sup>-1</sup> so that sampling has to be restricted to relatively short periods of time (often no more than 10 minutes), to avoid the culture plate becoming overloaded. This means that by taking just one sample in a day, it is possible to miss changes in spore concentration associated with diurnal periodicity, or changes in meteorological conditions. The choice of agar used in any study can also have a major influence, as moulds do not all have the same growing requirements and different culture media favour the growth of different moulds. This can be used to advantage when studying a particular mould (Mullins, 1994), but can lead to difficulties when trying to take a census of all fungal spores in the air. Growth on culture plates is also restricted to imperfect or asexual stages of fungi, which results in the absence of most *Ascomycetes*, *Basidiomycetes* and, of course, parasitic fungi which will only grow on host tissue.

The alternative method of conducting surveys of airborne fungi is to collect the spores onto glass slides for microscopic identification. As with the former method, a suitable apparatus, such as a Hirst spore trap or a Burkard trap (Hirst, 1952) must be used to impact the spores on to a layer of adhesive on the surface of the slide. Such an apparatus has the advantage that it samples continuously over a 24-hour or a seven-day period, so that the results are not biased by diurnal or meteorological changes, but identification of the spores under a light microscope, where most spores are below 10 µm in diameter, limits the specificity of the technique. It does however allow for identification of *Ascospores*, *Basidiospores* and spores of parasitic fungi such as rusts, smuts and mildews. Burge *et al.* (1977) compared spore concentrations obtained by spore trapping with those obtained using a culture plate technique and found that as *Cladosporium* spore concentrations (assessed by spore trapping techniques) rose, culture plate methods progressively underestimated prevailing spore concentrations, giving low estimates (20–40%) at levels below 100 spores m<sup>-3</sup> and falling to 5% at levels above 500 spores m<sup>-3</sup>.

The Composting Association has published a protocol for sampling of airborne microorganisms which targets mesophilic bacteria and the fungus *Aspergillus fumigatus* as appropriate indicators of the composting process (Composting Association 1999).

Hyde (1969) characterised the air spora as being “*an expression of climate, a reflex of the vegetation as a whole and an essential factor or complex of factors in the general environment*”. Thus, the spore content of the air is influenced by the general climate, the vegetation, diurnal periodicity and changing meteorological conditions.

In temperate climates, the air spora is dominated by *Cladosporium* (Richards, 1953) and culture plate studies in Copenhagen suggested an incidence of 68.9% (Larsen, 1981) and 77.8% (Larsen and Gravesen, 1991). In contrast, spore studies in Cardiff in 1995 indicated a *Cladosporium* incidence of 55%, but if non-culturable species are excluded, the incidence is 75% (unpublished data).

Studies of spores in the air in the UK were pioneered by Hirst (1953) and Gregory and Hirst (1957) at Rothhamsted, where surveys were run for short periods of time and the particular interest was the dispersal of plant pathogens. Volumetric surveys of fungal spores in the air of the UK started in Cardiff in 1954 and continued until 1997, and were undertaken in London (Paddington) from 1960 until about 1985 (although all these data have now been lost) and in Derby from 1965 to the present day. The longest running survey of air spora in the world was carried out in Cardiff by the Asthma and Allergy Research Unit with the installation of a Hirst spore trap on the roof of the National Museum of Wales in 1954. This was moved to the nearby roof of the University in Cardiff in 1963 and a daily survey was continued until 1997. From this survey, a sample year of 1995 gave the incidences of spores illustrated in Table 5.1.

**Table 5.1 Incidences of spores in Cardiff in 1995**

Spore type	Daily average concentration m <sup>-3</sup>	Percentage incidence
<i>Cladosporium</i>	1,191	55.0
Ascospores	368	17.0
Basidiospores	222	10.3
<i>Sporobolomyces</i>	90	4.2
<i>Pullularia</i>	71	3.3
<i>Mycelium</i>	67	3.1
<i>Aspergillus/Penicillium</i>	42	1.9
<i>Alternaria</i>	36	1.7

Surveys have also been undertaken elsewhere in Wales to compare spore incidence with Cardiff (Mullins, 2001).

The surveys were carried out at:

- Aberystwyth, on the roof on a University building facing the sea front
- Cefn Mably, just outside Cardiff in the grounds of a hospital, surrounded by mixed deciduous woodland
- Resolven, in the Swansea Valley alongside a plantation of conifers
- Tintern, in the Wye Valley, surrounded by mixed woodland
- Cleppa Park, an agricultural research station between Cardiff and Newport, alongside a field of barley
- Llwynypia, on the roof of a hospital on the east side of the Rhondda Valley.

The most obvious trend from these surveys is the higher levels of Ascospores and Basidiospores associated with woodlands, the higher general levels of spores in rural

environments and the reduction in spore levels in the Aberystwyth west coast sea front location and in the upland valley of the Rhondda.

### **5.2.2 Monitoring bioaerosols, inhalable dust, volatile organic compounds (VOCs), noise and odours in the vicinity of composting facilities.**

The Environment Agency reported (Environment Agency, 2001a) a monitoring programme of key environmental emissions at a full-scale composting operation and two small-scale composting trial sites. The Environment Agency also reported emissions from two turned-windrow, green waste composting facilities and one in-vessel composting facility treating a mixture of mixed municipal solid waste and source separated organic waste (Environment Agency, 2001b). The in-vessel facility was associated with a landfill and civic amenity site, one of the green waste facilities had a neighbouring sewage treatment works and the other had neighbouring farmland growing arable crops. There was therefore a potential for monitored values of bioaerosol, VOCs and inhalable dusts to be affected by sources other than the composting facilities.

On many occasions the concentrations of bioaerosol measured both upwind and downwind of the sites exceeded 1,000 cfu m<sup>-3</sup> total bacteria, 300 cfu m<sup>-3</sup> Gram-negative bacteria and 1,000 cfu m<sup>-3</sup> fungi. A comparison of Andersen sampler and filter results suggested that emissions contained clumps of organisms with a significant proportion of biological particles exceeding 20 µm and therefore liable to be deposited relatively close to source. Since simple Gaussian plume modelling is unlikely to be helpful and, as more complex models have yet to be applied to dispersions of bioaerosol, a simple straight line fit to the logarithm of the data was used as the means of estimating the distance to reference concentrations. This practice is questionable as evidenced by the observation that, for a number of occasions, bioaerosol concentrations increased with distance from site.

Only inhalable dust (particles up to 100 µm) was monitored whereas the greatest potential harm to respiratory health is posed by respirable particles less than 10 µm (PM<sub>10</sub>). VOC concentrations were found to be low in and around the plants but results indicated that odour may be a potential problem at some sites particularly with mixed wastes and source separated organic fractions. The distance from source, required for odour to fall to a reference level for the composting facilities reported in the above reports, varied from 80 m to 940 m. On one occasion, odour was seen to increase with distance from source either due to complex dispersion or to other sources in the area.

Different contained composting technologies have different efficiencies with regard to control of odours. Normally contaminated air is passed through a wet scrubber to eliminate ammonia and then through a biofilter to remove other odorous substances. There is a potential for noise levels to produce nuisance but the design of the sites monitored indicated that noise did not pose a major nuisance.

### **5.3 Potential health effects associated with the process**

Bioaerosols produced by composting have the potential to produce adverse health effects such as aspergillosis, hypersensitivity pneumonitis and exacerbation of asthma (Déportes *et al.*, 1995; Epstein, 2001; van Yperen and Rutten, 1997; Browne *et al.*, 2001; Douwes *et al.*, 1997; Bünger *et al.*, 2000; Douwes *et al.*, 2000). There is also the potential for disease if pathogens survive the composting process and are present in bioaerosols.



The ability of microorganisms to cause disease depends on the number of viable organisms, their virulence and the susceptibility of the exposed person. The numbers of ingested microorganisms required for infection are in the range  $10^2$ – $10^9$  for *Salmonella* spp.,  $10^4$ – $10^{10}$  for pathogenic *Escherichia coli*,  $10^9$ – $10^{10}$  for pathogenic *Streptococcus* spp., and  $0.9$ – $9 \times 10^6$  ‘focus forming units’ for rotavirus (Kowal, 1985). Protozoa may also be present in waste, for example *Cryptosporidium* spp., which has an infective dose in the range 10–100 cysts (Casemore, 1991). Pathogens which infect healthy people are often termed ‘primary pathogens’ whereas those which largely affect only individuals whose immune system is compromised are termed ‘opportunistic pathogens’.

The risk to health, for an individual exposed to bioaerosol from composting operations, depends upon the concentrations in air of different components of the bioaerosol as well as personal exposure and prior health status. However, there is evidence from occupational health and individual case reports which demonstrate the potential for health risks in uncontrolled settings.

*Aspergillus fumigatus* is an opportunistic pathogen in that it colonises and infects individuals who are immunocompromised (i.e. persons receiving immunosuppressive drugs, high doses of corticosteroids, or who have haematologic malignancies or human immunodeficiency virus). *A. fumigatus* may, in such people, colonise the lung airways with aspergillomas (fungus balls). Invasive growth may also occur with entry pathways from the nasal mucosa to the brain or from the lungs into the blood. Serious health effects have been reported: these include a fatal, locally invasive pulmonary aspergillosis in a garden worker (Zuk *et al.*, 1989), acute hypersensitivity pneumonitis from compost handling (Weber *et al.*, 1993) and allergic bronchopulmonary aspergillosis in an asthmatic residing in proximity to a municipal leaf composting site (Kramer *et al.*, 1989).

Cellular reactions are part of the normal response to inhaled biological agents. Systemic effects occur through release of bioactive substances from the cells of the lungs into the blood. In the lungs, after inhalation exposure, the initial stage in the inflammatory response is the activation of macrophages. These cells secrete a series of substances, which cause the migration of neutrophils from the blood into lung tissue (Henson and Murphy, 1989). This, in conjunction with fluid leaking from the capillaries, causes a toxic pneumonitis. This reaction, which occurs within hours after exposure, may give rise to influenza-like symptoms.

Hypersensitivity pneumonitis (HP), also known as extrinsic allergic alveolitis constitutes a spectrum of granulomatous, interstitial, bronchiolar and alveolar-filling lung diseases from repeated inhalation of, and sensitisation to, a wide variety of organic aerosols. Disease typically is characterised by a lymphocytic alveolitis and granulomatous pneumonitis, with improvement or complete reversibility if antigen exposure ceases. Continued antigen exposure commonly leads to progressive interstitial fibrosis (Rose, 2000).

Bacteria and fungi are commonly recognised causes of HP and some spores are deposited in airways and then made soluble, but most particulate antigens are of respirable size and deposit in the alveoli. These trigger a T-lymphocyte alveolitis and have the ability to fix complement, thereby activating inflammatory cells and amplifying immune responses (Salvaggio and Millhollon, 1992). Thermophilic actinomycetes are causally associated with ‘farmer’s lung’ disease and ‘mushroom worker’s lung’. Endotoxins contained in the cell walls of Gram-negative bacteria stimulate the cytokines, tumour necrosis factor-alpha and interleukin-1, amplifying the inflammatory responses leading to alveolitis (Brade *et al.*, 1993).

Human exposure to large quantities of airborne endotoxin, produce symptoms which include fever, diarrhoea, headaches, nausea, nasal irritation, chest tightness, cough and expectoration of phlegm (Olenchock *et al.*, 1982). *Aspergillus* species have been associated with HP in

compost and greenhouse workers (Meeker *et al.*, 1991; Yoshida *et al.*, 1993). Fungal contaminants have also caused HP in wood handlers (Amanuel *et al.*, 1996).

The latency period between exposure to an environmental antigen and onset of HP symptoms may vary from a few weeks to years (Rose, 2000). Hypersensitivity pneumonitis requires a chronic exposure to moulds or thermophilic actinomycetes which produce strong allergens (van den Bogart *et al.*, 1993). Chronic, low level exposures to airborne fungi, such as that which occurs in association with mouldy hay, may infrequently lead to lung fibrosis (Richerson, 1994).  $\beta$ -1, 3-glucans, which are a basic constituent of fungal cell walls, may cause chronic lung inflammation.

Mycotoxicoses are diseases caused by mycotoxins, which are endogenous compounds produced by fungi (Lacey *et al.*, 1994). Some mycotoxins have been shown to be carcinogenic when ingested (e.g. aflatoxin) but their effects following inhalation by man are unknown. However, experiments in rats indicate that inhaled toxin is more toxic than systemic administration (Creasia *et al.*, 1990). It has also been suggested that the immunotoxic effects of mycotoxins could render exposed individuals susceptible to adverse health effects from other components of the bioaerosol (Sharma, 1991). Mycotoxins, such as aflatoxins produced by *Aspergillus flavus*, and ochratoxins produced by *Penicillium* spp., suppress the activity of alveolar macrophages but their concentrations in air are low.

*Clostridium botulinum* is a soil-borne anaerobic bacterium. As feedstock and rotting material during the composting process are completely heterogeneous, there may be 'anaerobic compartments' present in compost. *C. botulinum* may produce toxins which are lethal to humans. The health effects may be caused by ingesting toxin or by ingesting spores, which colonise the intestine and produce toxin (CDC, 1998). Sampling of marketed bio-compost showed 50% of samples contained *C. botulinum* (Bohnel and Lube, 2000).

Asthma is a disease of the airways, characterised by airway narrowing with spontaneous reversibility, increased responsiveness of the airways to various stimuli and the presence of inflammation in the airways. Inhalation of specific allergens is a well recognised cause of exacerbations of asthma. Allergen inhalation not only causes immediate airflow limitation but can also cause an increase in non-specific airway hyper-responsiveness and may increase the overall clinical severity of asthma (Cockcroft *et al.*, 1977). The recognised causes of occupational asthma include proteins of vegetable and microbial origin (e.g. alcalase from *B. subtilis*) and the dusts of many different woods (Newman Taylor, 1987). Asthma may be caused by allergens of microbial or plant origin but the amounts of airborne allergens that sensitise and incite asthmatic or allergic episodes cannot be defined given the wide variation in host sensitivity.

A review of occupational exposure to bioaerosols and potential health effects has concluded that there is little published evidence of serious/chronic disease in compost workers, although there is evidence of early health responses to bioaerosol exposure.

#### **5.4 Critical appraisal of risk assessments**

A review of a number of site-specific risk assessments of composting facilities has been undertaken providing some consideration of health effects, source emissions, pathway modelling, bioaerosol background levels and sampling issues related to composting processes.

##### **Health effects**

In general, these identify the various components of bioaerosols with the potential to damage human health. However, although it is recognised that concentration thresholds giving rise to

clinical effects are unknown, some aspects of the relationships between exposure and potential health effects are overlooked. For chronic effects it is, in principle, correct to adopt a probabilistic assessment of cumulative dose (i.e. to address receiver occupancy and frequency of release events etc). However, for acute effects, if concentrations exceed a threshold on any particular occasion, then the effect occurs. Acute exposures to aero-allergens may cause immediate airflow limitation but may also cause an increase in non-specific airway hyperresponsiveness and may, thus, increase the overall clinical severity of asthma. Therefore, it is more conservative to proceed by estimating the concentrations at receptors under the most unfavourable conditions and these should not be sufficient to give rise to acute effects. It is now recognised that chronic exposure could lead to increased susceptibility to respiratory infections since fungal cell walls contain beta glucan which is known to reduce the number of alveolar macrophages and impair phagocytosis although, again, for this effect, concentration thresholds are unknown.

### **Source emissions**

Assumed emissions tend to be based upon levels reported in the research literature but use typical levels rather than worst case measurements. Published studies (Folmsbee and Strevett, 1999; Epstein *et al.*, 2001; Khalil *et al.*, 2001; Heldal *et al.*, 1997) suggest emissions of the order of  $10^5$  cfu  $m^{-3}$  for bacteria and total fungi in the range  $10^3$  to  $10^7$  cfu  $m^{-3}$ ; with up to  $10^6$  cfu  $m^{-3}$  of *Aspergillus fumigatus* being measured at sites.

### **Pathway modelling**

In principle, a sound source–pathway–receptor methodology is applied to bioaerosols from composting. However, dispersion modelling for bioaerosols (Déportes *et al.*, 1997) is not yet developed to a satisfactory accuracy. Some risk assessments employ a correction for the proportion of specific microorganisms which remain viable after a certain time (e.g. 100 seconds) at ambient environmental conditions. It is more conservative to assume that all microorganisms remain viable. Also, the potential for contaminants to be deposited on local crops is often overlooked, as is the impact upon receptors of PM<sub>10</sub> particles generated.

### **Background levels**

In the absence of quantitative dose–response data for microorganisms associated with the composting process, a precautionary approach is adopted, in which exposures at receptors are not increased above background levels. Background levels ie the total bioaerosol concentration and the species components, will vary with geographical location and with season. The logic underpinning this ‘target’ level requires clarification, because in a locality without a composting facility, the potential for acute health effects is greatest when the seasonal background concentrations of bioaerosol are greatest. A decision on permitting the siting of a composting facility in the locality might then, in part, be determined by what proportionate increase of this maximum seasonal background is deemed to be acceptable. The distance from the source, at which concentrations of specific components are reduced to background levels, is determined, in part, by the quantity of emissions.

### **Sampling**

The local background concentrations of bioaerosol may need to be measured and emissions may require monitoring. The principal methods of measuring airborne spores have depended upon entrapment on agar media and then incubating them so that counts can be made of developing colonies. However, the proportions of propagules that are non-culturable vary with species and the total that can be cultured can be as little as a few per cent. Non-culturable

spores may be as effective as culturable spores in triggering allergenic and non-immunological mechanisms. It may, therefore, be prudent for analysis to take into account some measure of total fungal burden, such as ergosterol concentrations. Also, a bacterial marker may be required to determine the concentration of non-culturable bacteria in bioaerosols.

## 6 Waste collection, transfer and recycling

### 6.1 The collection of waste from individual properties

No HIA has been carried out on waste collection itself, other than occupational health studies on waste collectors in North European countries (van Ooijen *et al.*, 1997; Wouters, 1999; Heldal *et al.*, 1997; Midtgård *et al.*, 1999). These studies examine the effects of different designs of equipment, different receptacles, weather conditions and working methods. One Dutch study (Wouters *et al.*, 2000) examined the issue of storing domestic waste indoors for longer periods. This is a recycling issue, because the kerbside collection of segregated wastes usually results in alternate fortnightly (or longer) collection intervals. Only countries spending large sums of money on waste collection have segregated collections at weekly (or more frequent) intervals (this is often achieved by the use of multi-compartment wheeled bins). Recycling is much improved by segregation at source, which usually results in longer storage periods.

The Dutch study (Wouters *et al.*, 2000) demonstrated that homes storing separated organic waste for more than one week resulted in levels of endotoxin, extracellular polymeric substances (EPS) and glucan which were 3.2, 7.6, and 4.6 times higher than the levels in homes in which only non organic residual waste was stored indoors. Increased levels of endotoxin and EPS were elevated by 2.6 and 2.1 fold over homes not storing organic waste indoors, when separated organic waste was stored indoors for less than one week. Storage of non-separated waste indoors for one week or less had no effect on microbial concentrations. The researchers concluded that these increased levels might increase the risk of bioaerosol-related respiratory symptoms in susceptible people.

Workers were monitored in Denmark (Breum *et al.*, 1996a) where personal samples of airborne contaminants were obtained to identify any difference in bioaerosol exposure between the different segregated waste collections. They found that workers collecting garden waste were more heavily exposed to bioaerosols than workers collecting other wastes. The predominant exposures from garden waste were fungi and actinomycetes. Spring season resulted in the heaviest exposures, and collection in bins or sacks made little difference. Their comparison with data gathered by other researchers at other stages within the waste management process suggests that waste collectors are generally exposed to fewer bioaerosols than workers inside waste transfer stations or incinerators. However, landfills and composting plants resulted in higher worker exposures. This suggests that HIAs should pay more attention to transfer facilities than collection systems. However, care is needed when comparing microbiological data which has been sampled and detected using different techniques. A French study (Ducel, *et al.*, 1976) using area sampling around waste collectors, suggests an order of magnitude reduction in micro-organisms at a distance of only 2–3 metres from refuse collection vehicles.

Another Danish study (Breum *et al.*, 1994) compared aerosol exposure amongst different waste collection crews and concluded that sack collection resulted in lower exposures compared with bin collection, where one operator continually loaded the vehicle. A more recent Danish study (Ivens *et al.*, 1999) found an exposure–response relationship between nausea and endotoxin exposure and between diarrhoea and exposure to both endotoxins and viable fungi. This research also produced a detailed job exposure matrix for bioaerosol exposure. Another similar study (Ivens *et al.*, 1997) found no positive trend between high exposures to bioaerosols and Gastrointestinal (GI) symptoms, but found an association between exposure to fungal spores and self-reported diarrhoea. The researchers comment that

this could be caused by VOCs being released from waste with a high microbial activity (Wilkins, 1994). Some studies in transfer stations show elevated VOCs and these substances have been reported as a cause of GI problems. Waste compaction trucks have been designed to reduce the release of bioaerosols between two and six-fold by mounting an air exhaust system behind a plastic lamella curtain (Breum *et al.*, 1996b).

A review of a number of studies examining bioaerosol exposure in waste collection (Nielsen *et al.*, 1997) agreed with the findings on garden waste and concluded that exposures were lower in the winter and were reduced by top-loaded vehicles, where predominant emissions were 3 m above ground.

The Danish Institute of Occupational Health, and others, carried out a wide-ranging review of all occupational health problems relating to waste collection (Poulsen *et al.*, 1995a). Most of the issues discussed do not relate to HIA, but they observed that little is known about actual incidence rates of these problems and that knowledge is sparse on causality. More data are required to define a dose–response relationship to underpin occupational exposure limits (OELs) which are often the starting point when defining what may be an acceptable exposure to the general public. Interactions between exposures could also be important (Poulsen *et al.*, 1995a). This is particularly relevant as much of this work is in an environment heavily affected by diesel exhaust emissions which may potentiate the effect of allergens in susceptible individuals (Scheepers and Bos, 1992). Similarly, endotoxins and VOCs, along with other substances released by the handling of waste, are likely to have a synergistic interaction (Norn *et al.*, 1986).

