Liverpool NHS
Health Authority

The City of Liverpool

Report on the Possible Increase in Cancer Cases in North Liverpool and Potential Links to the Site of the Former Incinerator at Fazakerley Hospital

Edited by:

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On behalf of the Fazakerley Incident Team

Contents

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- Local residents in Fazakerley ward.
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- Dr Andrew Sewart, formerly dioxins adviser to the Department of Health.
- Professor Andrew Semple, formerly Professor of Public Health Liverpool University and Medical Officer of Health for Liverpool (1950-1974)
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- Greenpeace UK
- Dr Vyvyan Howard, Senior Lecturer in Fetal Pathology , Liverpool University
- Dr Paul Aylin, Clinical Senior Lecturer at Imperial College London

EXECUTIVE SUMMARY

1.1. Study Overview

- Public concern that cancer deaths since 1974 linked to emissions from closed hospital incinerator.
- 25 cases, 18 deaths on Haven Road and 1 death on Longmoor Lane in Liverpool 9 over a 26-year period from 1974 to 2000.
- 5 streets on the Formosa Drive estate including Haven Road where cancer deaths are perceived to be higher.

Liverpool Health Authority, in conjunction with Liverpool City Council, has conducted a study into the concerns of local residents in the **Fazakerley** and Gillmoss area of the city, regarding the incidence of cancer there. This area is estimated to be that most likely to have been covered by emissions from the incinerator.

The particular concerns are that a number of cancer deaths since 1974 could be linked to emissions from the now closed and demolished incinerator at **Fazakerley** Hospital. An in-depth review was conducted by Merseyside and Cheshire Cancer Registry of cancer cases occurring in the neighbourhoods of **Fazakerley** and Gillmoss. Further specialised work on cluster analysis was commissioned from Lancaster University. National and local experts in the health impact of incinerators also agreed to provide an independent review of the study findings.

The study addressed the following three main questions:

- Is the spatial distribution of cancers of interest greater than would be expected based upon the known population density?
- Does the distribution of cancers of concern differ from that of cancers not thought to be associated with incinerator emissions?
- Do the cancer rates in the study area differ from those in other similar areas e.g. after adjusting for other known causes of cancer including socio-economic deprivation?

The Health Authority and City Council worked with all parties to ensure that concerns were addressed. The findings of the study are being made publicly available by a formal launch with copies supplied to all key stakeholders and by publishing this report on the **NW Regional Public Health Observatory's website, www.nwpho.org.uk** Copies of this report are also being sent to 3 Government Advisory Committees: **The Committee for the Medical Effects on Air Pollution, The Committee on Toxicology** and **The Committee on Carcinogenicity.**

1.2. Historical Summary of Air Pollution in Liverpool

Air pollution has been a long-standing problem in Liverpool, linked with rapid urbanisation in the nineteenth century and the use of coal in domestic grates. The Smokeless Zones created through the 1956 Clean Air Act dramatically improved the levels of smoke pollution in Liverpool, achieving a visibly cleaner atmosphere by the mid-1970s, when most homes had been converted to smokeless fuels.

Levels of industrial air pollution reflect the changing economic profile of the city, moving from a port-based economy to limited diversification by the middle of the twentieth century. Liverpool's manufacturing base was historically strongly tied to port activities – including food processing and light engineering. There has never been a heavy industrial sector in Liverpool, although there is a significant chemical industry nearby at Runcorn/Widnes. Industrial air pollution has probably been of secondary importance after domestic sources of air pollution in the city. Industrial sites in the **Fazakerley** district were explicitly developed in the 1930s for 'clean air' trades.

Liverpool City Council appears to have applied 'best practice' to issues of air pollution during the period of review. The archives in Liverpool and at the Public Record Office illustrate the involvement of Liverpool officials in a wide range of measures to reduce all sources of air pollution. The Medical Officer of Health, Professor Andrew Semple (1950-1974), maintained a vigorous approach to air pollution monitoring and the identification of individual polluters, including hospitals.

The **Fazakerley** Estate was constructed by Liverpool Corporation between 1919 and 1945 on the periphery of the urban area to provide affordable rental property. It has remained as a mainly working-class district, with the majority of its occupants classed as skilled or semi-skilled. The district has suffered from socio-economic deprivation since the 1960s, linked to the citywide economic recession. The studies conducted in the early 1970s indicated a range of problems that may also have had health outcomes.

1.3. Air Pollution Control Measures in UK in the Early 1990s

Air pollution control in the UK has been built up stage by stage in response to particular, primarily health-related, problems. Its development has been essentially pragmatic with the regulator working closely with the regulated to prevent/minimise emissions taking into account local, economic, and technological factors. Control is exerted at both the local (i.e. local authorities) and central (i.e. Her Majesty's Inspectorate of Pollution now the Environment Agency) level. Prior to the introduction of Environmental Protection Act 1990 there was a disparity in the way that HMIP and local authorities controlled emissions from non-domestic sources, partly due to the regulatory frameworks in which each operated but also due to differences in in-house expertise/approach.

It is impossible to state whether emissions from scheduled processes have been adequately controlled by HMIP without knowing the knowledge/resources that were available at any specific point in time.