It would seem that studies of the effects of exposure to bioaerosols in the waste collection environment should be examining low dose effects. Changes in Peak Expiratory Flow (PEF) variability and elevated concentrations of immunoglobulins can be used as indicators of sub-clinical effects of relatively low exposure levels to organic dust (Coenen *et al.*, 1997).

## **6.2 Transfer at purpose built sites for handling or sorting**

Again, most of the published research on transfer or sorting sites is focused on occupational health. Some proposed sites have been subjected to Environmental Impact Assessment (EIA), but these have not been subject to operational monitoring in a form suitable for publishing, and the EIA approach has concentrated on nuisance from dust, noise, vehicle movements etc.

One Canadian study evaluating health and safety risks (Lavoie and Guertin, 2001) includes some interesting data on microorganisms upwind and downwind of these types of plants. A review of data on total bacteria, gram-negative bacteria, and moulds inside and outside the plants concludes that microbial air quality outdoors, at 100 m downwind, was not affected by the operations carried out in the recycling plants. An older US study (Lembke and Kniseley, 1980), also measured total and faecal coliforms up and downwind over similar distances and found no deviations from ambient concentrations.

Workers at these plants have been studied in various countries. Aarhus University Hospital (Sigsgaard *et al.*, 1994a) examined lung function changes among 99 workers, concluding that there was a significant association between exposure to organic dust and a fall in Forced Expiratory Volume (FEV). They found no relation between endotoxins and FEV, but, endotoxin concentrations at the workplace were all below 100 ng m<sup>-3</sup>. Earlier studies in Denmark indicated occupational asthma as a problem (Sigsgaard *et al.*, 1990; Sigsgaard, 1998). This has reduced with improvements to operational design. Reported symptoms are still elevated for chest tightness, toxic alveolitis, GI and skin irritation.

Another Danish study (Sigsgaard *et al.*, 1994b) found a similar pattern with workers at this type of plant, but also included a higher prevalence of flu-like symptoms, itching eyes, itching nose, sore throat and a high prevalence of nausea vomiting and diarrhoea. Organic Dust Toxin Syndrome (ODTS) was found to be associated with refuse handling. Multivariate analysis showed ODTS to be associated with a familial disposition to atopy. A 1997 study of similar workers in Denmark involved in domestic waste handling, composting and paper sorting (Sigsgaard *et al.*, 1997), reported similar findings but included higher cadmium blood concentrations among the waste handlers. The conclusion was that this might stem from exposure to electrical batteries in the waste.

A variety of published studies monitored the internal air quality at transfer stations and all commented on elevated microbial concentrations. A Canadian study (Lavoie and Alie, 1997) commented on elevated concentrations of ammonia, carbon dioxide and hydrogen sulphide, although they note that outdoor air quality 100 m downwind was unaffected (however the sensitivity of the monitoring equipment used was probably not sufficient to detect outdoor air concentrations of the pollutants). A Finnish study (Kiviranta *et al.*, 1999) also highlighted higher concentrations of microorganisms and VOCs compared to landfill sites. Exposure to VOCs was found to be three times higher than at the landfill sites with the highest measurement considered to be at the limit for discomfort. VOC releases were highest during shredding of the waste (this was also higher than during collection). A wide variety of VOCs were identified, including chlorinated hydrocarbons, aliphatic, cyclic and aromatic hydrocarbons. The highest concentration monitored indoors was 3,000  $\mu\text{g m}^{-3}$ .

Some studies show an improvement in the health of workers after air handling equipment reduced dust levels within the plant. However, this often reduced bacteria and almost eliminated endotoxins but had no effect on fungi (Malmros *et al.*, 1992).

A wide-ranging review of occupational health problems and their possible causes from sorting and recycling domestic waste was undertaken in Denmark (Poulsen *et al.*, 1995b). Some issues of wider significance to this review include the frequent symptoms of ODTS, some cases of severe pulmonary disease, GI symptoms, and irritation of eyes and skin. They also commented on the lack of information on recycling glass and metal, biogas, risk and causal factors, OELs, as well as the technical monitoring problems which have not yet attracted sufficient attention. These monitoring problems could include:

- whether exposure measurements should state average or peak concentrations
- assessing aerosol exposure by measuring total particles or specified size ranges
- microbial variability in typical waste industry environments
- the use of viable or total bacterial counts
- comparisons between static area air sampling or personal air sampling
- an investigation of synergistic interactions between pollutants.

### **6.3 Materials recovery facilities**

There are probably well over 60 Materials Recovery Facilities (MRF) in the UK, depending how these plants are defined. The introduction of landfill tax and recycling targets has accelerated the growth of these facilities, or the conversion of basic waste transfer stations. Most published studies indicate similar findings relating to occupational health issues, as reported in the previous section (Gladding and Coggins, 1997). One UK study presents detailed microbial data, unfortunately all indoors. They also highlight the need for some

longitudinal studies on operatives before and during their course of employment (Gladding and Coggins, 1997). A recent study on nine MRFs in England and Wales (Gladding, 2002) concluded that workers exposed to higher levels of endotoxins and glucan exhibit various work related symptoms and the longer a worker is in the MRF environment, the more likely they are to become affected by various respiratory and GI symptoms. No significant seasonal differences were found and no significant concentrations of VOCs were measured in contrast with the Finnish study referred to previously (Kiviranta *et al.*, 1999).

#### **6.4 Scrapyards**

In scrapyards end of life vehicles (ELVs) and other material (mostly scrap metal) are dismantled and shredded thereby producing dust and vapours. The Government has suggested recycling targets for ELVs (ENDS, 2002) and scrapyard operations will probably increase. It is recognised that scrap metal cutting may present a health hazard (HoSF 1989) due to exposures to metals and chemicals (Malkin, 1995; Menzel *et al.*, 1998; Arion *et al.*, 2001). There is a dearth of information on scrapyard emissions and the Environment Agency has funded a project (Environment Agency, 2001c) to assess exposures.

#### **6.5 The production of refuse-derived fuel**

A small number of published references have examined the health impact of Refuse Derived Fuel (RDF), but only in relation to production workers. The surface of RDF pellets can have elevated microbial concentrations if the production temperatures are not sufficiently high (Mahar and Thorne, 1999). Workers report symptoms of sinus trouble, headaches, nose irritation and diarrhoea and workers employed for more than seven years had significant reductions in Forced Vital Capacity (FVC) and FEV (Mahar and Thorne, 1999). A similar US study (Mahar *et al.*, 1997) found no decrease in FEV and FVC but an increase in self reported symptoms such as headaches, rashes and hay fever.

There are published papers on emission testing of boiler plants operating on RDF. The reported air pollutants can be assessed using well established COMEAP methodologies, therefore it is not considered necessary to assess one of the various alternative fuels available to boiler operators.

#### **6.6 Recycling operations – but not secondary processing**

The detailed review of health problems from sorting and recycling referred to earlier (Poulsen *et al.*, 1995b), concluded that workers handling the source-sorted paper or cardboard fraction do not appear to have an elevated risk of occupational health problems related to bioaerosol exposure. Another Danish study (Breum *et al.*, 1999) generally agreed with this finding but observed that, with regard to viable bacteria, the dustiness of recyclable paper was comparable with mixed household waste. It is difficult to separate the difference in risk between ‘normal’ paper workers and paper-recycling workers (Zuskin *et al.*, 1998).

No published data have been found on the health effects of sorting or handling cullet (recycled glass). Personal communications through the closed EHNET (Environmental Health Network) system reveal that cullet handling is a common source of noise complaint but other issues have not arisen.

There are a number of published sources on the health effects of recycling operations. Unfortunately, they are unclear on the exact nature of the materials being reprocessed or the



resulting emissions. There is also a tendency to blur the distinction between specific industrial processes and reprocessing of waste. One example is a cross sectional epidemiological study on industrial waste recycling (Ahumada, 1998). This is probably a metal recycling operation on pollution abatement fly-ashes and dusts. A variety of expected health effects are reported but there is insufficient detail to add to any discussion on waste treatment options. Two US studies on battery recycling (Gittleman *et al.*, 1994; Wohl *et al.*, 1996) conclude that no public health impact was detected in the potentially affected community, however, one of the sites appears to have contaminated neighbouring properties and is reported to have adversely affected nearby children. Again, this is an example where specific emission data from plants is essential for the comparison of treatment options (Marchand *et al.*, 1995).

## **6.7 Reprocessing**

The reprocessing of recycled paper has been subject to allegations of increased cancer risk. Employees, numbering 5,377, at five paper recycling plants were included in a historical cohort study from 1965–1993 (Rix *et al.*, 1997). There was significantly more pharyngeal cancer cited among male workers, slightly more lung cancer and a doubling of risk for Hodgkin's disease. However, this was influenced by social confounders such as smoking and alcohol intake. It also appears similar to studies on normal paper mills.

There are published studies on metal exposures to scrap recycling workers (Lander *et al.*, 1999). These tend to be foundry furnace men and there is little difference in exposure between the scrap recyclers and other foundry workers. HIAs specific to metal recycling could use existing data on other metal processes if airborne releases have been adequately defined.

## **6.8 Priority areas for HIA**

As it has not been possible to critically appraise risk assessments, the following areas are considered essential elements to create quality data for use in future HIAs (in order of priority):

- 1) methods of minimising domestic waste storage but promoting source separation
- 2) downwind microbial surveys of transfer stations- MRFs
- 3) downwind surveys of VOCs
- 4) robust microbial measurement techniques
- 5) examination of synergy between biological and chemical parameters
- 6) the potential of releases from RDF stockpiles
- 7) emission rates for all pollutants from recycling and reprocessing facilities.

## 7 Community Mental Health

### 7.1 Introduction

Concern over possible health effects of exposure to hazardous waste sites has tended to concentrate on associations with adverse reproductive outcomes and various cancers. These serious diseases are relatively rare, yet any potential increased risk associated with residential proximity to waste sites arouses great public concern. However, the association between such waste sites and indicators of general physical and mental health should not be overlooked. Although potentially not as disabling to the affected individual, such symptoms are more common and would contribute greatly to the disease burden of any community.

This Chapter aims to identify and summarise the findings from studies that have compared the mental health of individuals residing near a fixed waste site with that of individuals living in an unexposed comparison area.

### 7.2 Mental health of residents in proximity to waste disposal sites

Eight primary studies have been identified which report data on psychiatric symptoms amongst residents in close proximity to a fixed site of waste disposal. Zmirou *et al.* (1994) and Deloraine *et al.* (1995) reported different analyses involving the same study sample from Montchanin, France. Overall, four study samples originated from USA, one was set in Australia, one came from the UK, and one sample was based in France. Five studies benefited from including samples of unexposed residents as a comparison group. All these studies involved hazardous waste, usually of an industrial chemical nature, disposed of in landfill sites. One study investigated mental health effects associated with a planned hazardous waste facility, whereas the remaining studies seemed to focus on sites which had ceased operations before the study period. However this was not always totally clear due to unspecified study characteristics. Psychiatric outcome measurements ranged from standardised questionnaires, for example the General Health Questionnaire 28 item (GHQ-28) and SF-36, to physician-rated psychiatric cases, and to using prescriptions for psychiatric medications as an indicator of psychiatric illness.

Table 7.1 summarises the results and methodology of the eight studies included in this review. The studies of Bachrach *et al.* (1989), Deloraine *et al.* (1995) and Zmirou *et al.* (1994) did not contain a comparison group of unexposed residents and the conclusions that can be drawn from the results are therefore relatively limited. Bachrach *et al.* (1989) reported that living near to a planned hazardous waste facility was associated with higher levels of non-specific psychological distress than would probably be expected in the general public. However, the relatively low income and educational level of the exposed residents probably contributed to some of this psychological distress and it would be difficult to infer much more from the data.

Deloraine *et al.* (1995) reported a higher prevalence of psychiatric cases defined by the local physician amongst the subjects living in the more exposed areas. However, this association might have been confounded by the greater levels of alcoholism amongst the residents of more exposed areas and biased by physicians' practice and expectations. Furthermore, since prevalence can be influenced by the duration of a disease, it might be more sensible to compare the incidence of new diagnoses of psychiatric cases. The authors reported no statistically significant difference in the incidence of psychiatric cases between the three areas of relative exposure. Finally, Zmirou *et al.* (1994) did not find any major difference in

prescription levels for psychiatric medications amongst their study sample before and after the closure of a toxic waste site.

The remaining five studies benefited from including samples of unexposed residents as a comparison group and their results should be seen as providing the main area of evidence in this review. Three of these studies reported a statistically significant difference in mean score on a scale of psychiatric morbidity between residents in the exposed area and residents of an unexposed area. Dunne *et al.* (1990) reported that exposed residents scored approximately six points higher on the GHQ-28 on average than unexposed residents, Foulks and McLellen (1992) reported that exposed residents scored 0.37 points higher on average on the Hopkins Symptom checklist-90 index (SCL-90), and Kilburn and Warshaw (1995) reported an average increase in 33 points on the Profile of Mood States (POMS) score in exposed residents. McCarron *et al.* (2000) found no significant difference in mean SF-36 score between residents exposed to a chromium waste landfill site and those not exposed, whilst Miller and McGeehin (1997) only found a significant association between diagnoses of anxiety, nervousness or depression and exposure to an oil processor site amongst individuals who were current drinkers.

Two studies investigated the effect of belief of harmful exposure on psychiatric morbidity. Dunne *et al.* (1990) stratified their study sample from the exposed town into those residents who believed themselves to be exposed and those who did not believe themselves to be exposed, and then compared these two groups with those residents from the unexposed town. Those who believed themselves to be directly exposed scored the highest on average on the GHQ-28 (Table 7.1). McCarron *et al.* (2000) found that the 25% of individuals who perceived chromium to be harmful to health scored on average at least 16 points lower on the SF-36 mental health dimension than did those who thought otherwise.

### **7.2.1 Discussion**

Although it was not possible to perform a meta-analysis to summarise the quantitative data in this review, there was some evidence to support the hypothesis that residents exposed to hazardous waste facilities exhibit greater levels of psychiatric morbidity than residents who are not exposed to such sites. This review focuses on the mental health of individuals living in close proximity to industrial hazardous waste sites, rather than municipal or domestic waste sites, due to the contents of the primary studies that were identified. Only eight references were confirmed to contain relevant data out of 71 potential identified abstracts. Of these eight references, the main area of evidence in this review arose from the five studies that included samples of unexposed residents as a comparison group. However, even these five studies suffered from limitations which make it difficult to infer causality in the relationship between belief in exposure to hazardous waste and poor psychiatric outcome.

## **7.3 Limitations of five primary studies that included unexposed comparison samples**

### **7.3.1 Sample selection and response bias**

In a cross-sectional survey it is important to derive a random sample of all those subjects who are potentially eligible, in order to generate a representative sample of the larger population of interest. Furthermore, once the sample has been selected, it is important to maintain its ability to represent the larger population by achieving a high response rate. Three of the five studies reported response rates (Dunne *et al.*, 1990, McCarron *et al.*, 2000, Miller and McGeehin, 1997), but it is difficult to assess whether this rate indicated the proportion of eligible subjects

included in the study samples, or instead the proportion of those who had been selected for sampling who actually responded.

McCarron *et al.* (2000) quoted an overall response rate, but it is of particular importance to assess whether the response rate varied by exposure. For example, the response rate amongst exposed individuals was systematically higher in the studies of Dunne *et al.* (1990) and Miller and McGeehin (1997). This differential response rate, which varied by exposure, could lead to a biased result overestimating the strength of association between belief in exposure to hazardous waste and psychiatric morbidity. None of the authors attempted to compare responders with non-responders, to assess the impact of possible response bias.

Finally, it is worth indicating that whatever the response rate in the studies by Foulks and McLellan (1992) or Kilburn and Warshaw (1995), the nature of the total eligible sample was likely to contribute to selection bias. For example, Foulks and McLellan were actually invited by the exposed residents to measure the psychological effects of living near chemical waste. Similarly, all of the exposed subjects in the study by Kilburn and Warshaw were either self-selected or were randomly taken from a roster of residents who were all plaintiffs in a class action suit against the waste disposal site operators.

### **7.3.2 Measurement of psychiatric morbidity**

All five studies relied on the individuals' self reporting of symptoms without any alternative method of assessment. This is prone both to random measurement error, which would tend to nullify any association between hazardous waste exposure and symptom reporting, and more importantly to bias. A systematic over-reporting of psychiatric symptoms amongst individuals exposed to hazardous waste would lead to a biased over-estimation of the strength of such an association. Local and media interest in the health of residents in proximity to a hazardous waste site might contribute to such biased reporting of symptoms.

Dunne *et al.* (1990) and McCarron *et al.* (2000) suggested that a belief of harmful exposure seemed to be the strongest predictor of psychiatric morbidity, and as such might add to reporting bias. This heightened concern and awareness of the putative health effects in the exposed population makes this bias almost unavoidable, and greatly adds to the difficulty in interpretation of study results (Ozonoff, 1982).

### **7.3.3 Confounding factors**

Important confounding factors that could influence psychiatric morbidity might include sex, age, general health, measures of socio-economic status, and measures of deprivation. These last three factors are particularly likely to also be associated with living in proximity to a fixed waste site. Therefore, any observed association between residing near a hazardous waste site and levels of psychiatric morbidity could be explained by variation in these confounders between the exposed and unexposed samples.

The study by Foulks and McLellan (1992) took no account of confounders whilst noting that the study area was particularly poor and underdeveloped. The remaining four studies attempted to select an unexposed comparison sample that was similar to the exposed sample mainly in age, sex and ethnicity, although Dunne *et al.* (1990) and McCarron *et al.* (2000) also attempted to control for socio-economic status or levels of deprivation. None of the studies presented results adjusted for physical health even though Dunne *et al.* (1990) reported this to be significantly worse amongst the sample of exposed residents.

### 7.3.4 Psychological morbidity – R&D Gaps

As noted above, confounding factors that may influence psychological morbidity include sex, age, general health, measures of socio-economic status and measures of deprivation. Knowledge, or belief, in the existence of harmful exposure seems to be a strong predictor of psychological morbidity and such belief may be affected by media reporting. There are three key R&D gaps identified in this review:

1. The need to explore whether it is the confounding factors that primarily determine psychological morbidity rather than proximity to putative hazard, suggests a cross-sectional approach could be adopted. Self-reported symptoms (e.g. GHQ-28, SF-36) could be compared in communities residing near operational hazardous landfill sites and in unexposed communities with comparable epidemiological and socio-economic characteristics.
2. To explore the potential for improving psychological morbidity in communities sited in proximity to, for example, hazardous waste landfill sites, an interventional approach could be adopted. Communities would be randomly allocated to intervention or non-intervention. Intervention communities would be targeted with educational / communication measures via an integrated approach involving Primary Care Trusts, Local Health Boards and Primary Care Trusts (Wales), General Practitioners as well as the Environment Agency and the Local Authority. Baseline measurements of psychological morbidity would be taken in all communities and post-intervention, these would be repeated.
3. The potential effects of special interest (campaign) group activity on the psychological morbidity of local communities that live in proximity to waste management sites, deserves attention. These groups have the potential to greatly influence psychological morbidity.

**Table 7.1 Studies that have investigated mental health amongst residents who live near a fixed site of waste disposal**

First author	Study design	Sample	Study period	Exposure	Outcomes	Main results	Ascertainment	Measurement	Confounding
Bachrach	Cross-sectional survey	Exposed sample from adjacent rural towns Rainbow Valley and Mobile, Arizona  N = 83 adults (20–80 yrs)  Comparison data from community mental health centre clients	Three months during 1986  Follow-up of 1982 survey	Planned hazardous waste facility	26 item demoralisation scale (Dohrenwend <i>et al.</i> , 1980) indicates non-specific psychological distress	Proportion of sample scoring above mean of community mental health centre clients  1986 = 41%  1982 = 36%	69% response (83/121)  57/83 also participated in 1982  22 ‘movers’ were younger, more educated, but had ‘similar levels’ of demoralisation	Self-reported outcome	Demoralisation scores highly correlated with perceived health status  $r = -0.58$  $P < 0.0001$  Sample were of low annual income and educational level
Deloraine	Physician’s practice based case-control study	432 cases  ‘conditions associated with dump emissions’  384 controls  ‘other conditions’  Montchanin, France	March – June 1990	Industrial toxic waste landfill within residential area. Operational 1979–1988	Psychiatry cases N = 56	Relative exposure category  0–100: prevalence = 4.7%  101–200: prevalence = 8.7%  > 301: prevalence = 8.0%  No significant differences in incidence of psychiatric cases	Eligible population of 6,000 inhabitants	Exposure assessment relies on indirect modelling.  Possible selection bias in practice consultation	Alcoholism greater in higher exposure categories  Prevalence estimates are unadjusted
Dunne	Cross-sectional survey	Exposed sample from Kingston, Queensland  Zone 1: < 300m radius  N=147 households	March– June 1989	Two open cut mines converted to municipal rubbish tip and	General Health Questionnaire (GHQ) 28 items (High score indicates worse	‘No statistically significant difference’ in GHQ-28 score between zones 1 and 2  Mean GHQ-28 scores	Zone 1 87% (147/169)  Zone 2 87% (110/126)	Self-reported outcome	Kingston mean scores on physical health symptoms checklist also higher than

		<p>Zone 2: 300–1,000m radius</p> <p><i>N</i> = 105 households</p> <p>Unexposed sample from Beenleigh 16 km away</p> <p><i>N</i> = 110 households</p> <p>Beenleigh matched to Kingston on four indicators of socio-economic status</p>		<p>unsupervised chemical waste disposal site.</p> <p>Operational 1954–1971 followed by residential development</p>	<p>health)</p>	<p>Kingston = 23.1</p> <p>Beenleigh = 17.5</p> <p><math>F = 12.3, df = 1358, P = 0.001</math></p> <p>Effect of belief of direct exposure</p> <p><i>N</i> = 82 Kingston believed exposed</p> <p><i>N</i> = 168 Kingston believed not exposed</p> <p><i>N</i> = 105 Beenleigh</p> <p>Mean GHQ-28 scores</p> <p>30.9 vs. 19.7 vs. 17.5</p> <p><math>F = 27.3, df = 2,350, P = 0.001</math></p>	<p>Beenleigh 69% (105/152)</p>		<p>Beenleigh (8.1 vs. 5.8 <math>F = 13.2, df = 1,358, P &lt; 0.001</math>)</p>
Foulks	Cross-sectional survey	<p>Exposed sample from a 30-household community</p> <p><i>N</i> = 72 adults</p> <p>Unexposed sample from similar sized community Greater Baltimore area, Maryland</p> <p><i>N</i> = 247 adults</p>	Unknown	<p>Chemical waste landfill ¼ mile from community.</p> <p>Operational for several decades</p>	<p>Hopkins Symptom checklist-90 item (SCL-90) (High score indicates worse health)</p>	<p>Mean (sd) SCL-90 score</p> <p>Exposed 0.79 (0.26)</p> <p>Unexposed 0.42 (0.19)</p> <p><math>P &lt; 0.05</math></p> <p>Mean (sd) depression sub-scale</p> <p>Exposed 1.78 (0.46)</p> <p>Unexposed 0.58 (0.19)</p> <p><math>P &lt; 0.05</math></p> <p>Mean (sd) anxiety sub-scale</p> <p>Exposed 0.61 (0.23)</p> <p>Unexposed 0.48 (0.18)</p>	<p>Unknown response rate</p> <p>Residents invited researchers to measure psychological effects of living near chemical waste</p>	<p>Self-reported outcome</p>	<p>No data on confounders</p> <p>Study area was a particularly poor, underdeveloped area</p>

						$P < 0.05$			
Kilburn	Cross-sectional survey	Exposed sample $N = 131$ (15–65 yrs) Unexposed sample from 35km away $N = 66$ Matched for age, sex and ethnicity	Unknown	Combustion Superfund site east of Baton Rouge, Louisiana.  Operational 1966–1983	Profile of Mood States (POMS)  (High score indicates worse health)	Mean (sd) POMS score Exposed 56.2 (39.9) Unexposed 23.2 (32.9) $P = 0.0002$  Mean (sd) depression subscale Exposed 15.5 (12.4) Unexposed 8.1 (9.7) $P = 0.001$	Exposed subjects either self-selected or randomly selected from 3,000 residents, all plaintiffs in suit against site operators	Self-reported outcome	Unexposed sample had more years of education and higher annual income
McCarron	Cross-sectional survey	Exposed sample from Cambuslang, Carmyle and Ruthergellan areas of Glasgow  $N =$ unspecified  Unexposed subjects from 10 km away Barrmulloch and Pollok area  $N =$ unspecified  Broad similarity in distribution of age, gender and Carstairs deprivation category	Unknown	Chromium waste landfill.  Operational 1820–1968	SF-36 health questionnaire  mental health dimension  (Low score indicates worse health)	Mean mental health score Exposed 69.1 Unexposed 68.9 Difference 0.2 (-3.2 to 3.5) $P = 0.92$ Adjusted difference 0.0 (-3.5 to 3.4) $P = 0.99$  Exposed group: 25% believed chromium harmful 75% believed not harmful  Mean mental health score 58.5 vs. 74.3  Difference -15.8 (-20.9 to -10.6) $P < 0.001$ Adjusted difference -15.0	400 houses from each area visited, overall response 78%.  Surveys 9am – 5pm led to unrepresentative sample in terms of employment and health status	Self-reported outcome	Adjustments made for sex, age, housing tenure, years lived at current address and perceived difficulty in selling house



						(-20.2 to -9.8) $P < 0.001$			
Miller	Cross-sectional survey	Exposed sample $N = 414$ subjects  Unexposed sample 4 miles east of target area, similar in race and ethnicity of population  $N = 360$ subjects	Unknown	Brio Refining Co. Inc. site and Dixie Oil Processor site  20 miles Southeast of Houston, Texas.  Operational 1957–1982	Frequent periods of anxiety, nervousness or depression diagnosed by health care provider	OR 4.4 (95% CI 1.2–16.1) for non-smoker, current drinkers  OR 1.5 (95% CI 0.3–6.3) for non-smoker, non-drinkers	55% exposed area  49% unexposed area	Self-reported outcome	ORs adjusted for age, race and sex
Zmirou	Retrospective cross-sectional survey	Exposed sample of local householders in General Social Security Plan  $N = 694$  Comparison of nationwide prescription rates	1987–1989	Industrial toxic waste landfill within residential area.  Operational 1979–1988 (as in Deloraine)	Morbidity estimated through mean number of consumption units of psychiatric medications prescribed by local physicians	All psychiatric drugs 18 months before closure = 4.32 units  18 months after closure = 4.26 units  % change -1.38  Nationwide % change +1.41	88.5% response	Drug prescription does not necessarily reflect disease prevalence	Likely influence of anxiety on drug use patterns and prescribing patterns

OR – odds ratio; 95% CI – 95% confidence interval

## 8 Conclusions

### 8.1 Introduction

Waste disposal options have been considered in relation to:

- the hazardous pollutants which may be generated
- current understanding of the relationship between dose and effect for the various pollutants
- current knowledge of public exposures arising from each
- epidemiological evidence of health effects associated with population exposure.

The following conclusions derive from these assessments.

### 8.2 Landfill

#### Emissions

The Environment Agency have reported on the composition and fluxes of trace components in landfill gas (Environment Agency, 2002a). The GasSim Lite software tool has also been developed to estimate annual mass emissions to air of Pollution Inventory substances from landfills.

#### Exposures

It is recognised that, to-date, there are insufficient data relating to exposures. However the Environment Agency has commissioned research through R&D Project P1-396 whose aim is to monitor levels of a wide range of air pollutants at the site boundary fence for two landfill sites (Environment Agency 2003a). Also the GasSim software tool has been developed to assess the potential exposure of residents in proximity to landfill.

#### Epidemiology

Epidemiological evidence from the USA suggests no excess risk of congenital malformations in women living within 1 mile of 1,281 landfill sites. However, a British study using a similar methodology reported a relative risk for all congenital abnormalities combined of 1.01 (99% Confidence Interval 1.005 to 1.023) for women living within 2km of 9,565 landfill sites, compared to a reference population who resided more than 2 km from all known landfill sites.

Epidemiological methods and data quality both require refinement if more robust risk estimates are to be developed. Refinements would include allowance for population

migration between zones and differential ascertainment through better registration of congenital malformations between zones, and over time. Much of the problem of differential under-ascertainment of congenital malformations could be addressed by comparing incidence before and after some major change in landfill status eg opening of a site, major change of operational gas control, although proximity, time and distance are still poor surrogates for exposure. The population at risk also needs to be defined more precisely than is currently allowed by the 'concentric circle' approach. For inhalation exposure, the population at risk could be defined by atmospheric dispersion modelling with due allowance for local meteorology and topography.

To date, biological monitoring of landfill workers and cytogenetic biomonitoring of population groups residing near landfill does not suggest that biological uptake of toxicants is of significance. However, such 'biomarker' research is in its infancy and the contaminant(s) of significance may not have been investigated. Epidemiological investigations should incorporate monitoring of biomarkers as possible confirmatory evidence of harm. The use of such biomarkers is common in occupational situations but is still being developed in wider population settings and has not been incorporated as a supplementary component of epidemiological studies.

It is also worth noting that public concern about birth defects, in particular, has the potential to deflect research efforts from investigating other potential adverse health effects such as renal and liver disease as well as effects on children's growth development and well being.

## **R&D gaps relating to landfill**

- A more sophisticated epidemiological approach should be developed to compare the incidence of congenital malformations for different magnitudes of exposure associated with the areas around landfill sites. This should address variation in ascertainment of birth defects as well as actual dispersion of potential pollutants based on local meteorology and topography, as opposed to the concentric circle modelling approach.
- More data are required on atmospheric concentrations of pollutants from landfills. Environmental monitoring of these sites might identify a subset of landfills where the potential risks are greatest and permit epidemiological effort to be focused on these. Such data should include measurements of particulate matter and the presence of pollutants on dust particles. The Environment Agency has funded on-going research in this area (Environment Agency, 2003a, 2003b).
- Personal exposure data, suitably corrected, for landfill workers would give an upper bound for conservative estimates of public exposures. The Environment Agency has funded research in this area (Environment Agency, 2000).
- The utility of biomarkers of exposure should be investigated in workers and in those residing in proximity to the emission sources.

## 8.3 Incineration

### Emissions

The older generation of incinerators were substantially worse polluters than those now in operation and were phased out once newer stricter emission and operation standards were introduced. Newer facilities are substantially less polluting and modern abatement technology will help reduce the hazard from emissions provided that they are properly operated at all times. The substances currently understood to be of relevance with regard to emissions from waste incineration are addressed by the Waste Incineration Directive.

### Exposures

Modern incinerators will emit pollutants into the environment, but it is unlikely that they would make a major contribution to the overall background level of air pollution in a particular area, if properly run and maintained. In many cases, incinerators do not make significant contributions to the overall level of pollution and emissions from other industries may present a greater hazard to health.

### Epidemiology

Research evidence, to date, does not suggest increased incidence of disease related to exposure to incineration emissions. The majority of studies are retrospective, use routinely collected data and are particularly reliant on inadequate exposure estimates. Most studies lack the statistical power needed to show a statistically significant excess of disease and/or are weakened by poor control of confounding factors.

It is reassuring that retrospective studies around the older generation of incinerators do not provide convincing evidence of a link with ill health, and where health effects have been reported, they typically disappear once potential confounding factors are taken into account (i.e. socio-economic deprivation, ethnicity, and personal lifestyle preferences such as smoking). Where some evidence of possible health effects remains, it cannot be directly linked with incinerator emissions.

The strength of many studies is weakened by poor estimates of exposure. Many still simply use distance from the incinerator as a proxy measure of exposure. The use of concentric circles to identify “at risk” populations does not take into account the influence of meteorological conditions or process characteristics (e.g. stack height, efflux velocity, plume temperature) and the zones of influence used (which can be up to 7.5 km distance) introduce considerable possibilities for confounding exposures. Improved studies using dispersion modelling and environmental monitoring will add to the scientific literature and we recommend that spatial epidemiology should be based on dispersion modelling of emissions and air monitoring data.

## R&D gaps relating to incineration

- Toxicological research, and to a limited extent epidemiological studies, have shown that airborne particulate matter becomes more toxic as the particle size decreases and the so-called ultrafine particles of less than 0.1 micrometres diameter are more toxic per unit mass than coarser particles within PM<sub>10</sub>. Concerns are often raised by campaign and local interest groups that these ultrafine particles can “slip” through the filtration devices on the incinerator plant, and therefore present a particularly toxic hazard to the community. Although this argument is not founded on fact (since filters are also very efficient in the removal of ultrafine particles), the size distribution of particles (particularly ultra fine particles) needs investigation and should be compared with background and other sources.
- There is also growing evidence that trace metals, and particularly transition elements, are major contributors to the toxicity of airborne particles, not through the inherent systemic toxicity of the metals, but due to their presence on the surface of particles leading to inflammatory reactions within the body. Whilst such ideas are still at an early stage of development, the question should be addressed as to whether the particles emitted by incinerators are any more enriched in trace metals than particles from other combustion sources, and therefore give rise to any possible concerns on these grounds. Trace metal content in emissions (specifically in particles) needs to be investigated and compared with background and other sources.
- Biomarkers can help demonstrate that exposure has occurred and biomarkers can be used to help identify exposed populations for spatial epidemiological studies, and also provide a useful comparison with other communities living outside the influence of an incinerator. The potential for biomarkers of exposure needs further evaluation.
- The Environment Agency should develop an approach to evaluating the effects of NO<sub>x</sub>/ozone interactions resulting from incinerator emissions.
- Environmental sampling programmes for dioxins and trace metals around new plant should be considered, as is specified in the PPC template for new MWI incinerators. Environmental sampling of air and soil before and after (for 5 years) the commissioning of new plants would add much to the literature.
- For many contaminants, it is necessary to account for existing body burdens and intakes from other sources during the risk assessment process. Assessing background exposures can place the risk from incinerator emissions into context. Further discussion and investigation involving the Foods Standards Agency (FSA) may be warranted to better evaluate the relevance of background exposures.
- Within the community living near to the incinerator, there may be individuals who obtain a significant fraction of their food from local farms, allotments and watercourses and could conceivably receive a higher exposure. Assessing the risk to such individuals is currently very difficult and there is little information on the relative consumption of locally-reared/grown foodstuffs by the general population (specifically those living in the vicinity of such combustion processes). Further research is required to evaluate the significance of local food chains and this should involve the FSA.

The Environment Agency has funded research aimed at quantifying public exposures arising from incineration and combustion techniques (Environment Agency, 2003c)

## 8.4 Composting

### Emissions

There is a growing body of data on emissions from composting operations. Data currently available give some indication of the magnitude of bioaerosol concentration expressed as colony forming units per cubic metre (cfu m<sup>-3</sup>) of total bacteria and total fungi. There is a need to supplement these data with information on components of bioaerosol currently not measured (e.g. endotoxin, mycotoxin, glucans bacteria and micro-organisms from intestinal flora of humans and domestic pets). There is a pressing need to develop sampling and analytical methodologies to assess the full range of risks associated with bioaerosol exposure (e.g. measurement of non-culturable spores which are likely to be as effective as culturable spores in promoting allergenic and non-immunological mechanisms).

It is recognised that the management of the composting process affects the levels of emissions and also that containment of the process should reduce bioaerosol concentrations at distance. More data are required to quantify the relative effectiveness of the operation and design of composting processes, to help reduce emissions. Improved management of the composting process is also likely to reduce offensive odours arising from undesirable degradation products (e.g. hydrogen sulphide, dimethyl sulphide, ammonia, aldehydes and octan-1-ol). Smells relating to these products are offensive and can be detected by people at very low environmental concentrations. This is not categorised as a risk to human health, but experience with odour from landfill indicates that offensive odour affects public perception of potential hazard and may lead to psychological stress.

### Exposure

Background levels are location, climate and season specific and may be affected by local activities (e.g. agriculture). Where dispersion modelling has been undertaken, the indications are that a  $3 \times 10_{10}$  reduction in emissions is achieved, in most cases, at a distance from source of the order of 250 metres. However, there are some reports of measured concentrations exceeding background levels at distances in excess of 250 metres. These data, allied to the uncertainties associated with modelling, indicate that within 250 metres of the source, exposures in excess of background levels may occur. If, therefore, human receptors are present within 250 metres of the source, a health risk assessment should be undertaken routinely. Until further understanding of human exposure to bioaerosols is gained through actual measurement, this “cutoff” distance should be treated as provisional, and should not be used to exclude such assessment for greater distances when circumstances (such as the proximity of other potential bioaerosol sources) suggest risk assessment would be prudent.

### Epidemiology

The literature search identified no epidemiological studies of the general population in proximity to composting operations, in contrast to the number of epidemiological studies which have been conducted around landfill sites and incinerators.

At high doses, or in susceptible individuals at lower doses, there is evidence of a causal link between some micro-organisms present as components of the bioaerosol from composting and adverse health effects in humans. For example, there are links between *Aspergillus*

*fumigatus* and invasive aspergillosis, bacteria and fungi and hypersensitivity pneumonitis, mycotoxins and mycotoxicoses, allergens and asthma as well as endotoxin and respiratory symptoms and fever. Occupational epidemiology in settings other than commercial composting has yielded much of this evidence eg workers exposed to mouldy hay or workers at mushroom farms. However, there are isolated case reports of hypersensitivity pneumonitis and bronchopulmonary aspergillosis associated with composting operations. There is some evidence that compost workers experience an increased incidence of health symptoms but most of this evidence is subject to possible bias eg a worker may report symptoms if he is told that these may be caused by work exposure. Dose-response data are also lacking for components of bioaerosols and this is unlikely to improve in any significant way in the near future.

### **R&D gaps relating to composting**

- Comparative environmental data on bioaerosols at distance from contained and uncontained systems should be collected
- More data are needed on background levels of bioaerosols according to season and location. Locations should be chosen to reflect differing background levels
- A cross-sectional study could be undertaken of sensitisation to specific allergens in receptors residing upwind and downwind of facilities
- The importance of composting facilities (as sources of bioaerosol) relative to other potential sources of the same pathogens needs to be determined
- A review of sampling and analytical techniques is required for the full range of pathogenic bioaerosol components. This is important for the adoption of standard protocols and the identification of gaps in methodology
- Modelling of bioaerosol dispersion requires development
- The threshold concentrations of specific components of bioaerosols giving rise to health effects is unknown and data are not likely to become available in the foreseeable future. The feasibility of inhalation studies of bioaerosol could be investigated. It is noted that the Environment Agency has funded research aimed at characterising emissions and health effects (Environment Agency, 2001a, 2001b)
- Consideration could be given to establishing a cohort study of workers involved with composting, recycling and related processes. This could incorporate measurement of serological biomarkers
- Personal exposure data for compost workers would give an upper bound for public exposures
- More data are needed relating to emissions of bioaerosols from a variety of processes.

## **8.5 Waste collection, transfer and recycling**

## Emissions and Exposure data

Further data are needed for waste collection, transfer and recycling operations. The Environment Agency has funded research into occupational and environmental exposures to particulate, bioaerosol and VOCs at 11 MRFs (Environment Agency, 2003d). Certain recycling operations (e.g. metal recycling on pollution abatement fly-ashes and dust) should be monitored to yield typical exposure profiles.

## Epidemiology

A small number of occupational epidemiological studies have been undertaken. The health impact of kerbside collection of segregated waste warrants investigation. Only countries spending large sums of money on waste collection have segregated collections at weekly (or more frequent) intervals. Homes storing separated organic waste have much higher levels of bioaerosol components.

## R&D gaps relating to waste collection, transfer and recycling

- Methods of minimising domestic waste storage but promoting source separation
- Emission rates for all pollutants from recycling and reprocessing facilities
- Downwind surveys of bioaerosol, VOCs
- Investigation of the efficiency of handling plant at controlling bioaerosols
- Noise and odour emissions from typical sites
- Investigation of home storage of separated organic waste
- Emissions from RDF stockpiles
- Development of measurement techniques for bioaerosol.

It is noted that the Environment Agency has funded research assessing risks to health at transfer stations (Environment Agency, forthcoming) and materials recovery facilities (Environment Agency, 2003d) as well as assessing exposures in scrapyards (Environment Agency, 2001c).

## 8.6 General conclusions

In terms of suitable HIA procedures within which the specific techniques fit, the majority of the experience developed in the UK has successfully made use of the Merseyside Health Impact Assessment Guidelines (Scott-Samuel *et al.*, 1998). However, experience in HIA is limited but new tools and approaches are under development. The Health Development Agency has produced its own generic guidance on HIA (Taylor and Blair-Stevens, 2002) which supports a flexible approach which can be adapted to local circumstances. The Health Development Agency also supports a website, the HIA Gateway, which holds copies of HIA reports and provides useful advice, including new approaches for carrying out HIA (<http://www.hiagateway.org.uk/>), and a similar website is being developed by WHO (<http://www.who.int/hia>). It would thus be inappropriate to recommend the use of any one approach for HIA, as the choice of the best tool will depend on the context.



It is difficult to produce convincing evidence that any particular policy making has been improved by use of HIA. However there is still a great deal of interest in application of this approach and a prospect that, with development of better processes, HIA could add value to the policy making process in many areas including that of waste management. The World Health Organization has provided advice on including HIA in Strategic Environmental Assessments (WHO European Centre for Environment and Health, 2001). At a project-level, those involved in HIA are generally convinced of its value, although there is a concern that without any legal status its effect on decision-making may be limited (Welsh Assembly Government, forthcoming). There are an increasing number of examples of the successful application of HIA (see, for example, BMA, 1998) where stakeholders have felt empowered by the process, but it is clear that HIA is not infallible and a poorly executed assessment may foster public opposition rather than constructive dialogue.

Evidence from HIAs which have been conducted, to date, indicate that best practice will involve the public in the identification of key health determinants, and that HIA needs to begin at the strategy level and be applied there before being applied to projects. It also indicates that quantified health risk assessments can improve confidence in a HIA to provide an evidence base against which to analyse impacts of concern. A key point is that the failure to engage with the public in identifying areas of concern is likely to damage public confidence in any assessment made.

The emissions of bioaerosol from non-incineration waste operations are of potential concern and data on exposures as well as dose effect data are lacking.

Emissions from landfill and composting are less well characterised than those from incineration. Dose-effect data are available for some of the more significant chemicals and metals emitted but are lacking for others.

Increasingly, biological tests which measure organ damage or dysfunction are employed in exposed and control populations. There is a trend for biological changes which are considered to result from cellular or organ damage to be considered as adverse health effects irrespective of whether clinical illness may be expected to result. Molecular biological techniques which, for example, investigate lymphocytes may prove useful in investigating links with chronic lymphocytic leukaemia (ATSDR, 1994b).

There is evidence to support the hypothesis that residents living in proximity to waste facilities exhibit greater levels of psychiatric morbidity than comparison groups. However, part of this association is likely to be explained by response bias, measurement bias and confounding, so the strength of the association is not clear. Perception of risk is clearly important in this connection and it may be that perception affects psychiatric morbidity or vice-versa. Psychiatric disorder is common, disabling and burdensome and any excess associated with waste disposal should be quantified.

Screening and scoping a particular development has a useful role but quantification of health impact is always the desirable goal. This in turn requires the availability of accurate exposure and epidemiological data. These data can be generated provided a systematic approach is adopted and that necessary resources are available.

## **Priorities for research to support HIA**

This review has identified that the priorities for research to support HIA of waste strategies are:

### **For all waste activities**

- A more sophisticated spatial epidemiological approach married to dispersion modelling of emissions that takes account of local meteorology and topography and preferably measurements of actual exposure
- A cross-sectional investigation of the role of confounding factors (e.g. deprivation, educational status) in determining psychological morbidity.

### **Landfill and Incineration**

- The development of biomarkers of exposure and effect
- The trace metal content of ultrafine particles should be compared to background and other sources.

### **Composting**

- Comparative environmental data on bioaerosols at distance from contained and uncontained systems
- Comparative measurements of specific priority pathogens in bioaerosols emitted by composting processes and other processes generating the same pathogens (sewage treatment, intensive agriculture?)
- Consolidation and implementation of a common set of sampling protocols and analytical techniques for bioaerosols
- Information required on source term emissions from different compost operations. This would support improved modelling of bioaerosol dispersal.

### **Waste Collection – Transfer – Recycling**

- Methods of minimizing domestic waste storage but promoting source separation
- Measurement of emissions e.g. bioaerosols, VOCs
- Investigation of control measures for emissions.