As noted by the RCEP:

> ***"Through the decisions reached with major industries about the nature and timing of measures to reduce airborne emissions the Inspectorate have effectively determined the pace of advance without any explicit reference to either local interests or to any generally formulated national policy objectives. We do not doubt that the pressures by outside factors, for example by advances in scientific understanding of the effects of pollutants…But the decisions are those of the Inspectorate alone (para 118)."***

However, the impression regarding local authority control is that at times it was inadequate. In the words of the RCEP:

> ***"We are clear that the quality of local control is…patchy (para 136)."***

1.4. Review of Current Research on Incinerators and Cancer Incidence

The aim of the review was to examine the evidence from epidemiological studies of an association of living near incinerators and an increased risk of cancer. The risk of cancer in workers at incinerators was also reviewed.

Few epidemiological studies were found. Findings were not consistent across the studies. Evidence of an association between living near or working at incinerators and cancer risk was weak. In general, studies were limited in their ability to detect excesses of cancer by their size, quality of exposure data measured and adequate adjustment for confounding especially socio-economic status.

The applicability of the findings from any one study is limited. The most relevant study to address community concerns in Liverpool was a study carried out by the Small Areas Health Statistics Unit in the UK, on 14 million people living near 72 municipal waste incinerators. This study found excesses in the incidence of all cancers and cancers of the stomach, lung and colo-rectal, which the authors felt were explained by deprivation. Deprivation could not be excluded as the cause of a small excess found of primary liver cancer near incinerators.

If epidemiological studies are carried out in the future, to obtain good quality information, the studies need to be large enough and long enough to detect small increases in risk over the longer term and also need to address possible confounding factors. Ideally, they would be combined with better measures of exposure to pollutants. The aim of the review was to evaluate the epidemiological evidence for carcinogenic effects of chronic exposure to incinerators on the population and on incinerator-workers. All epidemiological studies were identified using a search of bibliographic databases, reference lists of relevant articles and contact with experts.

All studies that reported actual cancer outcomes as opposed to cancer risk or laboratory data were included regardless of quality. Eight studies were identified which investigated effects on the general population. Two studies were identified which examined chronic occupational exposure. All studies were observational and of variable design and quality. Findings were not consistent. Positive associations were found in population studies with liver cancer, lung cancer, kidney cancer, soft-tissue sarcoma, non-Hodgkins lymphoma and childhood cancer. Occupational studies found associations with lung cancer and gastric cancer. Major limitations include quality of exposure data and adequate adjustment for confounding especially socio-economic. The applicability of the findings from any one study is limited. Emissions from the incinerators studied are likely to have been higher than from modern incinerators.

There is inadequate evidence from epidemiological studies to support or refute the hypothesis that chronic exposure to incinerators has a carcinogenic effect on the general population or on incinerator workers.

Few studies have been done about adverse health effects of chronic exposure to incinerators. There is conflicting evidence of associations with respiratory function.

1.5. Environmental and Toxicology Reviews

ENVIRONMENTAL REVIEW
The incinerator under consideration no longer exists as it was demolished after operation ceased in September 1995. This meant that it was not possible to carry out test firings and record dispersion plumes.

- When the Fazakerley incinerator closed down in 1995, it met or was better than the standards, which were in place at the time.
- Liverpool City Council's Environmental Health Department's report in April 1994 concluded that the weekly monitoring programme since November late 1993 had failed to provide any evidence which would justify the City Council requesting additional or more frequent monitoring of emissions from either the hospital incinerator or boilers.
- The worst case scenario produced by the MERMAID model showed that the highest concentration of sulphur dioxide (SO_2) from the incinerator at ground level was **0.012-0.024 parts per million**. These concentrations were found to exist at greater distance from the source than the properties on Lower Lane and complied with all the then (1994) air quality standards.
- The review of the environmental data and reports conducted by Manchester Metropolitan University concluded that even with the "worst case" scenario produced by the MERMAID model the maximum pollutant concentration levels were indicative of the incinerator operating within regulatory limits.

The Chemical Incident Response Service (CIRS) at Guys and St.Thomas' Hospital London were asked to contribute to the investigation. *They concluded that there are a number of lessons to be learned from the investigation of the Fazakerley incinerator incident:*

REGULATION
With the advantage of hindsight and more recent legislation in place, it should be remembered that when the Fazakerley incinerator closed down in 1995 (and demolished in 1996), it met, or was better than, the standards, which were in place at the time. Provisions in the legislation and the existence of Crown immunity prior to 1990 did not require emissions from hospital incinerators to be regulated or monitored. Current UK legislation will in future strictly regulate permitted levels of emissions from incinerators, and the Environment Agency will be required to consult with Health Authorities. The latter are expected to anticipate and assess the health impact of emissions on their local populations, as required under the Integrated Pollution Prevention Control legisltaion.

MONITORING
The lack of regulatory legislation and monitoring requirements for older incinerators prior to 1990 means that little data exists on the chemical emissions or their dispersion paths. Studies associating incinerators with specific disease entities are frequently based on proximity to the incinerators; associations cannot be accurately attributed to specific emissions or assessed for the effects of different concentrations of a chemical.

ADDRESSING PUBLIC CONCERN
Public concern about the hazards of emissions existed for many years but there was little acknowledgement of this. There has been a 'sea change' in attitudes to public concerns about environmental impact on health. The government has called for multi-agency consultation and public participation assessing health impacts of new major developments and review of existing industries by either the Environment Agency or the Local Authority under Pollution and Prevention Control legislation. The health impacts from waste incineration are still not well understood and the Environment Agency is working with the Department of Health to prepare national protocols and standards to assess health impacts.