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## GLOSSARY

Actinomycetes	A specific group of micro-organisms, that are found in soil, are capable of forming very small spores and resemble bacteria and fungi
Aerated Static Pile	Static Pile (Windrow) composting is the making of long 3' to 6' high rows of compost and then moving and remixing daily to help aerating. Aerated Static Pile (ASP) composting is similar but air piping is laid under the pile to reduce the need to mix every day
Aerobic	An organism or process that requires oxygen
Aflatoxins	A group of secondary metabolites that are cancer-causing byproducts of a mould that grows on nuts and grains, particularly peanuts
Aliphatics	A group of organic chemical compounds in which the carbon atoms are linked in open chains
Allergens	A substance, such as pollen, that causes an allergy
Alveolitis	Condition where the lungs are allergic to fungus and other allergens
Anaerobic	An organism or process that requires the absence of free oxygen
Ascospores	A sexually produced fungal spore formed within a membranous oval or tubular sporangium of an ascomycete
Aspergillosis	An infection or disease caused by fungi of the genus <i>Aspergillus</i>
Atopy	hereditary allergy characterized by symptoms (as asthma or hay fever) produced upon exposure to the exciting antigen
Bacteriophage	A virus that infects and lyses certain bacteria
Basidiospores	A sexually produced fungal spore borne on a basidium fungi
Bioaerosol	Micro-organisms suspended in the air
Biofilter	A biologically active filter that processes effluent gases containing volatile organic compounds (VOCs) and other toxic and odorous compounds into harmless end products, which are primarily carbon dioxide and water
Bronchiolar	Of, or relating to the fine, thin-walled, tubular extensions of the bronchial tubes, leading to the lungs
BTEX compounds	Environmental priority pollutants, and fuel oxygenates - benzene, toluene, ethylbenzene, o-xylene, m-xylene, p-xylene
Carcinogenic	Producing or tending to produce cancer
Carcinogens	A cancer-causing substance or agent
Centralised Composting Facility	A special facility where organic material is prepared and processed into compost
Chronic pulmonary effects	Long-term disruption to the lung's ability to supply oxygenated blood to the heart

Clostridium perfringens	Bacteria which grows anaerobically in soils, the intestines of humans and animals, and is present in sewage
Cohort	A group of individuals having a statistical factor (as age or risk) in common
COMEAP	Committee on the Medical Effects of Air Pollutants
Congeners	A chemical substance related to a generic group of similar chemical compounds
Congenital malformations	Malformation existing from birth
Covered Windrow	Long 3' to 6' high rows of compost that covered with a geosynthetic cover and are moved and remixed daily to help aeration
Curing	In the context of composting, the final stabilisation of organic waste, following mesophilic and thermophilic degradation
Cytogenetic measurements	Measurements of cellular components, particularly chromosomes, associated with heredity.
Cytokines	Any of several regulatory proteins, such as the interleukins and lymphokines, that are released by cells of the immune system and act as intercellular mediators in the generation of an immune response
Cytotoxic	Of, relating to, or producing a toxic effect on cells
Dehydratase	An enzyme that catalyzes the removal of oxygen and hydrogen from organic compounds in the form of water
Delta-aminolaevulinic acid	A protein produced by the liver that is increased when another protein (an enzyme) has reduced function
Determinand	A chemical substance that is the subject of chemical analysis
Dihydrobiopterin reductase	An enzyme involved in blood synthesis
Dioxins	Abbreviation for chlorinated dibenzo-p-dioxin – a general term that describes a group of chemicals formed by the burning of chlorine-based chemical compounds with hydrocarbons
Diurnal	Recurring every day; having a daily cycle
Dose-response	The quantitative evaluation of the potential of developing adverse health effects as a result of exposure to a chemical
Endotoxins	Certain (toxic) substances found within bacterial cells and which are released only on cell lysis
Enteric	Of, relating to, or being within the intestine
Oestrogenic	Of, relating to, caused by, or being an oestrogen
EPAQS	Expert Panel on Air Quality Standards
Epidemiological	A branch of medical science that deals with the incidence, distribution, and control of disease in a population

EPS	Extracellular Polymeric Substance
Etiology	The cause or origin of a disease or disorder as determined by medical diagnosis
Faecal Coliforms	Microscopic organisms that live in the intestines of warm-blooded animals
Furans	One of a group of colorless, volatile, heterocyclic organic compounds containing a ring of four carbon atoms and one oxygen atom
Geosphere	The solid Earth that includes continental and oceanic crust as well as the various layers of the Earth's interior
Glucans	A polysaccharide (as glycogen) that is a polymer of glucose
Gram-negative	Of, relating to, or being a bacterium that does not retain the violet stain used in Gram's method
Granulomatous	Of, relating to, or characterized by granuloma - either of two diseases that result from a defect in the ability of white blood cells to destroy bacteria and fungi
Haematologic	Of or relating to blood or to haematology
Halogenated	To be treated or combined with a halogen (e.g. chlorine, bromine or iodine)
Heterocyclic	Containing more than one kind of atom joined in a ring
Heterogenous	Composed of parts of different unrelated kinds
HIA	Health Impact Assessment
Immunocompromised	Incapable of developing a normal immune response, usually as a result of disease, malnutrition, or immunosuppressive therapy
Immunoglobulins	Proteins produced in blood plasma as protection against infection
Immunosuppressive	Suppression of the immune response, as by drugs or radiation, in order to prevent the rejection of grafts or transplants or to control autoimmune diseases
Immunotoxic	Toxic to the immune system
Interleukin	Any of various compounds of low molecular weight that are produced by lymphocytes, macrophages, and monocytes and that function especially in regulation of the immune system and cell-mediated immunity
In-vessel	Composting system contained within a tank
Lamella Curtain	A flexible curtain device designed to seal an area of contaminated air from an adjacent environment
Lignin	A complex polymer, the chief noncarbohydrate constituent of wood, that binds to cellulose fibers and hardens and strengthens the cell walls of plants
Lipids	Any of a group of organic compounds, including the fats, oils, waxes, sterols, and triglycerides, that are insoluble in water but

	soluble in nonpolar organic solvents, are oily to the touch, and together with carbohydrates and proteins constitute the principal structural material of living cells
Lipophilic	Having an affinity for, tending to combine with, or capable of dissolving in lipids
Lymphocytic	Presence of the nearly colorless cells found in the blood, lymph, and lymphoid tissues, constituting approximately 25 percent of white blood cells and including B cells, which function in humoral immunity, and T cells, which function in cellular immunity
Macrophage	Any of the large phagocytic cells of the reticuloendothelial system
Malignancies	Likely to prove fatal as opposed to benign
Mercaptans	Pungent chemical substances resembling alcohols in structure, but with sulphur atoms replacing oxygen
Mesophilic	The process or organism that occurs at temperatures below 45-50 degrees Celsius
Microflora	A small or strictly localized flora (as of a microenvironment)
Mucocilliary	Of, relating to, or involving cilia of the mucous membranes of the respiratory system mucociliary transport in the lung
Municipal Solid Waste	Solid waste collected by, or on behalf of local authorities
Mutagenic	Inducing or capable of inducing genetic mutation - e.g. some chemicals and X rays are mutagenic agents
Mycelium	The vegetative part of a fungus, consisting of a mass of branching, threadlike hyphae
Mycotoxins	A toxin produced by a fungus
Necrosis	Death of cells or tissues through injury or disease, especially in a localized area of the body
Non-chromosomal congenital anomalies	Malformation existing from birth but not inherited from parents
Ochratoxins	A mycotoxin produced by a fungus of the genus <i>Aspergillus</i> ( <i>A. ochraceus</i> )
ODTS	Organic Dust Toxic Syndrome
Open Air Windrow	The making of long 3' to 6' high rows of compost and then moving and remixing daily to help aeration
Phagocytosis	The engulfing and ingestion of bacteria or other foreign bodies by phagocytes
Pharyngeal	Relating to or located in the region of the pharynx
Phlegm	Thick, sticky, stringy mucus secreted by the mucous membrane of the respiratory tract, as during a cold or other respiratory infection
PM <sub>10</sub>	Mass concentration of particulate matter collected by a sampler with a 50% cut-point at an aerodynamic particle diameter of 10 µm;

	mostly particles with aerodynamic diameter of 10 µm or less
PM <sub>2.5</sub>	Mass concentration of particulate matter collected by a sampler with a 50% cut-point at an aerodynamic particle diameter of 2.5 µm, mostly particles with aerodynamic diameter of 2.5 µm or less
Pneumonitis	Inflammation of lung tissue
Polychlorinated biphenyls (PCBs)	A chloro-biphenyl organic pollutant produced in various industries
Polychlorinated dibenzodioxins (PCDDs)	A group of 75 congeners covered within the term 'Dioxin' that are produced as trace by-products during chemical manufacture and thermal incineration processes
Polychlorinated dibenzofurans (PCDFs)	A group of 135 congeners covered within the term 'Dioxin' that are produced as trace by-products during chemical manufacture and thermal incineration processes
Polychlorinated hydrocarbons	Hydrophobic contaminants common at coal conversion, wood-treating, and manufactured-gas plant sites, formed by chlorination of hydrocarbon chains
Polycyclic Aromatic Hydrocarbons (PAHs)	Hydrocarbon compounds with multiple fused benzene rings. PAHs are typical components of asphalts, fuels, oils, and greases
Prions	A microscopic protein particle similar to a virus but lacking nucleic acid, thought to be the infectious agent responsible for scrapie and certain other degenerative diseases of the nervous system
Sarcomas	Cancer arising in bone, connective tissue or muscle
Sphygmomanometer	An instrument for measuring blood pressure in the arteries, especially one consisting of a pressure gauge and a rubber cuff that wraps around the upper arm and inflates to constrict the arteries
Spirometric tests	Measurement of the vital capacity of the lungs
Synergy	The interaction of two or more agents or forces so that their combined effect is greater than the sum of their individual effects
Thermogenic	Of or relating to the production of heat
Thermophylic	The process or organism that occurs at temperatures above 45-50 degrees Celsius
Thermotolerant	Capable of surviving high temperatures, especially those of pasteurization
Tumour Necrosis Factor-Alpha	A polypeptide cytokine implicated in Septic Shock
Volatile Organic Compounds (VOCs)	A group of organic compounds that volatilise easily at ambient temperatures, some of which are toxic and/or carcinogenic
WISARD	A mathematical life cycle assessment model for waste management developed by the Environment Agency ( <b>W</b> aste <b>I</b> ntegrated <b>S</b> ystems <b>A</b> ssessment for <b>R</b> ecovery and <b>D</b> iposal)

## List of Acronyms

AF	<i>Aspergillus fumigatus</i>
APEG	Airborne Particles Expert Group
ATSDR	Agency for Toxic Substances and Disease Registry
BMA	British Medical Association
BTEX	Compounds of Benzene, Toluene, Ethylbenzene and mixed Xylenes
CDC	Centre for Disease Control and Prevention (USA)
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act 1980 (USA) known as Superfund
CI	Confidence Interval
CNS	Central Nervous System
CO <sub>2</sub>	Carbon Dioxide
COMEAP	Department of Health's Committee on the Medical Effects of Air Pollutants
COPA	Control of Pollution Act 1974
COT	Committee on Toxicity of Chemicals in Food and Consumer Products
CREH	Centre for Research into Environmental Health
DDT	Dichlorodiphenyltrichloroethane (insecticide)
Defra	Department for Environment, Food and Rural Affairs
DETR	Department of Environment, Transport and the Regions
DG SANCO	Directorate General Health and Consumer Protection (European Commission)
DH	Department of Health (formerly DOH)
EA	Environmental Assessment
EC	European Community
EEC	European Economic Community
EHNET	Closed network for messages to all Environmental Health departments - administered by the Chartered Institute of Environmental Health
EIA	Environmental Impact Assessment
EPA	Environmental Protection Act 1990
EPAQS	Expert Panel on Air Quality Standards
ERM	Environmental Resources Management
ETS	Environmental Tobacco Smoke
EU	European Union
FAO	Food and Agriculture Organization of the United States
FEV	Forced Expiratory Volume

FVC	Forced Vital Capacity
GHQ	General Health Questionnaire
GI	Gastrointestinal
GP	General Practitioner
H <sub>2</sub> S	Hydrogen Sulphide
HCl	Hydrogen Chloride (hydrochloric acid)
HDA	Health Development Agency
HIA	Health Impact Assessment
HIV	Human Immunodeficiency Virus
HNO <sub>3</sub>	Nitric Acid
HP	Hypersensitivity Pneumonitis
IARC	International Agency for Research on Cancer
ICES	Interministerial Economic Structure Strengthening Programme (Netherlands)
IEH	Institute of Environmental Health
IPA	Intersectoral Policy Office (Netherlands)
IPC	Integrated Pollution Control
IRIS	US Environmental Protection Agency Integrated Risk Information System
MERITS	Merseyside Integrated Transport Strategy
MRF	Materials Recovery Facilities
MSW	Municipal Solid Waste
NAW	National Assembly for Wales
NHS	National Health Service
NO	Nitrogen Oxide
NO <sub>2</sub>	Nitrogen Dioxide
NOAEL	No Observed Adverse Effect Level
NO <sub>x</sub>	Oxides of Nitrogen
NPL	National Priorities List of the Environmental Protection Agency (USA)
OCDD	Octachlorodibenzo- <i>p</i> -dioxin (dioxin congener with eight chlorine atoms)
ODTS	Organic Dust Toxin Syndrome
O/E	Observed/Expected ratio
OELs	Occupational Exposure Limits
OES	Occupational Exposure Standards
OR	Odds Ratio
PACs	Polycyclic Aromatic Compounds
PAHs	Polycyclic Aromatic Hydrocarbons



PBDD	Polybrominated dibenzo- <i>p</i> -dioxin (general term for a brominated dioxin)
PBDF	Polybrominated dibenzofuran (general term for a brominated furan)
PCBs	Polychlorinated biphenyls
PCDD	Polychlorinated dibenzodioxins
PCDF	Polychlorinated dibenzofuran (general term for a chlorinated furan)
PeCDD	Pentachlorodibenzo- <i>p</i> -dioxin (dioxin congener with five chlorine atoms)
PM	Particulate Matter (subscript denotes maximum particulate size of interest in $\mu\text{m}$ )
POMS	Profile Of Mood States
PPC	Pollution Prevention and Control
PTMI	Provisional Tolerable Monthly Intake
R&D	Research and Development
RDF	Refuse Derived Fuel
RfD	Reference Dose
RPBs	Regional Planning Bodies
RTABs	Regional Technical Advisory Bodies
SAHSU	Small Area Health Statistics Unit
SCE	Sister Chromatid Exchange
SCF	Scientific Committee on Food
SEA	Strategic Environmental Assessment
SO <sub>2</sub>	Sulphur Dioxide
SVOCs	Semi-Volatile Organic Compounds
TBTO	Tributyltin oxide (biocide)
TCDD	Tetrachlorodibenzo- <i>p</i> -dioxin (dioxin congener with four chlorine atoms)
TEQ	Toxicity Equivalents
VOCs	Volatile Organic Compounds
UK	United Kingdom
USA	United States of America
WHO	World Health Organization
WHO JEFCA	Joint FAO/WHO Expert Committee on Food Additives
WHOROE	World Health Organization Regional Office for Europe
WID REIA	Regulatory and Environmental Impact Assessment of the Proposed Waste Incineration Directive
WISARD	Waste Integrated Systems Assessment for Recovery and Disposal (Environment Agency's Life Cycle Assessment software tool)
WML	Waste Management Licence

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## Appendix A Questionnaire survey of local authorities' experience with HIA

The questionnaire sent to Environmental Health Officers and Planning Officers of local authorities in the UK is reproduced below:

*'The Environment Agency have commissioned a study entitled "A Health Impact Assessment of the Landfill and Government Waste Strategy" and this is being carried out by a consortium coordinated by Professor David Kay of the University of Wales Aberystwyth.*

*As part of this study, I would like to ask a few very short questions to get an idea of the experience that has built up in the field of health impact assessment in the UK; as part of our study, we clearly need to avoid reinventing the wheel. I would be grateful for very brief and honest responses (i.e. yes/no answers with clarification where appropriate) to the six questions below as soon as possible as the Environment Agency has set tight deadlines for completion of the overall study.*

- 1. Do you feel you understand what is meant by the term Health Impact Assessment?*
  
- 2. Has your council asked any developers to carry out a Health Impact Assessment of any project proposals? (If yes – how many times)*
  
- 3. Has your council received any Health Impact Assessments submitted as a section of an Environmental Impact Assessment with a planning application? (If yes – how many)*
  
- 4. Has your council received any Health Impact Assessments submitted as a separate document with a planning application? (If yes – how many)*
  
- 5. Has your council carried out (or commissioned) any of its own Health Impact Assessments of proposed projects? (If yes – how many)*
  
- 6. Has your council carried out (or commissioned) any of its own Health Impact Assessments of proposed plans or programmes? (If yes – how many)'*

## **Appendix B Analysis of HIA documents submitted in the UK**

### ***Wrexham Resource Recovery Centre***

This document is an Environmental Impact Statement which has a Health Impact Assessment Report as part of one of the technical appendices. The Health Impact Assessment Report is 55 pages long, not including figures and appendices, and so is a comprehensive piece of work. The authors of the report are a company called GIBB, based in Reading.

### **Issues considered**

Two main issues were examined. The first was potential contamination of the site, specifically soil and controlled water contamination, associated with the past use of the site. The second was the potential health effects of air emissions from the site once operational.

### **Approaches used**

For the potential past contamination, a quantitative risk assessment was carried out based on the Risk Based Corrective Action model for petroleum release sites developed by the American Society for Testing and Materials (ASTM, 1988).

For the potential health implication of air emissions, the assessment refers to the lack of specific UK guidance and instead uses the US EPA Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities (USEPA, 1998a,b,c). This process requires air dispersion modelling to obtain some of the values needed by the protocol.

The level of technical expertise exhibited in the document is impressive and the quantification of health impacts is also impressive in that various pathways are identified, concentrations predicted and comparison made with standards which exist – UK ones where they exist, WHO or other where they do not.

Two other approaches were used to look at the potential effects of air pollution. One was that of the Committee on Medical Effects of Air Pollutants (COMEAP, 1998), the other was a coefficient developed by the Institute of Occupational Medicine (IOM, 2000).

### **Stakeholder involvement**

There is no evidence of any stakeholder involvement at all. The Environmental Impact Statement of which this is part would be accessible to the public, but neither the public nor anyone else appears to have been consulted over perceptions of concern or over the selection of sensitive receptors used. The consultants have used their own expertise to identify these receptors based on protocols derived in the USA.

### **Conclusions**

For the contaminated land issue, the findings are that there is no groundwater ingestion pathway to consider as the groundwater is isolated from any contamination in the soil by a relatively impermeable boulder clay layer. It is also concluded that there is no potential risk to either construction workers or commercial workers in the operational phase.

For the air pollution, potential receptors (along with potential pathways) were identified as a school, adult and child farmers, adult workers at Wrexham Industrial Estates, adult and child residents and adult and child fishermen (the term used – presumably derived from the EPA

document!). Most exposure pathways were considered not to pose a health risk except the dioxin intake via ingestion of fish from a river. Using this pathway there would be an expected number of deaths of 1.9 over 25 years, although this is based on a total population within a 2 km radius of 8321 people – the majority of which would not ingest fish!

Of the two other approaches used, the COMEAP approach gives a figure for the number of deaths and respiratory hospital admission which could be expected due to the development (4.5 and 5.5 over 25 years respectively) whilst the IOM approach gives a potential relative mortality rate due to the development of 0.18%. However, two different figures are given in the report and the lower one of 0.12% appears in the executive summary!

### ***Integrated Recycling Facility at Sydallt***

This HIA forms part of an Environmental Assessment and makes specific reference to the National Assembly for Wales' 'Managing Waste Sustainably' document produced in July 2001 which indicated the desirability of carrying out HIA. The HIA is 21 pages long but has additional tables and appendices which were not supplied and follows the advice given in Annex 9 of 'Managing Waste Sustainably' in that the following elements are addressed:

- scoping
- profiling
- risk assessment
- risk communication
- risk management
- auditing.

### **Issues considered**

A scoping exercise identified a number of hazards/sources and these were considered with respect to the following separate operations of the plant:

- non-hazardous waste recycling operations
- metals and inert materials recycling
- hazardous waste recycling area
- anaerobic digester and gassifier building
- power generation
- outside operational area
- water management.

### **Approaches used**

For each of the operations identified, there is a description of the processes involved and a table indicating the relevant sources, pathways and receptors of potential risk together with the risk management, audit and communication measures used to alleviate the impacts at source. The approach is very much qualitative with no attempt being made to quantify risk beyond the grading of risk to receptors illustrated in Table A.1:

**Table A.1 Risk to receptors**

Negligible Level of Risk	N
Low Level of Risk	Y
Medium Level of Risk	YY
High Level of Risk	YYY

### **Stakeholder involvement**

There is a table which indicates the hazards and sources considered and their relevant pathways and receptors. However, there is no indication that there was any stakeholder involvement in this scoping exercise.

### **Conclusions**

The conclusions are that there is minimal risk to receptors for all operations except ‘hazardous waste recycling area’ for which ‘there is a risk to the receptors from waste delivered to this area’.

### **Shortstow development, Bedfordshire Health**

This HIA covers three options for the route of the A600 with regard to a proposed residential development (Shortstow). The HIA is a free-standing, 20-page document and was written by an individual who is no longer resident in the UK.

### **Issues considered**

Four separate issues were considered, although the exact reason for their choice is not clear. The author of the work did carry out site visits and read through existing literature on the proposed development:

- safety, including traffic accidents and crime and disorder
- the physical environment, mainly air pollution and noise
- the social environment, including the effects of community severance
- access to services and facilities.

### **Approaches used**

Methods were influenced by other available HIA work, particularly assessments carried out by the Liverpool Public Health Observatory. Basically, information was collected and analysed as follows:

- The different ways in which alternative routes of the A600 could have an impact on health were identified
- The evidence for the nature and strength of these impacts was then assessed
- This evidence was then applied to Shortstow by using a series of matrices to build an outline of the likely health impacts associated with different routes of the A600.

The matrices were specifically used to identify:

- the people within the community most likely to be affected

- the nature and size of the health impact
- the strength and evidence for the health impact
- how the health impact might be mitigated
- which option for the A600 had least impact.