1.7. Review of Cancer Cases in Fazakerley 1974 to 1998

There is no evidence from the study carried out by the Merseyside and Cheshire Cancer Registry (MCCR) that rates of cancer in the study area (postcode districts L9, L10 and L11) are unusually high compared with LHA. The study area is similar to Liverpool Health Authority in having significantly higher rates of some cancers when compared with Merseyside and Cheshire as a whole. Unfavourable lifestyle factors and higher deprivation are the most likely explanations for these variations.

- In general the overall pattern of cancer in the study area was similar to that in LHA and Merseyside and Cheshire as a whole, in relation to the distribution by cancer site, gender and over time. The exception is lung cancer, for which the proportions were higher in the study area and LHA compared with Merseyside and Cheshire for males and females.
- Overall cancer rates were similar in the study area and LHA for males and females, however both were higher compared with Merseyside and Cheshire. This finding was significant in all cases except for females in 1989-93 when the study area had similar rates compared with Merseyside and Cheshire as a whole.
- There were no significant differences between the incidence rates in the study area, LHA and Merseyside and Cheshire for: leukaemias, non-Hodgkin's lymphoma, and cancers of the colon and rectum in males and females, and cancer of the larynx in males only.
- There were no significant differences in the incidence rates of childhood cancer in the study area, LHA and Merseyside and Cheshire as a whole.
- There were no significant differences between the study area and LHA as a whole for the rarer cancers considered. Some statistically significant differences were found for liver and laryngeal cancer in males and females respectively, between LHA and Merseyside and Cheshire as a whole, although these are difficult to interpret.

LUNG CANCER

The high rate of lung cancer in Liverpool has been known for some time and reported in several publications produced by MCCR. Lung cancer has a strong association with deprivation, which is associated with higher rates of smoking in more deprived areas. Smoking is known to be the major cause of lung cancer and is thought to account for 90% of lung cancer cases.

The analyses undertaken by MCCR gave rise to the following conclusions in relation to lung cancer for the period 1974-98:

- Lung cancer rates in the study area were not significantly different from rates in LHA for males and females, and over time.
- Lung cancer rates in the study area and LHA were significantly higher than rates for Merseyside and Cheshire as a whole for males in both 1979-83 and 1989-93, although rates are decreasing over time.
- Lung cancer rates in females in the study area and LHA were significantly higher than rates for Merseyside and Cheshire as a whole, in 1979-83 and 1989-93 and rates are increasing over time.
- The rate of lung cancer in females in the study area almost doubled between 1979-83 and 1989-93.
- The socio-economic profile of the study area is similar to that of LHA, and both areas are more deprived overall compared with Merseyside and Cheshire.
- Within socio-economic group lung cancer rates in the study area were similar to those in LHA and Merseyside and Cheshire as a whole for males and females.
- Lung cancer rates in females were higher in the study area for most socio-economic groups, although this was not statistically significant.
- Colon and rectal cancers, chosen for their lack of known association with incinerator emissions, have shown similar patterns of distribution in the comparison areas over time, and by gender, suggesting that the study area does not generally have unusual background levels of cancer.
- The significantly higher overall lung cancer rates in the study area and LHA compared with Merseyside and Cheshire generally lose their statistical significance when rates are compared within socio-economic group.
- Higher population levels of deprivation are therefore a more likely explanation for the significantly higher lung cancer rates in the study area and LHA compared with Merseyside and Cheshire and this is further supported by their respective socio-economic profiles.

1.8. Specialist Geographical Analysis

This work was undertaken by Professor Peter Diggle and Dr. Mikala Jarner of the Medical Statistics Unit at Lancaster University. Data on cancer cases and control population were provided by Dr Kate Ardern of Liverpool Health Authority and Dr Lyn Williams of the Merseyside and Cheshire Cancer Registry. Ethical approval was obtained from the Chair of the Local Research and Ethics Committee for this aspect of the study.

This consisted of all known cases diagnosed between 1974 and 1998 within the area covered by post-code zones L9, L10 and L11.

Age and sex matched control locations were derived by random sampling from a data-base of GP registrations within the Liverpool Health Authority. This database listed the numbers of females and males in five-year age-bands resident at each unit post-code within the study area. The Townsend index (Townsend, Phillimore and Beattie 1988) was obtained for each census enumeration district (ED) within the study area and used as an individual-level adjustment for social deprivation.

Residual risk of cancer after adjustment for age, sex and deprivation was initially modelled parametrically as a non-linear function of distance from the incinerator . As an aid to interpretation of any possible variations in residual risk not attributable to proximity to the incinerator, a non-parametric generalised additive modelling approach (Kelsall and Diggle 1998) was used as a secondary analysis for the three most common types of cancer (adult colorectal, lung and leuakaemias and lymphomas).

The main overall conclusion was that after adjustment for the variations in risk factors attributable to age, sex and social deprivation, there is no significant evidence of a relationship between risk of developing cancer and proximity of residence to the site of the former incinerator.

- The additional analysis of any variation in the residual risk of cancer for the most common types of adult cancer in the study area - colorectal, lung and leukaemia and lymphoma showed a consistent excess of cancer incidence centred on the location 3 kilometres to east of the site of the former incinerator at grid reference (34000,39750) just inside the Cherryfield ward in Knowsley.
- There is a significant result of elevated residual risk for colorectal cancer which is strongly associated with lifestyle risk factors such as type of diet, obesity, lack of exercise and smoking and has no association (or biologically plausible link to) emissions from incinerators.
- The overall conclusion is that there is a statistically significant spatial variation on the risk of the more common types of adult cancer in the study area but that the pattern of variation does not show a simple distance-based relationship to the site of the former incinerator.
- The most likely explanations for this residual elevated risk is that it is either due to an unmeasured individual-level risk factor such as lifestyle risk factors: smoking, diet, exercise levels etc or to under-representation of the residents of this particular location in the GP registration data -base.
- Ideally, any further investigation should a prospective case-control study to obtain data on individual-level lifestyle risk factors such as smoking, diet and exercise levels. Failing this, consideration should be given to obtaining information on additional risk factors at Enumeration District level.

1.9. Conclusions and Recommendations

Currently in the UK, one third of all adults will die from some form of cancer. Thus cancers as a group of diseases are common. The investigation into the Fazakerley and Gillmoss concerns has taken account of the fact that Liverpool as a whole has high death rates from cancer. Over the period 1996-98 the average Standardised Mortality Ratio (SMR) for Liverpool for a common cancer such as lung cancer was in excess of 200 (persons) with the worst SMRs in the most socio-economically deprived wards. Vauxhall had the highest SMR for that period of 381. The high death rates for lung cancer were matched by high death rates for other diseases such as heart disease, chronic obstructive pulmonary disease and stroke associated with the 2 major risk factors - poverty and cigarette smoking.

In conducting this investigation, the Incident Team has been very aware of the complexity of investigating public concerns about the possible health consequences of incinerators and other industrial processes and environmental hazards.

The main points are:

- It is difficult to investigate a potential cancer cluster retrospectively when the main cause of concern no longer existed as a physical structure and therefore could not be monitored or tested.
- It is difficult to establish whether there was an increase in cancer cases that could not be explained by any other factor operating in the local population.
- Additional complications included establishing whether there are any other potential environmental, economic, social or lifestyle risk factors, and their relative contributions to cancer incidence in Fazakerley, and also whether the incinerator contravened the air quality standards in place at the time of its operation.
- Difficulties caused by paucity of primary information in some cases, eg lifestyle factors and its reliability, eg postcodes pre 1974, in others.
- Epidemiological studies carried out in the future, to obtain good quality information, need to be large enough and long enough to detect small increases in risk over the longer term, and also need to address possible confounding factors. Ideally, they would be combined with better measures of exposure to pollutants.
- In the event of future planning of waste incineration plants, specialist multidisciplinary, multi-agency knowledge should influence the decision of whether incineration is the best option. Environmental and health impact assessments will increasingly be required to be undertaken involving the Environment Agency, Local Authority, Health Authorities, CIRS and attention given to health impacts on populations.
- There is uncertainty about the impact of incinerator emissions on human health. It is the role of the government and of Public Health to communicate this uncertainty in an honest but understandable manner to the public. Issues about waste reduction, curtailing waste and the implications of incineration as a disposal option should be openly debated, so that the scientific community and the public are empowered to influence future projects and to minimise the consequent adverse effects on human health and wellbeing.
- This investigation points to the need for environmental monitoring and control in order to provide the necessary scientific tools to interpret public health concerns adequately.

RECOMMENDATIONS

Environmental Public Health Surveillance System:

This investigation has led to the Incident Team to call for and initiate the development of a formal surveillance system for environmental public health. A feasibility study has already commenced in collaboration with colleagues from other local and regional agencies Lessons from this investigation are also being used to prepare national protocols and standards to assess health impacts of industrial processes through a small working group co-ordinated by the Chemical Incident Response Service and members of the Incident Team.

Communicating Risk

The Incident Team recommends that the lessons learnt from this study are used by Governmental Advisory Committees to develop better public information on the potential risks to health from industrial processes and to consider how to develop public participation in reviewing research evidence. For example, the Cochrane Collaboration has a model of patient and service user involvement that could assist in this.

The Incident team recommends that lessons from this study on communicating risk are incorporated into the work currently being undertaken by the Chemical Hazards Management and Research Centre at Birmingham University and the Chemical Incident response Service at Guys and St Thomas' Hospital London in developing the Health Authorities' response, in their new role as statutory consultees, to Integrated Pollution Prevention Control applications .

The Merseyside and Cheshire Cancer Registry has already undertaken to revise and update its public information on cancer as a result of participating in this study

DISSEMINATION OF THIS REPORT

This report will be formally presented to both Liverpool Health Authority and Liverpool City Council. It will then be made publicly available by a formal launch with copies supplied to all key stakeholders. Following its launch it will be publicly available on the NW Regional Public Health Observatory's website: **www.nwpho.org.uk** and the Liverpool Health Authority website : **www.liverpool-ha.org.uk**

2 BACKGROUND TO INVESTIGATION

2.1. History of the Incident

Since 1974 there have been 26 cases of cancer, including 19 deaths in a small geographical locality in Liverpool. The area is in close proximity to the now defunct incinerator located at Fazakerley hospital, which operated from 1974 to 1995. The public perception is that these cases represent an excess of cancers which they believe may be associated with emissions from the incinerator.

The original simple incinerator was replaced in 1985 and again upgraded in 1992 to comply with interim emission standards in 1992. The incinerator was closed in September 1995 as it could not comply with the more stringent standards introduced under the Environment Protection Act which came into effect on 1 October 1995.

INCIDENT TIMELINE

1950s: Hospitals are bad polluters.

1956: Clear Air Act.

1974: Fazakerley becomes "Smokeless Zone."