### **Stakeholder involvement**

The public were not involved due to time constraints, but Bedford Borough Council were consulted, as were Bedfordshire Police and Bedfordshire County Council. The involvement was more a case of data gathering rather than two-way dialogue.

### **Conclusions**

The conclusions were that one road route was significantly better than the other two in terms of health impacts.

### ***Change of use of the former Psychiatric Hospital, 4 Maida Vale***

This is a rapid HIA very much based on the output from a facilitated workshop involving a good cross section of stakeholders. This is an initial document which will be developed based on consultation. The context is that Westminster City Council were carrying out an HIA of 'Best Value' using a rapid HIA approach of one topic selected in each of the authority's departments. This is just one of those rapid HIAs contributing to the wider goal.

### **Issues considered**

The workshop process described is very much about identifying the issues rather than dealing with them in any specific detail.

### **Approaches used**

The Merseyside Guidelines for Health Impact Assessment were used as a framework for this assessment. A workshop was conducted as part of a rapid HIA approach where the participants were asked to:

- Identify the key issues and priorities and the population groups which might be affected by the change of use of the former hospital
- Select the issues or health determinants felt to be most important and to apply rapid HIA to:
  - determine Westminster Council's activities in relation to the change of use which might influence the health determinants
  - identify the potential positive and negative impacts of these activities on health status or the wider determinant of health
  - identify recommendations to maximise positive health impacts and minimise negative ones.

### **Stakeholder involvement**

Twelve people were involved in the rapid HIA workshop, representing the health authority, architects, local authority professionals, local authority councillors and an independent public health consultant.

Discussions with ‘key informants’ are based on a list of key areas influencing health drawn from the Merseyside Guidelines for Health Impact Assessment. These are reproduced in Table A.2.

**Table A.2 Key areas influencing health. Source: Scott-Samuel et al. (1998)**

Categories of influences on health	Examples of specific influences (health determinants)
Biological factors	Age, sex, genetic factors
Personal/family circumstances and lifestyle	Family structure and functioning, primary/secondary/adult education, occupation, unemployment, income, risk-taking behaviour, diet, smoking, alcohol, substance misuse, exercise, recreation, means of transport (cycle/car ownership)
Social environment	Culture, peer pressures, discrimination, social support (neighbourliness, social networks/isolation), community/cultural/spiritual participation
Physical environment	Air, water, housing conditions, working conditions, noise, smell, view, public safety, civic design, shops (location/range/quality), communications (road/rail), land use, waste disposal, energy, local environmental features
Public services	Access to (location/disabled access/costs) and quality of primary/community/secondary health care, child care, social services, housing/leisure/employment/social security services, public transport, policing, other health-relevant public services, non-statutory agencies and services
Public policy	Economic/social/environmental/health trends, local and national priorities, policies, programmes, projects

## Conclusions

Two main recommendations, as opposed to conclusions, came from the process:

- early identification of requirements (of health authority/developers/RSLs and others) through the production of planning briefs
- the need to develop closer working relationships and look at all new applications while balancing what is ‘achievable’ with the ‘ideal’.

### **Community safety projects, Huyton SRB Area**

This is a HIA carried out by Liverpool Health Observatory for St. Helens and Knowsley Health Authority and supported by Huyton’s Single Regeneration Budget. This uses the Merseyside Guidelines for Health Impact Assessment but was carried out very early in the testing of those Guidelines.

## Issues considered

Key informants were used and brainstorming carried out to identify health impacts.



## **Approaches used**

The Merseyside Guidelines for Health Impact Assessment were used as a framework for this assessment.

## **Stakeholder involvement**

Two separate brainstorming sessions were carried out, the first including representatives from planning, community development, police, drug services, Groundwork Trust, environmental health and housing. The second contained representatives from the SRB working group already in existence on 'quality of life and community development'. In addition to these two sessions, a number of interviews were carried out.

## **Conclusions**

Chief recommendations based on the findings were:

- involvement of local people in designing out crime
- development opportunities for employment and training
- before and after surveys of residents
- increased participation on Community Safety Working Group
- participatory safety education for children
- project timetables
- recreation for local people
- reduce the environmental cues associated with fear of crime
- primary prevention – increased levels of preschooling.

## ***Alconbury HIA***

This is an HIA about the redevelopment of a former US Air Force base into one of Europe's largest rail and air freight distribution centres (the air freight proposal was later dropped). The HIA was carried out for Cambridgeshire Health Authority.

## **Issues considered**

The issues addressed were:

- transport
- noise and vibration
- air quality
- accident risks – construction and road traffic accidents
- employment and economic growth
- flooding
- contaminants
- visual impact
- impact on emergency and health services.

## **Approaches used**

An Alconbury Health Impact Group was established and based their approach on the Merseyside Guidelines for Health Impact Assessment.

Various techniques were used to examine likely health effects of the issues identified. Of most relevance are the studies on air pollution and associated effects, for which the technique used was that of COMEAP (1998), and also a technique drawn from the WHO three cities project (Dora *et al.*, 1999) to carry out a worst-case scenario prediction of ill health associated with air pollution resulting from the project.

## **Stakeholder involvement**

The Alconbury Health Impact Group comprises statutory bodies and representatives of the local population – 20 people in total.

## **Conclusions**

Separate conclusions were presented for each of the issues considered, but the main outcomes were summarised in a table demonstrating that:

- some houses would be affected by noise
- there is an increased risk of accidents for workers during construction and operation of the site
- there is an increased risk of road traffic accidents from 8500 extra vehicles a year leading to 1–19 injury-only accidents per year and one death every three to 60 years
- increased pollution from vehicles may lead to one admission every two years and one death every five years.

## ***National Botanic Garden of Wales***

This is a HIA about the National Botanic Garden of Wales, carried out retrospectively to assist future planning and to gain experience of using HIA within Wales

## **Issues considered**

‘Intermediate factors’ were identified as:

- employment (construction phase)
- employment (operational phase)
- cash injection into local economy
- travel
- effect on visitors
  - effect of visit for general visitors
  - educational visits for schoolchildren
  - educational visits for adult learners
- effect on volunteers
  - social involvement – volunteers (with learning disability)
  - social involvement – volunteers (general).

## **Approaches used**

The use of key informants and identification of issues to consider using these informants follows the Merseyside Guidelines approach, though this isn't specifically mentioned.

## **Stakeholder involvement**

The stakeholders were named as:

- garden project staff
- members of the Garden
- residents of Llanarthne
- funding organisations
- visitors
- Health Advisory Group of Garden
- trustees of the Garden.

## **Conclusions**

Parallels were drawn with the HIA of the International Astronomy and Space Exploration Centre in the Wirral in that much of the impact was relatively small magnitude on a large number of people. The approach taken was deemed to be satisfactory, with the stakeholder involvement being singled out for more effort in future studies. The uncertainty associated with the predictions made was highlighted.

## ***The health potential of the Objective 1 Programmes for West Wales and the Valleys***

This is a preliminary HIA of the Objective 1 programme and, as such, is directed at a strategic rather than a project-level.

## **Issues considered**

'Intermediate factors' were identified by the authors as:

- general economic, environment
- living and working conditions
  - work
  - living conditions
  - services and IT
- social and community influences
- individuals
  - capacity
  - lifestyles.

## **Approaches used**

As this is preliminary HIA the full approach has not been developed, but the procedure looks set to follow that of the Merseyside Guidelines for Health Impact Assessment.

### **Stakeholder involvement**

The preliminary HIA is supposed to be used as the basis for stakeholder involvement. As such, there has been no involvement to date

### **Conclusions**

The Objective 1 programme has a direct link with people's health and should make a significant contribution to efforts to achieve better levels of health.

## Appendix C Methodology

This Chapter explains the common approach used to identify relevant literature for the investigations into the health impacts of various waste treatment processes detailed in Chapters 3 to 6 and the consideration of the mental health effects resulting from waste treatment detailed in Chapter 7.

### Health Impact Assessment of composting, incineration, recycling and landfill

This literature search was carried out by staff in the Division of Information Services (University of Wales College of Medicine)<sup>1</sup> to support a HIA of the UK Landfill Directive Policy by the *Centre for Research in the Environment and Health* (CREH).

#### Databases searched

Searches were carried out over the following databases:

Biological Abstracts	(1980–2001)
BIOSIS Previews	(1970–2001)
CAB Abstracts	(1973–2001)
CancerLIT	(1975–2001)
CSA Environmental Science	(1984–2001)
Current Contents Agriculture	(2000–2001)
Embase	(1974–2001)
Medline	(1966–2001)
PreMedline	(to 19 Dec 2001)
SIGLE	(1980–2001/06)
Toxline	(1980–2001)

#### Search strategy

Database: OVID format

Search Strategy:

- 
- 1 landfill.sh. or landfill.mp.
  - 2 waste.mp.
  - 3 (waste management method or waste management or waste treatment method).sh.
  - 4 recycl:.mp.
  - 5 incinerat:.mp.
  - 6 composting.sh. or compost:.mp.
- 

<sup>1</sup> Mrs Hilary Kitcher, Mrs Mala Mann, Ms Lesley Sander, Dr Alison Weightman

7 (refuse adj3 (recycl: or treatment or transfer or disposal)).mp.  
8 or/1–7  
9 (health adj3 (impact or effect: or risk: or assessment: or hazard:)).mp.  
10 occupational health.mp.  
11 (allergy or hypersensitivity or pneumonitis or inflammation or  
asthma).mp.  
12 (irritation of mucous membrane or lung disease).mp.  
13 aspergillosis.mp.  
14 (toxic: or harm:).mp.  
15 (release or dust or dust emissions).mp.  
16 (source emission rate or dispersion).mp.  
17 exposure.mp.  
18 (bioaerosol: or aeroallergen:).mp.  
19 aspergillus fumigatus.mp.  
20 colony-forming units.mp.  
21 (toxoplasma or toxocara or cryptosporidium or lepitospira).mp.  
22 micro-organism:.mp.  
23 microbiology.sh.  
24 season:.mp.  
25 pathogen:.mp.  
26 (escherichia coli or salmonella or coliforms).mp.  
27 or/9–26  
28 8 and 27  
29 limit 28 to English language

The databases were searched between 17 and 21 December 2001.

An initial trawl of the results of the search (c.38,000 hits, including some duplicates missed by the *Reference Manager* import software) was carried out as follows:

- scan, titles and keywords (index headings) of relevant papers (looking at the health effects of waste treatment processes) to select the best words/phrases for the selection of papers to an ‘inclusions’ database;
- transfer references to the inclusions database (using a large number of relevant title and keywords) until very few relevant papers remained in the initial database (i.e.  $\leq 1$  in 300 abstracts were found to be relevant (c.5000 entries in the inclusions database));
- exclude any remaining duplicates from the inclusions database;
- scan abstracts in the inclusions database to make a final selection of papers, on the basis of agreed inclusion/exclusion criteria (see below);

- carry out a final search of the initial database for publications by all authors with four or more relevant papers, as identified in the final inclusions database, to double-check that no papers from experts in the area had been missed (1078 entries in the *final-inclusions* database).

Please note that the initial (huge) database has been retained and can be searched for additional papers, with any specific search requests identified by reviewers.

### **Inclusion criteria (for abstracts)**

The criteria for inclusion were:

1. all abstracts looking at the impact on health (of humans and other mammals) of methods of waste disposal (incineration, landfill, composting, recycling), including studies of hazardous waste, waste water (ground water) and animal waste (dead animals);
2. all abstracts looking at the measurement of microbes (bacteria, fungi, viruses, bacteriophages) as indicators of health risk, particularly in composting processes. (These papers are included in the ‘composting’ database – see below);
3. the following geographic areas: USA, Canada, Europe, Japan, South America
4. any studies looking at public anxiety (mental health) concerns associated with these processes (NB This is additional to the specific search strategy used for this issue, and detailed below).

### **Exclusion criteria**

The following were excluded:

1. all geographic areas not identified above;
2. radioactive waste;
3. sewage;
4. health studies looking at microbes, fish, plants;
5. animal waste other than animal carcasses (e.g. manure);
6. industrial waste/effluents unless treated by incineration.

The final inclusions database was then tagged appropriately so that groups of references could be retrieved under the following headings (some publications appear under two or more headings):

1. Incineration ( $n = 159$ )
2. Landfill ( $n = 129$ )
3. Composting (and microbiology) ( $n = 144$ )
4. Recycling/Transfer ( $n = 43$ )
5. Hazardous waste ( $n = 523$ )
6. Animal waste ( $n = 22$ )
7. Waste water/ground water ( $n = 168$ )
8. General waste ( $n = 47$ )
9. Public anxiety ( $n = 6$ ; cross-checked against specific literature search (see below))

Although the databases searched include some ‘grey’ literature (particularly from the database SIGLE), coverage is likely to have been limited. Relevant grey literature was sought and supplied to reviewers.

## **Public anxiety/mental health effects**

### **Databases searched**

Biological Abstracts	(1980–2001)
BIOSIS Previews	(1970–2001)
CAB	(1973–2001)
CSA Environmental Science & Pollution Management	(1984–2001)
Current Contents Agri, Bio, Environ Sci	(2000–2001)
CancerLIT	(1975–2001)
Embase	(1974–2001)
Medline	(1966–2001)
PsychINFO	(1967–2001)
PreMedline	(to 19 December 2001)
Toxline	(1980–2001)
SIGLE	(1980–2001/06)

### **Literature Search Terms**

- 1 landfill.sh. or landfill:.mp. [mp=title, abstract or subject heading,]
- 2 (waste management method or waste management or waste treatment method).sh.
- 3 waste.mp
- 4 incinerat:.mp.
- 5 composting.sh. or compost:.mp.
- 6 (refuse adj3 (recycl: or dispos:)).mp.
- 7 or/1–6
- 8 (depression or depressive or anxiety or worry or neuros: or neurotic or mental: or psychiat:).mp.
- 9 7 and 8

Final number of relevant abstracts = 71;

18 were selected for full-text analysis.



## Appendix D A Review of the Health Effects of Key Chemical Contaminants Associated with Waste Disposal

Industrial waste and Municipal Solid Waste (MSW) contain differing quantities of chemicals and metals. Industrial waste may contain bulk chemicals and MSW may contain chemicals and metals such as mercury, lead, chromium, cadmium, zinc and copper derived from batteries, plastics, glassware and ferrous metals. Chemicals present in the raw materials for composting may be degraded during the composting process. However, heavy metals cannot be degraded and there is evidence to show that heavy metals such as arsenic, cadmium, lead, nickel, mercury, selenium and chromium are present in raised levels in mature compost (Deportes *et al.*, 1995). Leachates from the composting process may also contain heavy metals. Therefore waste incineration, recycling, composting or landfill all have the potential to give rise to emissions of various pollutants.

In all cases the key question is the extent to which they will reach the wider environment and what degree of exposure to these chemicals will be encountered by the general public, either in the immediate vicinity or more widely. The question then is whether waste disposal results in a significant increase in exposure over background. Background exposure is an important consideration since many of the chemicals that are likely to be present in environmental media as a consequence of waste disposal will be found in the environment due to sources other than in waste disposal and often at higher concentrations. Many of these chemicals arise from a wide range of sources and are ubiquitous in the environment (e.g. dioxins, PCBs, PAHs) (Enviros Aspinwall, 2002).

There are many substances that can arise in waste disposal and there are various approaches to determining which are the most important. The Agency for Toxic Substances and Disease Registry (ATSDR) was created in the USA by the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) of 1980, known as Superfund. ATSDR's mission is to prevent adverse effects to human health resulting from exposure to hazardous substances in the environment, primarily arising from waste disposal sites that received toxic waste in relatively uncontrolled circumstances in the past. There are approximately 40,000 sites on the Environmental Protection Agency's (EPA) inventory of uncontrolled waste sites and of these approximately 1300 have been added to the National Priorities List (NPL). The NPL represents those sites that pose the most significant threats to public health. The ATSDR's hazardous substance release/health effects database (Haz Dat) listed 2000 unique substances identified by EPA during site characterisation studies. Over a hundred different chemicals can be found at a single waste site and exposures to multiple chemicals are more common than exposures to single chemicals. However, most of these will be present in very small quantities often at, or even below, normal background levels. A prioritized list has been developed of 275 hazardous substances that pose the greatest hazard to human health (De Rosa *et al.*, 1996).

ATSDR has undertaken environmental monitoring to determine which of these substances are found most frequently in completed exposure pathways (ATSDR, 1994a). The percentage of sites of which the 10 substances most frequently found in completed exposure pathways are: Lead (79%), Trichloroethylene (66%), Benzene (64%), Arsenic (60%), Chromium (57%), Cadmium (52%), Tetrachloroethylene (49%), Toluene (45%), Di-2-ethylhexyl phthalate (43%) and Vinyl Chloride (41%).

Tri and tetrachloroethene appear on the ATSDR list partly because of their potential to migrate to groundwater, where they can build up over time. In the UK there are very few

individuals that use private supplies and ground water used for public supply is closely monitored for substances such as these. It would, therefore, be unusual to find them present at concentrations above the drinking water standards. This is also true of many other substances. As a consequence public water supplies are not generally an exposure route of concern in the UK. Some substances occur widely in the environment (e.g. Di-2-ethylhexyl phthalate, benzene and toluene) and exposures arising from waste disposal is unlikely to give rise to a significant increase above background levels.

Many substances are highly volatile and exposure through loss to the atmosphere and subsequent inhalation is the most likely route of exposure. However, this exposure will be influenced by local climatic conditions. Other substances are less volatile and of low water solubility and as a consequence will adsorb to particles and soil. The primary source of exposure will be through dust and other particles, which will also be influenced by local climatic conditions and other factors such as the movement of soil and dust off site by vehicle movements. Some substances such as metals may be in a form that is both of low solubility and low bioavailability so the chemical speciation of the metal in the specific circumstances is key to not only exposure but also uptake by exposed humans.

The food chain may also offer a potential route of exposure. Heavy metals and other substances may enter food through the deposition of dust and rain, containing the metal, on crops. Deposition to soil or application of mature compost may also make these elements potentially available for uptake by plants. However, once in the soil environment, many substances will typically become bound to clay and organic matter and will be effectively immobilized. Uptake by plants and leaching into water supplies is expected to be low and highly variable between plant species and soil types. Furthermore, most people in the UK receive the majority of their food from a large number of separate and geographically diverse sources and therefore, the potential for exposure will be comparatively low.

A number of substances are subject to regulations associated with waste disposal (e.g. the Waste Incineration Directive), whilst others have been detected in landfill gas (Parker *et al.*, 2002). It is not possible to consider all contaminants but short reviews are given below for a number of substances prioritised on the basis of both their intrinsic toxicities and concentrations detected in emissions from waste disposal operations. .

Data are presented for levels of these contaminants in air, food and water. Public water supplies are rigorously monitored at the supply point, consumer taps, service reservoirs and water treatment works and are required to meet health based water quality criteria. Therefore the potential for the consumer to be exposed to harmful levels of substances in drinking water is extremely low and most supplies contain levels well below the UK drinking water standards (Water Supply Regulations, 2000).

Since private water supplies are less frequently monitored breaches of the standards are possible if the supply becomes contaminated. However in the UK relatively few people obtain their drinking water from a private supply other than in rural areas.

Waste waters from incineration and other disposal options may be discharged directly to surface waters. However, such emissions will be subject to tighter controls on contaminant levels than for discharge to sewer. Even if these surface waters were then extracted for public supply, they will be subject to treatment processes designed to minimise contaminant levels and to routine monitoring for contaminant content. Even if trace levels of contaminants in surface waters were taken up by aquatic organisms, such organisms represent only a small fraction of typical human food intakes.

The final question that arises concerns complex mixtures and interactions between chemicals at low concentrations. Although there are examples of one chemical interacting with another to give rise to a multiplicative effect, i.e. synergism, in most cases the effects are either additive or there is no interaction or there may even be antagonism. This issue needs to be addressed on a case by case basis, although the development of tolerable intakes of substances such as dioxins and PCBs, which always occur as mixtures, already takes this into account.

The extent of the risk to health relates to the level of exposure, usually over time, since most although not all of the tolerable intakes determined by bodies such as WHO relate to long-term average exposure. The level of exposure is therefore important if the chemical is persistent and bioaccumulates. Short-term exceedences of the tolerable intake may not, therefore, constitute an excess risk to health.

A number of substances are of less concern for health than for their impact on sensory perception or because there is a significant public concern about them. Illustrative examples of these types of substances are also listed. Those such as the mercaptans, which may be produced in landfill, are volatile and give rise to a very unpleasant smell. Their impact is, therefore, more important in relation to psycho-social well being rather than toxicity. Other substances are less likely to give rise to exposure of the public through waste disposal. Pesticides are an example of such substances which are increasingly controlled both in the way they are used and in the way in which they are disposed of. In general where pesticides have given rise to concern this has been for drinking water, which is closely monitored and for which there are extremely stringent standards set and which are well below the health based guideline values for most pesticides.

# Arsenic

## Is it important?

Arsenic is a metalloid element. It is chemically reactive and can form a variety of inorganic and inorganic compounds in the trivalent (+3) to pentavalent (+5) valence states. Arsenic is widely distributed in the earth's crust and can be found naturally in drinking water. It is also widely used in industry.

Arsenic is a relatively common pollutant. Emissions from incinerators will contain small amounts of arsenic and arsenic is also present in the solid waste residue and wastewater discharges. Many materials sent to landfill (including solid waste residues from incineration) will also contain arsenic.

## Why is it important?

Acute exposure to high levels of arsenic can result in a number of serious health effects. Inhalation exposure can cause coughing, breathing difficulty, chest pain, and severe damage to the respiratory tract. Nasal perforations have also been noted following acute inhalation exposure. Ingestion can result severe gastrointestinal irritation and symptoms typically include vomiting, oesophageal and abdominal pain, bloody "rice water" diarrhoea and shock. Facial swelling, muscle cramps, cardiac abnormalities, anaemia, decreased white blood cell count, and enlargement of the liver have also been noted in acute ingestions (Meditext, 2002). These effects can be immediate or delayed in onset.

Nervous disturbances involving mainly sensory neuropathies similar to the clinical appearance of tetanus have been described as delayed effects to acute exposure.