1990: Interim EPA, UKCCR study starts, NHS crown immunity ceases.

1993: Child B diagnosed with cancer.

1994: Child B dies.

1995: Full EPA, incinerator closes.

1997: Civil actions against Trust.

2000: In April, Liverpool Health Authority Department of Public Health commissioned by Liverpool City Council - start of study.

2.2. Management of the Investigation

In accordance with the Guidelines issued by the Committee on the Medical Effects of Air Pollution in Feb 2000, the study proceeded through, as far as the Health Authority and City Council deemed to be reasonable on current available evidence, five stages:

- A preliminary assessment of the complaint(s) and its/their biological plausibility in terms of the alleged health effects led by the Director of Public Health.
- An assessment of possible sources of pollution and an examination of local monitoring data led by either the Local Authority or Environment Agency.
- A more detailed assessment of the health profile led by the Director of Public Health
- A more detailed assessment of emissions, including whether additional monitoring or modelling is required led by the Local Authority or Environment Agency.
- A more extensive investigation or study, involving either a more detailed examination of the health effects led by the District Director of Public Health or Regional Office and /or more extensive environmental exposure modelling led by the Local Authority or Environment Agency.

THE INCIDENT TEAM

An Incident Team has been established to manage the investigation which comprised:

Dr. Kate Ardern, Consultant in Public Health (chair)

Dr. Ruth Hussey, Director of Public Health

Dr Emer Coffey, Specialist Registrar in Public Health

Mr. John Watson, Director of Corporate Affairs (Until Feb 2001)

Ms Jean Scott, Executive Officer (from Feb 2001)

Dr. Lyn Williams, Senior Lecturer in Public Health at Liverpool University and Director of the Merseyside and Cheshire Cancer Registry (MCCR)

Mr. Andy Hull, Environmental Health Manager Liverpool City Council

Dr. Virginia Murray, Consultant in Occupational and Environmental Toxicology and Director of the Chemical Incident Response Service, Medical Toxicology Unit, Guys and St Thomas' Hospital.

With help, advice and assistance from the following people:

Mr. John Wood, Corporate Affairs Manager Aintree Hospital Trust

Mr. Jim Laird, Maintenance and Technical Services Manager Aintree Hospital Trust

Dr Adrian Watson, Senior Lecturer in Geography and Environmental Sciences at Manchester Metropolitan University on plume modelling.

The Environment Agency NW on records of industrial processes in Fazakerley and Gillmoss and contaminated land issues.

Dr Amin Anjum, PIR/RSR Regulator Specialist, the Environment Agency.

Dr Sally Sheard, Medical Historian Liverpool University on the historical context of environmental and housing background in Liverpool.

Dr Andrew Sewart, formerly dioxins adviser to the Department of Health.

Professor Andrew Semple, formerly Professor of Public Health Liverpool University and Medical Officer of Health for Liverpool (1950-1974).

Professors Tony Gatrell and Peter Diggle and Ms Mikala Jarner of Lancaster University on cluster analysis and geographical epidemiology.

Ms Tracey Clegg and Mrs Kath Chesters of Merseyside & Cheshire Cancer Registry.

Dr Margaret Meltzer (SpR Public Health Medicine) now Consultant in Communicable Disease Control, Hillingdon Health Authority.

Greenpeace UK

Dr Vyvyan Howard, Senior Lecturer in Fetal Pathology , Liverpool University.

Dr Paul Aylin, Clinical Senior Lecturer at Imperial College London (external peer reviewer).

The Incident Team met monthly from May 2000. Its purpose is to review progress, to agree the development of a project plan, to review the lessons learnt from this incident, to review the need for external expertise and to co-ordinate responses to the public, local politicians and the media. The Incident Team was responsible for reviewing and agreeing the final report on the incident. *The Incident Team agreed the following outputs from the study:*

- That a final Incident Report would be submitted to Liverpool Health Authority, Liverpool City Council, Alt Valley and Kirby Primary Care Group/Trust Boards and local residents.
- In addition, members of the Incident Team would prepare papers for publication in peer-reviewed journals and presentation in suitable professional and public forums.
- The lessons learnt from this incident will be reported to the Committee on the Medical Effects of Air Pollution with appropriate recommendations on the development of the current Guidance.
- Lessons learnt will also be used to help develop and update public information on cancer.
- The literature review will be used as a valuable source of information to assist the Health Authority in any future requests from the Environment Agency under the new legislation on Integrated Pollution Prevention Control on the licensing of new and existing processes.

2.3. Research Methods

CASE FINDING

The Incident Team agreed that cancer cases which might possibly be linked to the incinerator operations should be identified by searching for all cancer cases from 1974 onwards in the postcode areas of L9, L10 and L11 and the relevant street names. The rationale for this is that the complaints directed at the hospital incinerator start with cancer deaths in 1974 and the Cancer Registry data including postcode are valid and robust for this period. In addition, although the postcode areas will be wider than the area that was covered by the plume from the incinerator, the four civil actions pending against the Trust are from individual residents in each of these postcode sectors. In addition, the environmental data from the incinerator are very limited and it may be very difficult to produce a robust dispersion plume and therefore it was felt that it was better to look for cases in a wider geographical area than that which might be identified by plume modelling. **MCCR identified the relevant street names for the postcode areas and completed the case search**. The Cancer Registry also looked for co-variant information on risk factors such as smoking and occupational history where possible. *Three main routes were used to find cases in the defined area and over the time period:*

- Recent press coverage.
- Workforce data from University Hospitals Aintree - As part of their case finding, MCCR were sent the occupational health records of staff who operated and maintained the incinerator. The staff were approached individually by Aintree Trust and their written consent for access to their records obtained.
- Local Dermatology consultants at Aintree Hospitals NHS Trust and the Royal Liverpool and Broadgreen University Hospital Trust were contacted to see if they had treated any cases of chloracne (a symptom of acute dioxin poisoning) from North Liverpool in the time period 1974 to 1998.
- Merseyside and Cheshire Cancer Registry (MCCR), routinely collected data between 1974-1998.
- Written consent was obtained from the two main complainants to access medical records and, in one case, approach clinical colleagues involved in the case.

Dr Ardern briefed the board of Alt Valley Primary Care Group and the Chief Executive of Kirkby Primary Care Group on the residents' concerns and the aims of the study. The local residents are registered as patients with general practitioners who belong one or other of these PCGs. Dr Ardern requested that GP colleagues in both PCGs inform her if they notice the occurrence of any unusual cancers or patterns of cancers amongst their patients.

CLUSTER ANALYSIS

Once the geographical searches were completed and cancer cases identified. It was possible to establish whether there were any individual cancers which merited further investigation (see below) or whether the casemix was what would be broadly expected (ie common cancers are common) and therefore whether an area based cluster analysis would be more appropriate. Suitable controls from an area without an incinerator were identified and matched for age, sex and socio-economic deprivation (using appropriate socio-economic indices such as Carstairs, Superprofiles and the recent Index of Deprivation) and comparisons made to assess the relative risk of cancer generally and biologically plausible cancers (larynx, lung, childhood lymphomas/leukaemias) between the study group and the control group.

SPECIALIST INPUT (COMMISSIONED FROM LANCASTER UNIVERSITY)

Professor Gatrell who is a national expert in this field advised the Incident Team on the most appropriate type of cluster analysis that he and colleagues at Lancaster University could undertake. This broadly falls into two main categories.

Firstly, in collaboration with MCCR assisting the Cancer Registry with area based analyses that would use Kernal estimation to establish background risk and pinpoint areas of elevated risk. This would be very similar to the service offered by the Small Area Health Statistics Unit's rapid inquiry facility. This would take account of co-variant data on smoking, socio-economic status, deprivation etc. This might be very useful with regards to developing public information about how common types of cancer really are.

Secondly, if the initial case finding revealed that there are individual cancers which require more in-depth investigation - i.e. testing the hypothesis that the closer an individual lives to the point source the more likely they are to develop a particular cancer. This would include modelling the cancer of interest as a function of proximity to the point source (in this case the incinerator). This would establish the relative risk of contracting a particular type of cancer and distance of residence from the point source. The modelling would take into account, as far as possible, other risk factors- smoking, socio-economic status, deprivation factors and environmental information such as prevailing wind direction.

INVESTIGATION OF RISK

The initial focus was to look at cancers in postcode sectors L9, L10, and L11 (hereafter the 'study area'), for the time period 1974-1998 (the latest for which complete data are available). Cancers were selected for inclusion based upon the results of the literature search and the level of concern expressed by the local residents. Cancers of interest therefore included: adult lung, liver, leukaemias and lymphomas, larynx, nasopharynx and all sites, and all childhood cancers (subdivided by the categories of leukaemia, lymphoma and others). Consideration was also given to extending the number of sites based on the initial findings e.g. to include kidney and bladder tumours.

The next stage of the investigation was to determine whether the level of selected cancers in the study area was significantly higher when compared with the background (or expected) risk. *Several different ways of assessing background risk may be used here:*

- **Is the spatial distribution of selected cancers greater than would be expected based upon the known population density?** Background population data was obtained from the age sex registers of General Practitioners for this purpose. **Permission was sought and obtained from the Chair of the Local Research and Ethics Committee to use fully postcoded Central Operations Group (COG), and identifiable Cancer Registry data.**
- **Does the distribution of the cancer of interest differ from that of cancers not thought to be associated with incinerator emissions?** This was tested this using adult cancers of the colon and rectum, as examples of cancers not thought to be associated with incinerator emissions.
- **Do the cancer rates in the study area differ from those in other similar areas? We propose to compare the rates for the selected cancers in the study area with those of Liverpool Health District and Merseyside and Cheshire as a whole.** The denominators for the study area calculations will be based on 1981 and 1991 census data, and will thus be able to give only a crude indication of comparative risk.

Data from MCCR and COG were sent to Professor Peter Diggle at Lancaster University for spatial risk analysis. Lancaster University required full postcodes for both cases and controls in order to get accurate grid references.

The dataset from MCCR included, the following data items for selected cancers occurring in the study area between 1974-1998:

- Age
- Gender
- Full postcode
- Superprofile classification (where possible)
- Site code (plus key)
- Date of diagnosis (mm/yy)
- Related factor (plus key)

The dataset from COG specified the numbers of males and females in 5 year age bands for each individual postcode in the areas L9, L10 and L11:

- Age
- Gender
- Full postcode

All relevant cases of cancer (defined by time period of diagnosis, area of residence, and site) registered by the MCCR were included in the study. Controls were assigned a unique study identifier which remained within Liverpool Health Authority. The control population was necessary to form the denominator for the cluster analysis

Lancaster University undertook not to map individuals so as to preserve anonymity and to destroy postcoded data after the analysis was completed. The Medical Statistics Department at Lancaster University completed data protection forms and designated a named person to undertake the analysis and have responsibility for the data.