However, acute toxicity resulting from environmental exposure is extremely uncommon, but chronic exposure to arsenic may result in undesirable toxic effects.

We are all exposed to arsenic every day with the main route of arsenic exposure being ingestion of food and water containing arsenic. Mean exposure to total arsenic in the diet in the UK is  $0.065 \text{ mg day}^{-1}$ , estimated for the general UK population (FSA, 2000a). The Joint FAO/WHO Expert Committee on Food Additives (JECFA) has established a Provisional Tolerable Weekly Intake of  $0.007 \text{ mg/kg bodyweight}$  (WHO, 2002a). Drinking water will be the major route of exposure where supplies are obtain from groundwater sources with elevated arsenic concentrations. The vast majority of supplies contain levels well below the UK drinking water standard of  $10 \text{ } \mu\text{g/l}$  (Water Supply Regulations, 2000).

Inhalation exposure from industrial emissions may be an important source of exposure. Mean total arsenic concentrations in ambient air from remote and rural areas range from  $0.02$  to  $4 \text{ ng/m}^3$  while in urban areas total concentrations typically range from  $3$  to about  $200 \text{ ng/m}^3$  (WHO, 2002a). However, emissions from incinerators are well regulated and ground-level concentrations arising from incinerations should only be a relatively small proportion of the existing background concentration.

Likewise, fugitive emissions from composting facilities could potentially be a source of exposure but it is unlikely that contaminant levels in ambient air around such facilities would be high enough to cause acute effects.

Arsenic in soil as a result of deposition, compost or sewage sludge may be a potential hazard but once within the soil environment, arsenic will typically become bound to clay and organic matter and will be effectively immobilized. Uptake by plants and leaching into water supplies is expected to be unlikely. Furthermore, the levels of these compounds should not be great enough to pose a health risk to the general public.

### **Possible effects**

As a result of monitoring and regulatory emission limits, it is extremely unlikely that the general population will be exposed to concentrations high enough to cause acute effects. The intake of arsenic from air will, typically, be only a minor portion of the total intake from all sources. The intake of arsenic (primarily inorganic) from water is also typically low for most persons (WHO, 2002a).

The major health effect of concern in relation to long-term ingestion or inhalation is cancer, with the skin and the lungs the main target organs. Inorganic arsenic compounds are classified by IARC as carcinogenic to humans (Group 1) with long-term ingestion of inorganic arsenic an established cause of skin cancer. Many epidemiological studies have demonstrated that communities exposed to elevated levels of arsenic in drinking water suffer elevated rates of skin cancer. A number of studies also suggest that ingestion can cause cancers of the lung, kidney and bladder. Evidence for cancer comes primarily from epidemiological studies where exposed populations were exposed to arsenic concentrations in drinking water of at least several hundred micrograms per litre. Few studies examine the cancer risk from exposure to lower concentrations.

Epidemiological studies strongly indicate a clear dose-response relationship between drinking water concentrations and the risk of skin cancer. Increased risks of lung and bladder cancer and of arsenic-associated skin lesions have been observed at drinking-water concentrations of less than 50 µg/l (WHO, 2001b). The World Health Organisation considers a drinking water concentration of 10 µg/l (the UK water quality standard) to be associated with an estimated excess lifetime risk of skin cancer of  $6 \times 10^{-4}$  (WHO, 1996a; WHO, 2001b). Routine monitoring of drinking water supplies in the UK makes the potential for exposure to such concentrations above the water quality standard highly unlikely.

Chronic ingestion can cause hyperpigmentation of the skin, particularly on the palms of the hands and the soles of the feet, anaemia, and cirrhosis of the liver. Arsenic compounds are also contact allergens and can cause papular eczema or follicular swelling and pustules, warts, and increased or decreased pigmentation that may develop into skin cancer. Skin lesions can be delayed in onset and can occur following exposure by any route.

The World Health Organisation does not recommend a safe level for inhalation exposure and estimates the lifetime risk of cancer at an air concentration of 1 µg/m<sup>3</sup> to be  $1.5 \times 10^{-3}$  (WHO, 2000a). However, emissions to atmosphere from waste disposal operations should only represent a small part of the overall background level of arsenic in ambient air.

# Cadmium

## Is it important?

Cadmium (Cd) is a soft, ductile, silver-white metal that has relatively low melting (320.9 °C) and boiling (765 °C) points and a relatively high vapour pressure. Cadmium is a relatively rare element and is not found in a pure state in the environment. Instead it is typically found associated with ores containing zinc, lead, and copper. It is widely used in industry, including as a component of Ni-Cd batteries, welding rods, control rods, plating, semiconductors, solar cells, dry film lubricants, and automotive paints.

In Europe, approximately 85–90% of total airborne cadmium emissions arise from anthropogenic sources, mainly from smelting and refining of nonferrous metals, fossil fuel combustion and municipal waste incineration (WHO, 2000a). Cadmium is emitted to the atmosphere predominantly as elemental cadmium and cadmium oxide and from some sources as cadmium sulfide (coal combustion and nonferrous metal production) or cadmium chloride (refuse incineration).

Cadmium is also found in the solid waste residues and wastewater discharges from incinerators and many materials sent to landfill (including solid waste residues from incineration) or composting will also contain sources of cadmium.

## Why is it important?

Cadmium highly toxic to animals and man at relatively low concentrations. Cadmium is a severe lung and gastrointestinal irritant that can be fatal by inhalation and ingestion (Meditext, 2002). The symptoms of acute poisoning after inhalation exposure may be delayed 12 to 36 hours and may include chest pain, cough (with bloody sputum), difficulty breathing, sore throat, ‘metal fume fever’ (shivering, sweating, body pains, headache) dizziness, irritability, weakness, nausea, vomiting, diarrhea, tracheobronchitis, pneumonitis and pulmonary edema (Meditext, 2002). Cadmium is also toxic by ingestion, with symptoms usually appearing in 15 to 30 minutes. These include abdominal pain, burning sensation, nausea, vomiting, salivation, muscle cramps, vertigo, shock, unconsciousness and convulsions (Hazardtext, 2002).

However, acute toxicity resulting from environmental exposure is extremely uncommon, but chronic exposure to cadmium may result in undesirable toxic effects. The most important issue is low level chronic exposure which will affect the kidney but the major exposure is through cigarette smoke.

Cigarette smoking can represent a route of substantial exposure, which may equal or exceed that from food. Inhalation exposure by people living close to a cadmium emission source may be a significant source of local exposure but typically will still be an order of magnitude less than exposure via food.

Cadmium is present at low concentrations in most foods, with foods that are consumed in larger quantities making the greatest contributions to dietary exposure of the general population (e.g. the Potatoes and Bread groups (both 25 per cent) made the greatest contributions to the population dietary exposure estimated from the 1997 Total Diet Study) (FSA, 2000a).

Drinking-water typically contains very low concentrations of cadmium and cadmium intake from drinking-water based on a daily consumption of 2 litres is usually less than 1 µg (WHO,

1992; WHO, 1996a). Most supplies contain levels well below the UK drinking water standard of 5 µg/l (Water Supply Regulations, 2000).

Cadmium can contaminate the environment through the application of phosphate fertilisers and sewage sludge to soils and, particularly, through the combustion of coal and fossil fuels and the incineration of municipal waste. Cadmium is relatively mobile in soils and can be taken up by plants but, except at high concentrations, phytotoxicity by cadmium is rarely reported. This may result in the consumption of cadmium rich plant material, without the plant displaying any obvious signs of cadmium stress.

### **Possible effects**

As a result of monitoring and regulatory emission limits, it is extremely unlikely that the general population will be exposed to concentrations high enough to cause acute effects. However, there is concern over long-term exposure to low levels of cadmium, particularly via the foodchain.

The kidney is the critical organ after long-term occupational or environmental exposure to cadmium. Chronic exposure (by inhalation or ingestion) results in kidney damage, as well as gastrointestinal symptoms, loss of sense of smell, nasal discharge, nose and throat irritation, lack of appetite, weight loss, nausea, tooth discoloration, bone structure defects, liver damage, anaemia, pulmonary emphysema, chronic bronchitis, bronchopneumonia and death (Meditext, 2002). Chronic environmental cadmium exposure was the aetiology of Itai-Itai (“ouch-ouch”) disease in Japan, which was associated with renal tubular dysfunction, osteomalacia, anaemia, generalized pain and death.

IARC has classified cadmium and cadmium compounds as Group 1 human carcinogens (by inhalation), having concluded that there was sufficient evidence of cadmium being carcinogenic to humans and animals. However, there is no evidence of carcinogenicity by the oral route (WHO, 1996a).

The US Environmental Protection Agency Integrated Risk Information System (IRIS) has derived an inhalation unit risk for cancer of  $1.8 \times 10^{-3}$  per µg/m<sup>3</sup> (IRIS, 2002). However, emissions to atmosphere from waste disposal operations should only represent a small part of the overall background level of cadmium in ambient air.

IRIS sets an oral reference dose (RfD) (based on the assumption that a threshold exists for certain effects) of 3 µg/kg/day (food) and 0.5 µg/kg/day (water) (IRIS, 2002). The Joint FAO/WHO Expert Committee on Food Additives (JECFA) has established a Provisional Tolerable Weekly Intake of 0.015 mg/kg bodyweight (WHO, 2002a). Average daily intake from food in most countries is probably at the lower end of the range of 10-25 µg (WHO, 1992) and in the UK the estimated general population exposure to cadmium through the diet is about 12 µg /day (approximately 0.17µg/kg/day for a 70 kg adult) (FSA, 2000a). The recent recommendation by the World Health Organization of a guideline of 5 ng/m<sup>3</sup> specifically to prevent any further increase of cadmium in agricultural soils, which could increase the dietary intake of future generations, also reduces the potential for exposure to potentially harmful concentrations (WHO, 2000a). Rigorous monitoring of drinking water supplies makes the potential for exposure above the oral reference dose highly unlikely.

# Chromium

## Is it important?

Chromium can exist in a range of oxidation states with the most stable forms in the environment being the trivalent (+3) salts and the hexavalent (+6) chromates. It is widely used in industry.

Emissions from incinerators will contain small amounts of chromium compounds and chromium is also present in the solid waste residue and wastewater discharges. Many materials sent to landfill (including solid waste residues from incineration) will contain sources of chromium.

## Why is it important?

The toxicity of chromium is dependent on the ability of the organism to absorb it. Chromium is mainly absorbed by inhalation but also via the skin and gastrointestinal tract. Hexavalent compounds are efficiently absorbed while trivalent chromium compounds tend to be insoluble. The greater toxicity of hexavalent chromium is therefore attributed to its stronger oxidising power and higher membrane transport (Katz and Salem, 1993).

Hexavalent chromium is corrosive by ingestion, inhalation and dermal contact and tissue damage, irritation and allergic reactions are all well documented. Acute toxicity can result in irritation causing wheeze and cough and in severe cases chest pain and fever. Hexavalent chromium can cause chronic respiratory tract irritation and can result in chronic ulceration of the nasal septum, and chronic rhinitis and laryngitis.

However, acute toxicity resulting from environmental exposure is extremely uncommon, but chronic exposure to chromium may result in undesirable toxic effects.

Chromium can contaminate the environment through waste disposal practices, for example the airborne deposition of chromium from incinerator emissions may contaminate crops and the soil environment. Similarly, chromium levels in sewage sludges, derived from solid waste residues from incineration or in mature compost may also be elevated (Deportes *et al.*, 1995). However, once within the soil environment most metals, including chromium, become bound to clay and organic matter and typically will be effectively immobilized. Uptake by plants and leaching into water supplies is also expected to be unlikely, although hexavalent chromium is more bioavailable than trivalent.

The mean dietary exposure to chromium in the UK is estimated to be 0.1 mg/day (FSA, 2000a). Most of the chromium present in food is in the trivalent form which is an essential nutrient. The hexavalent form is not normally found in food.

Most supplies contain levels well below the UK drinking water standard of 50 µg/l (Water Supply Regulations, 2000).

## Possible effects

As a result of monitoring and regulatory emission limits, it is extremely unlikely that the general population will be exposed to concentrations high enough to cause acute effects. However, there is concern over the long-term effects of low level exposure to chromium.



There is sufficient evidence for human carcinogenicity of hexavalent chromium compounds commonly used in chromate production, pigment production and plating (Richardson and Gangolli, 1994). Hexavalent chromium is classified IARC group 1 and occupational exposure to hexavalent chromium is strongly associated with respiratory tract cancer (Von Burg and Liu, 1993). Lung cancer is the health effect of concern at environmental exposure levels.

Animal tests have shown that chromium can be transported to the foetus in the uterus. Chromium concentrations in the blood of newborn babies tend to be higher than those found later in life.

The World Health Organisation does not recommend a safe level for inhalation exposure and estimates the lifetime risk of cancer at an air concentration of  $1 \mu\text{g}/\text{m}^3$  to be  $4 \times 10^{-2}$  (WHO, 2000a). The USEPA IRIS reference concentration for chronic inhalation exposure to chromic acid mists and dissolved hexavalent chromium aerosols is  $0.008 \mu\text{g}/\text{m}^3$  and for hexavalent chromium particulates it is  $0.1 \mu\text{g}/\text{m}^3$  (IRIS, 2002).

However, strict regulations should ensure that emissions to atmosphere from waste disposal operations should only represent a small part of the overall background level of chromium in ambient air. The World Health Organisation stated that whilst exposure to chromium through inhalation and skin contact could potentially pose health problems for the general population, they concluded that there was no reason, to be concerned that chromium in the air presents a health problem, except under conditions of industrial exposure (WHO, 1988).

# Lead

## Is it important?

Lead has been found in the environment as a consequence of human activity since pre-Roman times. It was used for a wide variety of purposes as a metal because of its great malleability. Lead can be found naturally in some waters as a consequence of leaching by acid waters but most lead in the environment arises as a consequence of human activity. This activity continued from pre-Roman times to the present day when considerable efforts have been made to reduce exposure from lead.

Emissions from incinerators will contain small amounts of lead and lead will also be present in the solid waste residue and wastewater discharges. Many materials sent to landfill (including solid waste residues from incineration) or composting will also contain sources of lead.

Waste treatment and disposal is estimated to contribute around 7% of the total airborne lead emissions in the UK (approximately 105 tonnes) (EPAQS, 1998).

## Why is it important?

Lead is a cumulative toxin that affects a wide range of biochemical processes in the body. Pregnant women, the foetus, the new-born, infants and children up to the age of six are most susceptible to lead poisoning. The major effects of lead include anaemia and effects on the nervous, reproductive, cardiovascular, hepatic, renal, endocrinal and gastrointestinal systems. Acute poisoning from a single exposure is rare.

Exposure to lead can occur through food, water and dust. However, exposure has considerably reduced over the past 10 to 20 years with the introduction of unleaded fuel, new standards for lead in drinking water from lead plumbing, withdrawal of lead pigments in paint and withdrawal of lead solders for copper drinking water pipes and cans for food. There are extensive surveys of blood lead levels available for populations in England (IEH 1998) and these show that blood lead levels have fallen by a factor of between 2.6 and 3.0 between 1984-87 and 1995. Mean blood lead concentrations are now less than 3.7 µg/100 ml with the lowest concentrations in children between 11 and 15 years. Concentrations increase with age, which may in part be due to historically higher exposure stored in bone and because concentrations are gradually released over time combined with higher exposure from sources such as cigarette smoke. Individuals with high exposure can be identified and these may be due to lead in water considerably above the standard or from dust in houses, often arising as a consequence of sanding old paint that contains lead pigments.

Food is considered to be the main source of lead intake for most people with beverages, vegetables and milk being the main food groups containing lead. Lead may enter food through the deposition of dust and rain, containing the metal, on crops. In root crops, the contribution of deposited lead to the lead content of the edible portion of the plant is probably slight, but in leafy crops and cereals it may be more important (EPAQS, 1998).

Most supplies contain levels well below the UK drinking water standard of 50 µg/l (Water Supply Regulations, 2000).

Waste waters from incineration and other disposal options may be discharged directly to surface waters. However, such emissions will be subject to tighter controls on contaminant levels than for discharge to sewer. Even if these surface waters were then extracted for public supply, they will be subject to treatment processes designed to minimise contaminant levels and to routine monitoring for contaminant content. Even if trace levels of contaminants in surface waters were taken up by aquatic organisms, such organisms represent only a small fraction of typical human food intakes.

Likewise, fugitive emissions from composting facilities could potentially be a source of exposure but it is unlikely that contaminant levels in ambient air around such facilities would be high enough to cause acute effects.

Lead in soil as a result of deposition, compost or sewage sludge may be a potential hazard but once within the soil environment, lead will typically become bound to clay and organic matter and will be effectively immobilized. Uptake by plants and leaching into water supplies is expected to be slight and highly variable between plant species and soil types. Furthermore, the levels of these compounds should not be great enough to pose a health risk to the general public.

### **Possible effects**

Lead has been known to cause severe effects on the central nervous system at high doses for many centuries but more recently it has been recognised that more subtle effects can occur at much lower intakes. Lead absorption varies with different circumstances. In particular it is affected by a wide range of other substances in the diet including calcium, phosphate, selenium and zinc, which reduce absorption. Absorption is higher in individuals with iron deficiency. Lead absorption is also affected by its chemical form in the environment. Although lead is rapidly distributed to soft tissues, there is a slower redistribution to bone. The half-life in bone is much longer than the 28-36 days for soft tissues and blood. Lead does cross the placenta to reach the foetus.

Lead can inhibit a number of enzymes, for example inhibition of delta-aminolaevulinic acid dehydratase and dihydrobiopterin reductase is observed at relatively low blood lead levels but the biological significance of such small changes is uncertain. The best studied endpoints relate to neurological effects in children, particularly on IQ. However, the nature of the studies and the potential for confounding by other factors means that it is difficult to be sure of exact causal relationships with lead exposure. Nevertheless WHO concluded that “the size of the apparent IQ effect, as assessed at 4 years and above, is a deficit between 0 and 5 points (on a scale with a standard deviation of 15) for each 10 µg/100 ml increment in blood lead level, with a likely apparent effect size of between 1 and 3 points.” They also conclude that “below the blood lead range of 10-15 µg/100ml, the effects of confounding variables and limits in the precision of analytical and psychometric measurements increase the uncertainty attached to any estimate of effect. However, there is some evidence of an association below this range.” These effects may persist for some time after the cessation of lead exposure (WHO, 1995). At the very low blood lead levels now encountered adverse effects would be very difficult to separate from social, genetic and educational effects. WHO (2000b) have also suggested the following relationship between IQ deficits and blood lead levels.

**Table A.3 Net decrease in IQ associated with blood lead concentration (After WHO, 2000b)**

Concentration of lead in blood ( $\mu\text{g}/100\text{ ml}$ )	Median IQ decrement (95% confidence interval)
5	0.4 (0.0-1.5)
10	1.7 (0.5-3.1)
15	3.4 (1.1-5.0)
20	5.5 (1.6-6.9)

Lead exposure has also been associated with a small increase in blood pressure, particularly in men (WHO, 1995; IEH, 1998). This increase is small and may be due to confounding variables rather than a causal relationship with lead exposure. However, there is sufficient evidence for this to be considered seriously in assessing the possible health effects of environmental lead exposure.

There are also some data to suggest adverse pregnancy outcomes at blood lead levels of about 15  $\mu\text{g}/100\text{ ml}$  and above. Damage to kidneys occurs at high levels of exposure, about 60  $\mu\text{g}/100\text{ ml}$  but there is some evidence of more subtle effects at lower exposures.

WHO have established a provisional tolerable weekly intake for lead of 25  $\mu\text{g}/\text{kg}$  body weight for infants and children. This value was derived from a study in infants and is the intake at which no accumulation is expected to occur (WHO, 1993c).

The UK Expert Panel on Air Quality Standards (EPAQS) recommends an Air Quality Standard for lead in the United Kingdom of 0.25  $\mu\text{g}/\text{m}^3$  measured as an annual average. This recommendation is intended to protect young children, the group regarded by the Panel as those most vulnerable to impairment of brain function. However, emissions to atmosphere from waste disposal operations should only represent a small part of the overall background level of lead in ambient air.

# Mercury

## Is it important?

Mercury is a naturally occurring metal, which has several forms. Mercury in its metallic form is a heavy, shiny, silver-white, odourless liquid at room temperature. When heated it forms a colourless and odourless gas. Its vapour pressure is sufficiently high to yield hazardous concentrations of vapour at temperatures normally encountered both indoors and outdoors under most climatic conditions

Mercury is found in the solid waste residues and wastewater discharges from incinerators and many materials sent to landfill (including solid waste residues from incineration) or composting will also contain sources of mercury. It is used in thermometers, barometers, sphygmomanometers, dental amalgams, electrical appliances (lamps, arc rectifiers, mercury cells) and as the cathode in the electrolytic production of chlorine and caustic soda.

## Why is it important?

Mercury is highly toxic to animals and man at relatively low concentrations and will bioaccumulate in food chains, although the diet is not a significant source of exposure to elemental mercury. The general population dietary exposure to mercury estimated from the 1997 Total Diet Study was 0.003 mg/day with fish contributing 33 per cent to this total (FSA, 2000a).

Because of its low vapour pressure, inhalation of mercury vapour is the most common route of exposure. Approximately 80% of inhaled mercury vapour is retained and retention occurs almost entirely in the alveoli (WHO, 1991b). Mercury may also be absorbed through the skin, although studies on human volunteers indicate that uptake of metallic mercury vapour via the skin is about 1% of uptake by inhalation (WHO, 1991b). Liquid metallic mercury is poorly absorbed by ingestion.

Vapour inhalation can cause coughing, chest pains, dyspnoea, nausea, vomiting and haemoptysis (coughing up of blood), diarrhoea and general malaise. Exposure to high concentrations causes severe respiratory damage including corrosive bronchitis and interstitial pneumonitis and death from respiratory insufficiency. Other symptoms, which may appear within a few hours of vapour exposure, include weakness, chills, metallic taste and visual disturbances (Meditext, 2002).

Delayed effects from acute exposure include central nervous system effects and renal damage, gingivitis, and stomatitis (Meditext, 2002). Psychotic reactions characterized by delirium, hallucinations, and suicidal tendency have been reported. Both metallic mercury vapour and mercury compounds have given rise to contact dermatitis (WHO, 1991b).