ENVIRONMENTAL ASSESSMENT

Additional support was sought from Dr Adrian Watson, Senior Lecturer in Geography and Environmental Sciences at Manchester Metropolitan University on plume modelling, from Mr Mike Eastwood, Research Fellow in Environmental Epidemiology at Liverpool John Moores University on historical environmental data and from the Environment Agency NW on records of industrial processes in Fazakerley and Gillmoss. The Atmospheric Research Information Centre at Manchester Metropolitan University were asked to investigate the possibility of using the limited environmental data which exists for the incinerator to produce a plume dispersal model indicating the geographical area covered by the plume.

INCINERATORS AND HEALTH LITERATURE REVIEW

The Chemical Incident Response Service carried out a literature search on cancers which are biologically plausible ie head and neck and lung cancers, childhood lymphomas and possible links to dioxins, furans and incinerators. Currently there is no published systematic review of the literature on these subjects. Whilst, it was not possible to undertake a Cochrane type systematic review, Dr Emer Coffey, on behalf of the Incident Team conducted a overview of the current information to establish the current state of knowledge in this field, identify problems and consistent findings. Supervision on the toxicological material was provided by the Chemical Incident Response Service and Dr Andrew Sewart formerly Dioxins adviser to the Department of Health. Advice was also obtained from Greenpeace UK and Dr Vivian Howard, Senior Lecturer in Fetal Pathology at Liverpool University who very kindly allowed access to his database.

HISTORICAL LITERATURE REVIEW

Dr Ardern made an initial review of the historical records contained in the Medical Officer of Health for Liverpool's Annual Reports for the period 1950 to 1973. These contain relevant information on atmospheric pollution and enforcement of smoke abatement particularly with reference to Crown Property which set the context for the current study. The Annual Reports also refer to research that was being conducted during the Fifties on the possible relationship between lung cancer and atmospheric pollution which was funded by the Medical Research Council and the British Empire Cancer Fund. As a result of this initial review, the Incident Team commissioned Dr Sally Sheard, Medical Historian at Liverpool University to conduct a review of all relevant historical data pertaining to air pollution in Liverpool from both local sources and from material contained in the Public Records Office in London. Dr Ardern and Dr Coffey also interviewed Professor Andrew Semple, formerly the Medical Officer of Health for Liverpool between 1950 and 1974.

PEER-REVIEW

The Incident Team asked Dr Paul Aylin, Clinical Senior Lecturer in Epidemiology and Public Health and Honorary Consultant Public Health Medicine, Department of Epidemiology & Public Health, Imperial College of Medicine at St. Mary's London to conduct a peer-review of the final Incident Report and its findings.

3.1. Preliminary Investigation

PROCESS

During stage 1, complaints about possible effects were collected and collated. It was important to discover whether complaints relate to potential chronic effects (as in this instance). *To establish this, the following questions were addressed:*

- How many people are actually complaining?
- Is it a small group of vulnerable people (e.g. children or older people) that are complaining or the general community?
- How frequent are the complaints?
- How widespread are they compared to the alleged source?
- Do the people complaining have a pre-existing condition that would mean that they might suffer more significant effects than the general population?
- Are there any other plausible explanations for the effects other than environmental exposure? (e.g. migration, lifestyle, social or biological risk factors)
- Is there biological plausibility for the effects that are ascribed to exposure to the emissions?
- Is there consistency amongst the people's complaints?
- Might the range of pollutants/chemicals being released be responsible for the reported symptoms?
- Is there any relevant, reliable environmental data available or easily obtainable?

3.2. Health Profile of Fazakerley

Fazakerley ward is, for the purposes of health service commissioning, in the Alt Valley Primary Care Group. Information from the ward's quality of life profile and from the Annual Report of the Directors of Public Health on Merseyside (Liverpool section) 2000[1] is set out in this sub-section.

POPULATION PROFILE

Table 3.1.Numbers of people (all ages) living in Fazakerley ward in 1999

AGE RANGE	MALES	FEMALES	PERSONS	%WARD POPULATION (PERSONS)
0-4	440	435	875	3.18
5-19	1404	1295	2699	2.84
20-44	2498	2469	4967	2.86
45-64	1301	1451	2752	2.92
65-84	766	1031	1767	2.98
85+	49	163	212	2.87
TOTAL	**6458**	**6844**	**13302**	**2.9**

Source: Small Area Database (1999 Population Estimates)

VITAL STATISTICS

Table 3.2. Total live births and total deaths for 1998 by number for Fazakerley ward.

STATISTICS	NUMBER
Total live births	182
Total male live births	92
Total female live births	90
Total deaths	168
Total male deaths	85
Total female deaths	83

Source: Office of National Statistics

INDEX OF MULTIPLE DEPRIVATION RANKING

The first figure shown indicates the ward's ranking out of 8414 English electoral wards. The figures in brackets indicate the ranking within Liverpool.

Table 3.3. Fazakerley's Deprivation Ranking 2000

SUBJECT	RANKING
Education	538 (16)
Employment	255 (22)
Income	394 (23)

Source: Index of Multiple Deprivation 2000 DEFRA

ENVIRONMENT: AIR QUALITY

The main monitoring station, since 1996, (inlet 3.5 metres high) for Liverpool is within self-contained, air-conditioned housing within a park in central Liverpool. The park is encircled by a busy 2-4 lane urban road some 20 metres from the site. The surrounding area is urban comprising retail shops and businesses. Liverpool's air quality around the city centre has improved over the last 5 years, especially sulphur dioxide, nitrogen dioxide and particulates that are known to have negative impacts on health.