## Possible effects

As a result of monitoring and regulatory emission limits, it is extremely unlikely that the general population will be exposed to concentrations high enough to cause acute effects. However, chronic exposure can have health effects. Symptoms of chronic exposure include mouth and gum inflammation, excess salivation, loose teeth, kidney damage, muscle tremors, jerky gait, and limb spasms. Chronic effects can include central nervous system effects such as personality changes, hallucinations, delirium, insomnia, decreased appetite, irritability, headache and memory loss (Meditext, 2002; WHO, 1991b).

Chronic occupational exposure to mercury vapour gives rise to neurological effects characterised by a fine high-frequency intention tremor (usually involving the hands) and neurobehavioural impairment. Long-term, low-level exposure has been associated with less pronounced neurological symptoms (WHO, 1991b).

Both the UK drinking water standard and the WHO guideline for drinking-water is 1 µg/litre (Water Supply Regulations, 2000; WHO, 1996b). The vast majority of supplies contain levels well below the UK drinking water standard of 10 µg/l (Water Supply Regulations, 2000).

There is no UK standard for environmental levels of mercury vapour, but the United States Environmental Protection Agency's IRIS reference concentration for chronic inhalation exposure is 0.3 µg/m<sup>3</sup>. However, emissions to atmosphere from waste disposal operations should only represent a small part of the overall background level of mercury in ambient air.

# Nickel

## Is it important?

Nickel is widely found in trace levels in the environment. It is used widely as a metal, primarily in alloys that are used for their hardness, strength and resistance to both corrosion and heat. It is important in stainless steel and other non-ferrous alloys, but it is also used in nickel-cadmium batteries, coins and jewellery, and in electroplating. In addition it is used in some pigments and in welding products. WHO (1991a) estimated that 8% of nickel manufactured is used in household appliances.

Nickel is also found in the solid waste residues and wastewater discharges from incinerators and many materials sent to landfill (including solid waste residues from incineration) or composting will also contain sources of nickel.

## Why is it important?

Nickel can contaminate the environment through waste disposal practices, for example the deposition of nickel from incinerator emissions may contaminate crops and the soil environment. Similarly, nickel levels in sewage sludges derived from solid waste residues from incineration or in mature compost may also be elevated. However, once within the soil environment most metals, including nickel, become bound to clay and organic matter and will be effectively immobilized. Therefore uptake by plants and leaching into water supplies is expected to be unlikely.

In terms of human health effects, nickel carbonyl is the most acutely toxic nickel compound. The effects of acute nickel carbonyl poisoning include frontal headache, vertigo, nausea, vomiting, insomnia, and irritability, followed by pulmonary symptoms similar to those of a viral pneumonia (WHO, 1991a). Pathological pulmonary lesions include haemorrhage, oedema, and cellular derangement. Liver, kidneys, adrenal glands, spleen, and brain are also affected. Cases of nickel poisoning have also been reported in patients dialysed with nickel-contaminated dialysate and in electroplaters who accidentally ingested water contaminated with nickel sulfate and nickel chloride (WHO, 1991a).

However, acute toxicity resulting from environmental exposure is extremely uncommon, but chronic exposure to nickel may result in undesirable toxic effects. Chronic effects, such as rhinitis, sinusitis, nasal septal perforations, and asthma, have been reported in nickel refinery and nickel plating workers (WHO, 1991a).

The primary source of non-occupational exposure to nickel appears to be through food. There is a large variation in the nickel content of foods with the norm being in the range 0.01 to 0.1 mg/kg. However, there is generally more nickel in whole meal products and in nuts and beans. Exposure of vegetarians in the UK measured through a duplicate diet study showed that the mean level of intake of nickel was 0.17 mg/day while the mean exposure of the general population was 0.13 mg/day (FSA, 2000a). It has been suggested that nickel may leach from stainless steel cooking pots into food but there are inconsistencies in the data. It may be that the extent of leaching will depend on the quality of the stainless steel.

Levels in ambient air are usually very low and emissions from incinerators should only contribute a very small proportion of the existing background concentration. Cigarette smoke

may be a significant source in indoor air. Mainstream cigarette smoke from one cigarette is reported to contain between 0.04 and 0.58 µg nickel.

Levels in drinking water in Europe are reported to be less than 10 µg/l but this will depend to an extent on circumstances. Where water is in contact with nickel-plated fittings for an extended period, concentrations in first draw water may be much higher. There are also data to indicate that some electric kettles in which the elements are electroplated may give rise to concentrations in boiled water of 100 to 400 µg/l (WHO, 1991a).

Most supplies contain levels well below the UK drinking water standard of 50 µg/l (Water Supply Regulations, 2000).

### **Possible effects?**

As a result of monitoring and regulatory emission limits, it is extremely unlikely that the general population will be exposed to concentrations high enough to cause acute effects. However, some nickel compounds are considered to be possible human carcinogens, particularly in some occupational situations and IARC have classified inhaled nickel compounds as carcinogenic to humans. However, there appears to be little or no carcinogenic risk associated with current occupational exposure levels (WHO, 1991a). There is a lack of evidence regarding the possibility of carcinogenicity by the oral route. Nickel does not appear to be mutagenic but it may cause chromosome aberrations following exposure to very high levels.

With regards to dietary exposure, no tolerable intakes for nickel have been established, although the WHO have established a provisional guideline value for drinking water of 20 µg/l based on a tolerable daily intake of 5 mg/kg body weight and an allocation of 10% of that value to water (WHO, 1993b, 1998b). However, the levels of nickel associated with emissions from waste management should not be great enough to pose a health risk to the general public.



# Dioxins

## Are they important?

The polychlorinated dibenzodioxins (PCDDs) are a group of chlorinated organic compounds that contain at least four chlorine atoms. The term dioxins is usually used to also cover the related polychlorinated dibenzofurans (PCDFs) and PCBs with dioxin like properties.

Dioxins are found as trace contaminants in some chlorinated industrial and agricultural chemicals. They are also formed by combustion of some wastes, by burning fossil fuel and in forest fires. Dioxins are a family of substances with similar properties and they are ubiquitous in the environment, although at very low levels. Dioxins have low volatility and water solubility, a high lipophilicity, an extremely long environmental half-life and can accumulate in biological tissues leading to bioconcentration in the food-chain. They will tend to adsorb to particles, soils and sediment. For example, airborne dioxins will attach to particles and will be deposited from the air with the particles. Analysis of archived soil samples from a semi-rural area in the south east of England, known to have only been contaminated by deposition from air, showed that dioxins were present in all of the samples, which were first collected in 1846 (WHO, 1989).

Brominated dibenzodioxins and dibenzofurans may also be formed although when an excess of chlorine is available the chlorinated congeners will be formed preferentially. The brominated congeners are similarly persistent to the chlorinated congeners. Although data are more limited with regard to the brominated compounds the data that are available indicate that they are present at very much lower concentrations than the chlorinated compounds (WHO, 1998c).

Data on dioxins show that levels are higher in cities where the combustion sources are more numerous. Environmental levels have fallen, more recently, in response to the decrease in domestic use of fossil fuels (particularly coal), better methods of chemical manufacture, more modern internal combustion engines, reduced burning of waste in open bonfires and controls on incinerator emissions. However, it is almost certainly impossible to achieve zero emissions of dioxins to the environment.

## Why are they important?

PCDDs, PCDFs, and the dioxin-like PCBs (non-ortho and mono-ortho substituted PCBs) exert a number of biochemical and toxicological effects mediated through the aryl hydrocarbon receptor (Ah receptor). Different congeners show widely varying toxicity, the 2,3,7,8-TCDD and 1,2,3,7,8-PeCDD congeners are considered to be the most toxic while OCDD is considered to be the least toxic. The PCDFs mirror the PCDDs in terms of varying toxicity. Since the PCDDs and PCDFs always occur in a mixture with varying congeners and proportions of different congeners, the system of toxic equivalents (TEQ) was established compared against 2,3,7,8-TCDD. These include a number of PCBs that display dioxin-like properties and have recently been considered by WHO (WHO JEFCA, 2001) and the Committee on Toxicity of Chemicals in Consumer Products and the Environment in the UK (COT, 2001).

The effects of acute exposure are typically dermal, characterised by the disfiguring skin condition, chloracne, but data from occupational or accidental exposures suggest other

symptoms such as liver fibrosis, nausea, vomiting, headaches, severe muscular aches and pains, fatigue, loss of appetite and weight loss. However, acute toxicity resulting from environmental exposure is extremely uncommon, but there are concerns that chronic exposure to low levels of dioxins may have serious consequences.

The principal pathway for exposure to dioxin-like chemicals is food. Food contamination occurs mainly through the contamination of plants by airborne dioxins, which because of their hydrophobicity and extreme persistence accumulate in the lipid reservoirs of animals consuming those plants. Actual plant uptake of dioxins from soil is minimal because dioxins become strongly bound to soil, which greatly reduces their bioavailability. Contamination of the foodchain may also occur when animals consume soil containing dioxins during feeding. This may result in increases in the levels in milk, meat and eggs over the normal background levels.

Approximately 95% of human exposure is estimated to occur through the diet with the consumption of fats and fatty foods being the predominant sources. Exposure to dioxins in drinking water is considered negligible because of the hydrophobic properties of dioxin-like chemicals. Likewise, inhalation exposure is low owing to the low vapour pressures of these contaminants.

The presence of dioxins in an area does not automatically mean that humans will be exposed and even if local contamination occurs, it is extremely unlikely to result in an increase in dietary exposure because of the limited contribution of locally grown food to the overall diet of the general public. An additional route of exposure in some circumstances, for small children in particular, is the ingestion of soil and dust through hand to mouth transfer.

Estimates of intake from dietary and blood measurements show that there has been a general decline in exposure in line with the reduction of emissions to the environment. This has also been helped by the change in diet to reduce saturated fat intake. The average intake of dioxins in the UK has fallen from 7.2 pg and 18 pg TEQ/kg body weight (bw)/day for adults and toddlers in 1982 to 1.8 and 4.6 pg TEQ/kg bw/day in 1997. With regard to extreme intakes, these have fallen from 13 and 28 pg TEQ/kg bw/day per day to 3.1 and 7.2 pg TEQ/kg bw/day (FSA, 2000b; COT, 2001). This situation is reflected in decreasing body burdens with many countries reporting decreases in dioxins concentrations in human milk.

There are still uncontrolled sources such as fires, including forest fires, but the indications for the UK are that emissions to the atmosphere have fallen by ca 70% over the last decade. In particular, there has been a marked decline in the relative contribution to overall emissions from municipal waste incinerators. This has been reflected in studies of dietary exposure that show there has been a fall in exposure to PCDDs, PCDFs and dioxin-like PCBs in the UK by about 75% over the past 20 years or so (FSA, 2001).

## **Possible effects**

The dioxins have been shown to cause a number of potential effects in experimental animals, some at extremely low doses. Among these are effects on the immune system and effects on the developing foetus, including effects on cognitive development, malformations and cancer. They are considered to possess endocrine disrupting properties and this is believed to be the primary mechanism of toxicity. In particular they are known to effect the Ah receptor, although this mechanism has proved much more complex than first thought, with significant potential for interspecies and inter-individual variation. In human exposure studies the only proven effect is that of chloracne. Although there have been strong suggestions of a range of

effects in epidemiological studies, mostly of highly exposed populations, there are considerable difficulties in determining exposure levels and eliminating confounding factors.

However, the potential effects of long-term exposure to low-levels of dioxins in the environment is a cause for concern. The most toxic form, TCDD, is considered a probable human carcinogen and has been linked with soft tissue sarcomas. There is much research on possible effects on the immune and hormonal systems and thyroid function in regard to background exposure, especially in relation to health effects on infants to dioxin exposure via breast milk and in utero exposure.

The most sensitive endpoint in animal studies is considered to be that of effects on sperm quality of the male rat foetus. It is considered that there is a threshold to the effects of dioxins and tolerable levels have been established on this basis (WHO JECFA 2001, SCF 2001, COT 2001).

The provisional tolerable monthly intake (PTMI) proposed by WHO is 70 pg TEQ/kg bw and in the UK COT have recommended that a provisional tolerable daily intake of 2 pg TEQ/kg bw derived on a similar basis to the WHO figure be adopted. The use of a PTMI by WHO reflects the long-term nature of the toxicity of dioxins and the concept of an average exposure over time. Exceeding the tolerable intake will not necessarily give rise to any health effects but the margin of safety will be gradually reduced. This is particularly so with short-term exceedences of the tolerable intake.

The PBDDs and PBDFs appear to possess similar toxic properties to their chlorinated equivalents, although they appear to be of slightly lower toxicity. There are, however, far fewer data on the brominated compounds.

# Polychlorinated Biphenyls (PCBs)

## Are they important?

Polychlorinated biphenyls are based on the structure  $C_{12}H_nCl_n$  where  $n$  is 1-10 atoms of chlorine. They were used widely in electrical equipment such as transformers and capacitors but manufacture ceased in the 1970s and they were gradually phased out. They are now banned from use but continue to be introduced into the environment in electrical waste, although significant controls have been introduced in most countries. They always occur in mixtures and are frequently associated with chlorinated dibenzodioxins and dibenzofurans. There are 209 theoretical congeners but in practice only 130 are found in commercial PCB mixtures. All of the PCBs are highly lipophilic and of low water solubility. They can be very persistent in the environment although the persistence depends on the degree of chlorination. Most PCBs adsorb to particulate matter very readily and are only slowly desorbed. However, the extent of adsorption does depend on the extent of chlorination and the lower chlorinated congeners adsorb less strongly.

There is a potential for exposure of individuals living near sources of PCBs from dust and soil to which PCBs may be adsorbed. Since they are also of varying volatility, there is also potential for deposition in particles washed from the atmosphere by rainfall.

## Why are they important?

PCBs are found associated with a number of wastes and their persistence along with the potential to bioaccumulate means that they are of potential concern. In addition there is considerable public disquiet regarding PCBs, even though food is normally considered the greatest source of exposure although levels in the diet have fallen by about 75% over the past 20 years (FSA,2001). The concentrations in human milk are from 0.5 to 1.5 mg/kg fat and the dietary intake can be up to 100  $\mu\text{g}/\text{week}$  (WHO, 1993a).

## Possible Effects

PCBs are found widely in the environment and are present at concentrations of between 0.001 to 0.24  $\mu\text{g}/\text{l}$  in precipitation (WHO, 1993a). Because of the differences in behaviour of the different congeners, there is redistribution in the environment of the congeners found in commercial mixtures

The assessment of health effects in humans as a consequence of exposure to PCBs is complicated by the presence of differing congeners of differing toxicity and the presence of PCDDs and PCDFs. In addition some PCBs are included in the risk assessment of PCDDs as dioxin like PCBs. These are included in the WHO Toxicity Equivalents for dioxins, and consist of 4 “non-ortho” PCBs and 8 “mono-ortho” PCBs. The majority of these compounds are significantly less active than 2,3,7,8-tetrachlorodibenzodioxin, which is the benchmark against which the others are measured. However, 3,3',4,4',5-pentachlorobiphenyl and 3,3',4,4',5,5'-hexachlorobiphenyl are much more active than the others (WHO JECFA, 2001).

The biological half-life also varies significantly between congeners. The higher chlorinated congeners have a much longer biological half-life and show bioconcentration factors of up to about 70,000. In terms of health effects from PCBs, there is some uncertainty. However, there is evidence from animal studies that PCBs can cause suppression of the immune system in

mammals. The doses at which this is likely to occur in humans is uncertain (WHO, 1993a). PCBs do not appear to be teratogenic but Rhesus monkeys given 0.03 mg/kg body weight per day of the commercial PCB mixture Aroclor 1016, showed reduced birthweight in the offspring. At a dose of 0.01 mg/kg body weight, hyperpigmentation of the skin was observed (WHO, 1993a).

PCBs do not appear to be genotoxic and there is considerable uncertainty over their possible carcinogenicity to man, although they have been shown to increase liver tumours in rodents (WHO, 1993a).

Some PCBs have been shown to possess weak estrogenic activity *in vitro* and the possibility of their causing endocrine disruption in man is being investigated. The European Scientific Committee on Toxicity and Ecotoxicity (1999) have indicated that the evidence for actual endocrine disruption in humans by chemicals in the environment remains equivocal. In laboratory animals studies there have been indications of effects on sex hormones, thyroid and corticosteroids. However, it must be emphasised that the doses used were considerably in excess of environmental exposure.

The determination of possible effects on human health as a consequence of exposure to PCBs remains difficult because of concurrent exposure to other persistent organochlorine compounds that are of greater concern.

# Polycyclic Aromatic Hydrocarbons (PAHs)

## Are they important?

Polycyclic aromatic hydrocarbons (PAHs) are ubiquitous in the environment and are produced by high-temperature reactions such as incomplete combustion and pyrolysis of fossil fuels and other organic materials. PAHs contain only carbon and hydrogen, and consist of two or more fused benzene rings in linear, angular or cluster arrangements. The broader class of polycyclic aromatic compounds (PACs) incorporates a range of substituent groups and/or heteroatoms (N, O, S) in the ring structure. It has been established that the lower molecular weight PAH species are present in air mainly as vapours, with higher molecular weight species being physically adsorbed on particulate surfaces. The greater fraction of particle-associated PAH is associated within the respirable particle size range (Harrison *et al.*, 1996).

Natural sources of PAH include forest fires, biosynthesis of algae, bacteria and plants and synthesis from degraded biological matter (e.g. fossil fuels) (Harrison *et al.*, 1996). Anthropogenic sources are the major contributors of the more hazardous PAH species and principally include motor vehicles, power generation, refuse incineration and coke manufacture. However, little data exist on the exact nature of PAH emissions from waste incinerations, particularly from modern plant operating under strict regulatory regimes. Incineration is estimated to contribute less than 0.01% of the total airborne (particulate and vapour) PAH emissions in the UK (EPAQS, 1999).

Since PAHs are formed during incomplete combustion, materials sent to landfill or composting will contain negligible PAH.

## Why are they important?

Many PAHs are known or suspected carcinogens and due to their widespread presence and persistence in the urban environment, human exposure is inevitable. Exposure occurs principally by direct inhalation of polluted air or tobacco smoke, ingestion of contaminated food and water, or by dermal contact with soot, tar and oils. PAHs generally partition into lipid-rich tissues, and their metabolites can be found in most tissues. In general, PAHs are more hydrophobic, and consequentially more persistent in the environment, as molecular weight increases.

Raw food does not normally contain high levels of PAH, but they are formed by processing, roasting, baking, or frying (WHO, 1998d). Cooking meat or other food at high temperatures, which happens during grilling or charring, increases the amount of PAHs in the food. Vegetables may be contaminated by the deposition of airborne particles or by growth in contaminated soil. The levels of individual PAH in meat, fish, dairy products, vegetables and fruits, cereals and their products, sweets, beverages, and animal and vegetable fats and oils are typically within the range 0.01-10 µg/kg (WHO, 1998d).

PAHs are absorbed through the pulmonary tract, the gastrointestinal tract, and the skin (WHO, 1998d).

## Possible effects

Typically humans are exposed to mixtures of PAHs. Since individual PAHs have differing toxicities this makes any evaluation of the health effects associated with exposure difficult. PAHs have a low order of acute toxicity in humans but irritation of the eyes and respiratory tract have been reported after chronic exposure to PAH in coal tar (Meditext, 2001). Cancer is the most significant PAH toxicity endpoint.

Occupational studies have demonstrated that exposure to high concentrations of PAH mixtures can result in an increased incidence of tumours of the lung, skin and possibly the bladder and other sites (EPAQS, 1999). Animal studies have confirmed such findings and have demonstrated that individual PAHs vary in their carcinogenic activity and, for several compounds, their capacity to induce tumours is uncertain or unknown (EPAQS, 1999). At present a total of seven PAHs are considered to be possibly carcinogenic to humans. The IARC classifies benzo[a]pyrene, benzo[a]anthracene and dibenzo[ah]anthracene as 'probably carcinogenic to humans' (Group 2A) and benzo[b]fluoranthene, benzo[k]fluoranthene, indeno[123cd]pyrene and chrysene as 'possibly carcinogenic to humans (Group 2B). At present other PAHs are classified at Group 3 'not classifiable'.

Tobacco smoking is the most important single factor in the induction of lung tumours and also for increased incidences of tumours of the urinary bladder, renal pelvis, mouth, pharynx, larynx, and oesophagus (WHO, 1998d), although this is not due solely to the presence of PAHs. The contribution of PAH in the diet to the development of human cancer is not considered to be high (WHO, 1998d).

Inhalation exposure can lead to lung cancer. Taking benzo[a]pyrene as a marker for the total mixture of polycyclic aromatic hydrocarbons in the UK, the Expert Panel on Air Quality Standards (EPAQS) recommend an air quality standard of  $0.25\text{ng/m}^3$  B[a]P as an annual average (EPAQS, 1999). This recommendation is intended to reduce any risk to the population of the UK from exposure to PAHs to one, which EPAQS believes would be so small as to be undetectable.

The World Health Organisation estimates the lifetime risk of lung cancer at an air concentration of  $1\ \mu\text{g/m}^3$  to be  $9 \times 10^{-2}$  (WHOROE, 2000). Strict regulatory limits on modern incinerators should ensure that emissions only represent a small part of the overall background level of PAH in ambient air.

# Pesticides

## Are they important?

There is a wide range of pesticides that are used for controlling pests in agriculture, animal husbandry, public health, management of amenity areas and in the home. Pests can be insects, rodents, weeds or fungi. Pesticides are closely controlled under European and UK legislation because they are widely used in the environment. The greatest concern usually relates to insecticides, and particularly some of the older organochlorine and organophosphorus insecticides.

The organochlorine insecticides, such as aldrin, dieldrin and DDT, are of relatively low acute toxicity but they are very persistent in the environment and can accumulate through the food chain. The older organophosphate insecticides are of high acute toxicity but tend to break down in the environment and are not persistent. They both tend to be lipophilic and bind to particles and sediment, although they can volatilise into the atmosphere. Most of the highly toxic pesticides are no longer used.

Pesticides are not normally a significant contributor to waste and their presence in waste should be controlled. Even in the past the quantities that were expected to enter waste was considered to be small because most disposal was on the farm. The exception would be timber treated with wood preservatives that could be present in timber disposed of by incineration, to landfill or by composting. Of particular concern under these circumstances would be the inorganic copperchrome arsenates, which are considered under arsenic. However, there is evidence that the herbicide mecoprop can be found in leachate from landfill and it is known to have contaminated groundwater at at least one site in the UK. Drinking water is not an issue because there is extensive monitoring of drinking water to ensure that the European and UK precautionary value of 0.1 µg/l is met (WHO, 1996a).

## Why are they important?

Whilst there have been cases of accidental exposures to pesticides and evidence of effects in pesticide sprayers, primarily from the use of organophosphate insecticides, acute toxicity resulting from environmental exposure is extremely uncommon.

The majority of exposure to low levels of pesticides is through food, but there still is considerable public disquiet about pesticides in the environment and in waste and this is the primary reason for their consideration. There is little evidence for significant exposure as a consequence of their disposal but some of the older pesticides may be disposed of illegally to landfill. There would be concern about stores of old pesticides, which should have been correctly disposed of as special waste, if there was access by children.

## Possible Effects

There are a wide range of differing pesticide molecules with widely differing toxicity and a wide range of different possible toxic effects, depending on the particular pesticide. Those of greatest concern are, perhaps, those that are both persistent and which bioaccumulate such as the organochlorines indicated above. Other pesticides, such as the organophosphate insecticides are primarily considered to be acute toxins. The wood preservatives include pentachlorophenol, TBTO and organoarsenicals. However, many pesticides are of low



mammalian toxicity and the primary concern arises from risks of ecotoxicity, particularly in the aquatic environment.

Examples of acceptable or tolerable daily intakes associated with a number of pesticides of possible concern are given below (WHO, 1999).

**Table A.4 Tolerable daily intakes associated with a number of pesticides**

Pesticide	Use	Chemical Group	Acceptable Daily Intake
Aldrin/Dieldrin	Insecticide	Organochlorine Discontinued	0.1 µg/kg bw
Atrazine	Herbicide	Triazines	0.5 µg/kg bw
DDT	Insecticide	Organochlorine Discontinued	10 µg/kg bw
Fenitrothion	Insecticide	Organophosphate	5 µg/kg bw
Lindane	Insecticide	Organochlorine	1 µg/kg bw
Malathion	Insecticide	Organophosphate	300 µg/kg bw
Pentachlorophenol	Wood preservative	Chlorophenol	3 µg/kg bw
TBTO	Wood preservative	Organotin	0.25 µg/kg bw

# Mercaptans

## Are they important?

Mercaptans are a family of thiol containing organic compounds that are formed naturally in biological systems, they can be found in the volatile components of freshly crushed aromatic bulbs, such as onions. They are likely to be found associated with putrescible material and have a very unpleasant odour at very low concentrations in air and water. They are, therefore, expected to be widely associated with a number of waste streams, although there appear to be few data on exposure to mercaptans from waste.

## Why are they important?

They have a very low odour threshold and a very unpleasant odour and are widely used as odourants in a variety of circumstances, most notably to impart a distinctive and unpleasant odour to natural gas. As a consequence of their volatility, they will easily be lost to atmosphere from waste disposal options involving putrescible material and biological activity. They will be easily detected by their unpleasant odour and, although they appear to be of relatively low toxicity, some are also used as approved food additives, their presence would be a cause of distress for residents close to the site.

## Possible effects

Low concentrations in the air can give rise to nausea and headache due to the strong, offensive smell associated with most mercaptans. Methylmercaptan, which has an odour of rotten cabbage is considered to be similar to hydrogen sulphide in toxicity. Ethylmercaptan was studied in human volunteers in whom a concentration of 3 ppm for 4 hours per day over several days appeared to be largely without effect. Benzylmercaptan, which is found naturally in coffee, is reported to cause slight irritation to mucous membranes. The concentration at which propylmercaptan is unlikely to be a health hazard is 0.5 ppm, which is the occupational threshold limit value in the USA, it is reported that most individuals would detect it by odour at this concentration. At much higher concentrations they can cause irritation of eyes and mucous membranes and at very high concentrations they can also induce CNS depression (HSDB).

The lower molecular weight mercaptans appear to be of low toxicity below the odour threshold. However, it is unclear at what concentrations odour fatigue can occur.

# BTEX Compounds

## Why are they important?

The BTEX compounds are a group of petroleum hydrocarbons comprising benzene, toluene, ethylbenzene and the xylenes that are found in petroleum fuels containing low molecular weight components but they are also used in their own right as solvents. They are water soluble and are volatile. The BTEX compounds are readily biodegraded under anaerobic conditions and they generally have a half-life of a few days in air. They are likely to be present in waste but exposure from other sources, particularly petroleum fuels would be expected to be very much greater. They are found widely in indoor air, particularly where there are smokers (IEH, 1999).

## Possible effects

The BTEX compounds are well absorbed from the lungs and gastrointestinal tract. They are acutely toxic at high concentrations giving rise to central nervous system depression with a concentration of approximately  $65 \text{ g/m}^3$  of benzene capable of causing death. The other BTEX compounds are much less toxic, although they probably act additively. At much lower concentrations, of benzene vapour such as are found in occupational exposures to greater than  $162 \text{ mg/m}^3$ , benzene is toxic to the haematopoietic system giving a range of adverse effects, particularly relating to white blood cells. Chronic occupational exposure to toluene may be associated with an increased risk of spontaneous abortion and there is some evidence that it may cause foetotoxicity and developmental effects with chronic exposure to high concentrations, above  $300 \text{ mg/m}^3$  (WHO, 2000a; WHO, 1985). Xylenes have also been shown to be foetotoxic in animal studies but there are only very limited data on ethylbenzene (WHO, 1996b, 1970).

The most important potential health effect associated with benzene is that of carcinogenicity. There is sufficient evidence that chronic exposure to benzene can give rise to leukaemia and IARC have classified benzene as group 1, sufficient evidence of carcinogenicity in humans (IARC, 1990). However, the concentrations with which associations with an increased risk of cancer have been observed are in the region of  $325 \text{ mg/m}^3$  and above, which are several orders of magnitude above ambient concentrations (WHO, 2000a; IARC, 1987; EPAQS, 1994b). There is no indication that the other BTEX compounds are carcinogenic. Standards and WHO guidance levels in air for BTEX compounds in air range from a proposed  $3 \text{ } \mu\text{g/m}^3$  for benzene to  $0.26 \text{ mg/m}^3$  (toluene),  $0.87 \text{ mg/m}^3$  (xylenes) and  $22 \text{ mg/m}^3$  (ethylbenzene). There is a drinking water standard for benzene of  $1 \text{ } \mu\text{g/l}$  and WHO drinking water guideline values of  $300 \text{ } \mu\text{g/l}$  for ethylbenzene,  $500 \text{ } \mu\text{g/l}$  for xylenes and  $700 \text{ } \mu\text{g/l}$  for toluene, although these last three are above many of the reported odour thresholds in water (Commission of the European Communities, 1998).

# Methane

## Is it important?

Waste materials that are disposed of in landfill sites undergo a number of complex microbial degradation processes. Initially, degradation is aerobic and generates water and carbon dioxide as the major decomposition products. As oxygen becomes deficient, anaerobic microbes continue the decomposition process. Initially complex organic molecules are degraded to release hydrogen and carbon dioxide. Further degradation processes lead eventually to the production of methane and water.

Methane is a colourless, odourless gas and methane, together with carbon dioxide, are the principal components of landfill gas. In addition, other microbial, chemical and physical processes can produce a range of other trace components. For example, methane is often found mixed with hydrogen sulphide, causing the distinctive "rotten egg" smell.

## Why is it important?

Methane gas is highly explosive when mixed with air at a volume between 5% and 15% (50,000 ppm to 150,000 ppm), which are the lower and upper explosive limits of methane.

As gas is produced within a landfill site an increase in pressure will develop. This pressure may force the gas into the atmosphere by diffusion through permeable rocks or along natural or man-made faults. The evolution of landfill gas in this way is dependent on a number of factors, including atmospheric pressure. The accumulation of methane gas in structures (e.g., basements, crawl spaces, utility ducts) can result in explosion and fire. Methane gas migrating underground can damage or kill vegetation in surrounding areas because it displaces oxygen and effectively suffocates plant roots.

Exposure is generally through inhalation, with skin and eye absorption minimal. Because methane is very volatile and has low water solubility, it is usually not found in food or drinking water. Occasionally very low-level exposure can occur when contaminated water is used for drinking and/or for food preparation.

## Possible effects

Methane is not toxic but is an asphyxiant and can displace oxygen in enclosed spaces. Exposure to oxygen-deficient atmospheres may produce dizziness, nausea, vomiting, loss of consciousness, and death. It can form flammable or explosive mixtures with air and is violently reactive with oxidizers and some halogen compounds. The concentrations at which flammable or explosive mixtures form are much lower than the concentration at which asphyxiation risk is significant. Therefore, before suffocation could occur, the lower flammable limit for methane in air will be exceeded; resulting in both an oxygen deficient and an explosive atmosphere.

**NOTE:** Landfill gas may also contain more toxic gases, albeit at lower concentrations than methane, such as hydrogen sulphide and volatile organic compounds (VOCs). The perception of an odour may also cause a nuisance.

# Particulate Matter

## Is it important?

Airborne particulate matter has a primary component, which is emitted directly from sources such as road traffic and industry including incineration, and a secondary component which is formed in the atmosphere by chemical reactions of gases, most notably sulphur dioxide, oxides of nitrogen and volatile organic compounds (APEG, 1999). Environmental modelling in the UK has identified three predominant contributors to PM<sub>10</sub> mass in the UK; traffic, secondary particles, and coarse particles arising from the resuspension of surface soils and dusts, sea spray and construction activity. PM<sub>10</sub> is used to define particles in the atmosphere with an aerodynamic equivalent diameter of less than 10 micrometres in diameter.

Emissions from incinerators can be a source of particles and there are strict regulatory limits on the amount of particles emitted. Incineration and the metals industry contribute about 7% of the total airborne particle (not secondary) emissions in the UK (EPAQS, 1996a).

The fine particle fraction (PM<sub>2.5</sub>) is composed of predominately secondary particles, including both inorganic salts and organic compounds, and primary combustion-generated particles, mainly from road traffic and in some areas of coal burning and industry. Combustion sources tend to produce small particles made up mainly of carbon with other material adsorbed onto the surfaces of the particles and blended in their interiors.

Waste disposal via landfill and composting may also generate dust albeit the majority coarse. This dust, particularly from composting, may have a biological element and the potential health implications of this bio-aerosol may be very difficult to ascertain. Whilst part of the aerosol produced by composting facilities and landfills will contain appreciable amounts of respirable particles, there is little research into the levels of dust and particulate matter in areas around such sites. However, a carefully sited, well-designed and properly managed facility should not present a significant threat to public health in the area. Practices at landfills to reduce fugitive dust emissions, for example damping down, will also significantly reduce the amount of dust emitted.

## Why is it important?

Inhalation is the major route of exposure to airborne particles and those particles that penetrate deep into the lungs are of greatest concern. Road traffic is the main source of primary particulate matter. Nationally, road transport contributes around 25-30% of PM<sub>10</sub> emissions but in urban areas this may be much higher. For example, in London traffic contributes 77% of emissions (APEG, 1999). Emissions in mainland Europe also contribute to primary particle levels in the UK.

It has been suggested that the majority of particulate matter from incinerators is ultra-fine in size (<0.1 µm in size) and that current air pollution devices on incinerators cannot prevent these emissions. However, both filtration theory and experimental measurements on filters demonstrate that filter collection efficiency increases for very small particles and that trapping of the ultra-fine fraction is likely to be rather efficient.

## Possible effects

There is much epidemiological evidence linking ambient particulate pollution with both acute and chronic health effects, with the size of the particles that individuals might be exposed to being very important. Recent research has demonstrated that low levels of atmospheric particulate matter (particularly fine and ultra-fine particles) can have measurable effects on the health of the population. These health effects include respiratory and cardiovascular morbidity and mortality. The Department of Health's Committee on the Medical Effects of Air Pollutants (COMEAP) reviewed the research on the effects on health of (non-biological) particles and concluded that epidemiological studies clearly demonstrate a relationship between adverse health effects in persons with pre-existing respiratory and cardiac conditions and levels of particulate matter (COMEAP, 1995). For example, particulate pollution can exacerbate asthma. However, COMEAP found no evidence that healthy individuals suffer any ill-health as a result of acute exposure to typical ambient air concentrations.

Many studies have described a relationship between hospital admissions and PM<sub>10</sub> concentrations in ambient air. One such study examined the impact PM<sub>10</sub> had on the health of people living in Birmingham (Wordley *et al.*, 1997). This study found that a 10 µg/m<sup>3</sup> increase in 24-hour PM<sub>10</sub> concentration corresponded to a 2.4% increase in daily respiratory admissions (to hospital) which in a population of 1 million would represent 0.5 extra respiratory admissions per day. Similar results have been reported elsewhere.

Much recent work has attempted to identify a no-effect threshold level for exposure to PM<sub>10</sub> but to date no such threshold has been identified. Therefore, the current UK air quality standard for PM<sub>10</sub> has been based on a level where the effects on the population as a whole would be relatively small. The current air quality standard for PM<sub>10</sub>, as set by the Expert Panel on Air Quality Standards (EPAQS) in 1996, identified a level of 50 µg/m<sup>3</sup> at which one might expect one additional hospital admission (for respiratory disorders) per day in a population of one million (EPAQS, 1996a).

Recently, COMEAP estimated that some 8000 deaths (all causes) had been "brought forward" by particulate pollution in urban areas of Great Britain, although it is not possible to estimate by how long (COMEAP, 1998). Furthermore, particulate pollution in urban areas resulted in 10,500 new or brought forward hospital admissions (for respiratory complaints) per year. It should be stressed that these effects are believed only to occur in patients with severe pre-existing disease. There is no evidence for effects in healthy individuals.

Toxicological studies indicate that it is the fine and particularly the ultra-fine particles (<0.10 µm) that are most strongly associated with adverse health effects. The ultra-fine particles appear to be capable of producing inflammatory reactions in the lungs and of promoting the clotting of blood. Even though they would account for only a small proportion of the total mass of PM<sub>10</sub>, they may represent a high proportion of the number of particles present. The main sources of other such particles are road transport and combustion processes.

Finally it is important to realise that there is increasing evidence that the chronic effects of exposure to airborne particles are as or perhaps more important in public health terms than the acute effects upon which the air quality standards are based. Methods for estimating chronic impacts are very much in their infancy. However, the impact of modern incinerators upon long-term average concentrations of particulate matter is likely to be extremely small, and therefore any chronic impact of particles from this source upon public health will also be small relative to that of other sources.

# Sulphur dioxide

## Is it important?

Sulphur dioxide (SO<sub>2</sub>) is a colourless, soluble gas with a characteristic pungent smell. The principal source of SO<sub>2</sub> levels in ambient air is from large power stations generating electricity through the combustion of fossil fuels containing sulphur. Other major sources of SO<sub>2</sub> are the combustion of fossil fuels by other industries and domestic coal burning, which is still permitted in some areas. Whilst motor vehicles are a relatively unimportant source nationally, they can make a small contribution to background levels in urban areas (EPAQS, 1995).

Emissions from incinerators can also be a source of SO<sub>2</sub> and there are strict regulatory limits on the amount of SO<sub>2</sub> emitted. Other waste disposal options are unlikely to be major emitters of SO<sub>2</sub>. Waste treatment and disposal is estimated to contribute less than 0.01% of the total airborne SO<sub>2</sub> emissions in the UK (approximately 4 thousand tonnes) (EPAQS, 1995).

## Why is it important?

Inhalation is the major route of exposure and SO<sub>2</sub> is a potent respiratory irritant and both causes and aggravates symptoms particularly in subjects with pre-existing asthma. Typically atmospheric levels of SO<sub>2</sub> tend to fluctuate widely from day to day and show a marked seasonal pattern with levels tending to be higher during the winter. The pattern of SO<sub>2</sub> pollution in most of the UK is characterised by short-term peak concentrations, typically lasting a few hours, at some point downwind of the industrial source (usually a power station).

## Possible effects

Typically asthmatics may experience tightness of the chest, coughing and a deterioration of lung function on exposure to SO<sub>2</sub> concentrations exceeding 262 µg/m<sup>3</sup> (100-200 ppb over a few minutes). An annual mean concentration of SO<sub>2</sub> of 60-140 µg/m<sup>3</sup> is associated with increased respiratory symptoms in adults and there are reports that concentrations of between 140-200 µg/m<sup>3</sup> increase respiratory illness in children. Current evidence suggests that there is not a threshold of effect of SO<sub>2</sub> for either mortality or hospital admissions. In addition, the effects of SO<sub>2</sub> may be magnified during co-exposure to other air pollutants such as particulates and nitrogen dioxide (NO<sub>2</sub>). There is evidence that children may be more susceptible to SO<sub>2</sub> when co-exposed to particulates, while co-exposure to SO<sub>2</sub> and NO<sub>2</sub> can increase the sensitivity to allergens of some patients with asthma.

A number of studies have begun to examine the relationships between concentrations of SO<sub>2</sub> and daily variations in a number of indices of health such as numbers of deaths, hospital admissions and lung function. Recently the Committee on the Medical Effects of Air Pollutants (COMEAP) has attempted to quantify the effects of SO<sub>2</sub> on human health (COMEAP, 1998). Their best estimates of the acute effects are that a 50 µg/m<sup>3</sup> rise in the 24 hour average concentration of SO<sub>2</sub> will raise the death rate by 3% for all causes, 4% for cardiovascular diseases, and 5% for respiratory disease. It must be stressed, however, that these effects relate to effects in patients with pre-existing respiratory disease. Currently COMEAP estimate that SO<sub>2</sub> contributes to the advancement of around 3,500 deaths and

results in 3,500 hospital admissions for respiratory disease in the urban populations of the UK annually. The latter figure is based on a coefficient of effect of an increase of 0.5 - 0.6 % per  $10\mu\text{g}/\text{m}^3$   $\text{SO}_2$  as a 24 hour mean (i.e. for every  $10\mu\text{g}/\text{m}^3$  increase in  $\text{SO}_2$  concentrations, there will be an increase in hospital admissions on the UK of 0.5%).

There are a number of environmental guidelines and standards for  $\text{SO}_2$ . Most relevant are the UK and WHO air quality standards for sulphur dioxide. The UK National Air Quality Standard for a 15 minute average is  $262\mu\text{g}/\text{m}^3$  (100 ppb) and the WHO air quality guidelines are  $500\mu\text{g}/\text{m}^3$  for a 10 minute averaging period,  $125\mu\text{g}/\text{m}^3$  for a 24 hour averaging period and an annual guideline of  $50\mu\text{g}/\text{m}^3$  (DETR, 2000; WHOROE, 2000). Emissions from incinerators are well regulated and ground-level concentrations arising from incineration should only be a relatively small proportion of the existing background concentration.



# Oxides of Nitrogen

## Are they important?

All combustion processes, including incinerators, produce oxides of nitrogen ( $\text{NO}_x$ ) and there are strict regulatory limits on the amount of  $\text{NO}_x$  emitted. Nitrogen dioxide ( $\text{NO}_2$ ) is produced both directly as a primary air pollutant and indirectly as a secondary air pollutant due to the conversion of nitrogen oxide ( $\text{NO}$ ) to  $\text{NO}_2$  via reaction with chemically active species such as ozone. However, in the UK, over 50% of all atmospheric  $\text{NO}_x$  is produced by motor vehicles and as a result  $\text{NO}_2$  concentrations tend to be higher in urban areas due to the levels of traffic. Exposure may also occur in the home with gas cookers being a major source of  $\text{NO}_x$ .

In remote, unpolluted areas levels of  $\text{NO}$  are a very small fraction of those of  $\text{NO}_2$  but in more polluted urban areas where the oxidising capacity of the atmosphere may be limited,  $\text{NO}$  levels may approach or occasionally exceed  $\text{NO}_2$ .

Emissions from incinerators can also be a source of  $\text{NO}$  and there are strict regulatory limits on the amount of  $\text{NO}$  emitted. Other waste disposal options are unlikely to be major emitters of  $\text{NO}$ . Waste treatment and disposal is estimated to contribute less than 0.1% of the total airborne  $\text{NO}$  emissions in the UK (approximately 4 thousand tonnes) (EPAQS, 1998).

## Why is it important?

Inhalation is the main source of exposure and  $\text{NO}_2$  is an irritant of the airways. Exposure to high concentrations can produce narrowing of the airways (bronchoconstriction) in both asthmatic and non asthmatic individuals.

## Possible effects

Exposure to concentrations of approximately  $560 \mu\text{g}/\text{m}^3$  (300 ppb) for 30 minutes can produce small effects on the lung function of asthmatics, while in non-asthmatics exposure to concentrations of  $1800 \mu\text{g}/\text{m}^3$  (1 ppm) is necessary to produce the same response (EPAQS, 1996). Adverse effects are unlikely to occur below a concentration of 200 ppb ( $400 \mu\text{g}/\text{m}^3$ ) for a 1 hour exposure.

Chronic exposure to nitrogen oxides has been associated with decreased respiratory function in children that can result in increases in respiratory disease and also increased airway resistance in asthmatics and people with chronic obstructive airways disease.

The National Air Quality Strategy includes two objectives for  $\text{NO}_2$  (DETR, 2000); an hourly air quality standard of 105 ppb ( $200 \mu\text{g}/\text{m}^3$ ) and an annual mean of 21 ppb ( $40 \mu\text{g}/\text{m}^3$ ) derived from the revised WHO air quality guidelines (WHOROE, 2000).

Since asthmatics have airways that are unusually sensitive to irritant pollutants, they represent a susceptible subgroup of the population. Young children, the elderly, and individuals with other chronic respiratory disease (e.g. bronchitis, emphysema) are also sensitive to nitrogen dioxide pollution. Exposure may also increase the response to allergens in particularly sensitive individuals.

COMEAP regard the effects of NO<sub>2</sub> as among the most difficult to assess and current data have not allowed COMEAP to predict estimates of the effects of NO<sub>2</sub> on mortality and hospital admissions (COMEAP, 1998). Available evidence provided by time-series studies is less well developed in the case of NO<sub>2</sub> than in the case of particles and sulphur dioxide and it is possible that the apparent effects of NO<sub>2</sub> are due to confounding by particles.