The figures in Table 3.4 in bold denote years above the national targets. All measurements are in micrograms per cubic metre.

Table 3.4. Air Pollution data for Fazakerley for the years 1996 to 2000

POLLUTANT	TARGET	2000	1999	1998	1997	1996
Sulphur dioxide - 1hour mean	<24	0	0	0	4	3
Sulphur dioxide- 24 hour mean	<35	0	0	0	2	0
Sulphur dioxide - 15 minute mean	<35	2	6	5	52	39
Nitrogen dioxide - 1 hour mean	<18	0	0	0	0	0
Nitrogen dioxide - annual mean	40	35	40	38	44	47
Particulates - 24 hour mean	<35	5	10	23	36	48
Particulates- annual mean	<40	24	26	29	32	33
Carbon monoxide - 8 hour running mean	0	0	0	0	0	0
Ozone - daily maximum running 8 hour mean	<10	2	6	3	6	7

Source: UK National Air Quality Archive

HEALTH INDICATORS

The Annual Report of the Director of Public Health for Liverpool 2000[1] has a section describing the electoral ward based social indicators comparing the statistics from 1996 to 1999.

Fazakerley appears in the middle third of wards in Liverpool for most poverty and education indicators. The table 3.5.below shows the 1996 and 1999 ward poverty data set against that of Liverpool.

Table 3.5. Poverty Indicators for Fazakerley compared to Liverpool

POVERTY INDICATOR	FAZAKERLEY 1999	LIVERPOOL 1999	FAZAKERLEY 1996	LIVERPOOL 1996
Council tax (% of households receiving council tax benefit)	46.0	45.7	45.7	Not available
Housing benefit (% of households receiving housing benefit)	31.7	36.7	32.4	Not available
Free school meals (% of children eligible to receive free school meals)	39.7	42.1	36.7	Not available

The table 3.6 below shows the main health indicators for Fazakerley for 2000 (98/99 data) and 1996 compared to Liverpool average. Fazakerley generally is ranked within the middle third of Liverpool wards.

Table 3.6. Health Indicators for Fazakerley compared to Liverpool

HEALTH INDICATOR	FAZAKERLEY 2000	LIVERPOOL 2000	FAZAKERLEY 1996	LIVERPOOL 1996
Emergency admissions (age standardised per 10,000 population - all ages)	1,014	1,059	1,092	981
Standardised Mortality Ratios (for all causes of death for persons aged 0-74 years with 5 year confidence limits-in brackets) 1996-1998	148 (129-167)	143 (139-146)	N/A	N/A
Caries free children (% of 5 year olds with no tooth decay - 97/98 data)	48%	49%	N/A	N/A

Note: The Standardised Mortality Ratio for England and Wales is 100. Any result where 100 is not between the lower and upper confidence level can be regarded as significant.

3.3. Specific Local Health Concerns

Currently in the UK, one third of all adults will die from some form of cancer. Thus cancers as a group of diseases are common. The investigation into the Fazakerley and Gillmoss concerns had to take account of the fact that Liverpool as a whole has high death rates from cancer. Over the period 1996-1998 the average SMR for Liverpool for a common cancer such as lung cancer was in excess of 200 (persons) with the worst SMRs in the most socio-economically deprived wards. Vauxhall had the highest SMR for that period of 381. The high death rates for lung cancer were matched by high death rates for other diseases such as heart disease, chronic obstructive pulmonary disease and stroke associated with the 2 major risk factors poverty and cigarette smoking.

Twenty-five cases, and 19 deaths from cancer in individuals residing in Haven Road and Longmoor Lane (Liverpool 9), were initially reported by local residents as occurring over a 26-year period from 1974 to 1st Jan 2000. The cancers reported are of different types. The main concern of the residents has been that there is a perceived problem with "throat" cancer. Although, clearly, this description might apply to a number of different cancer sites. The majority of deaths were in people over the age of 50 years. The residents concern was that there are 5 streets on the Formosa Drive estate including Haven Road where cancer deaths are perceived by local residents to be higher than might be expected.

One case was recruited into a national study on childhood cancer in the early 1990s. As part of the research for this study, environmental monitoring for radon gas and electromagnetic radiation was conducted in both the home of the case and other relevant locations. The Incident Team obtained the results of this monitoring from the principal scientific investigator, Professor Cartwright at Leeds University. The results for the locations studied were normal.

3.4. Preliminary Environmental Findings

Environmental data were problematic. Firstly, Fazakerley was an area that had a number of different industrial processes operating concurrently with or pre-dating the operating period of the hospital incinerator. These included a signal works and an iron foundry.

- **The Incident team received information from the Team Leader for Processes in Industry regulation at the Environment Agency North West that there were no major industrial processes that were licensed by either the Environment Agency or its predecessor, HM Inspectorate of Pollution in Fazakerley and Gillmoss between the years 1970 and 1999.**
- **Secondly, Fazakerley and Gillmoss are heavily trafficked areas. Vehicle movements per day have been rapidly increasing since the 1970s and road traffic is a major source of particulates and volatile organic compounds (especially benzene) which are potential or, in the case of benzene, known carcinogen.**
- **Thirdly, historically, very little environmental monitoring was carried out in Fazakerley as up until 1985 there were only 3 monitoring stations for the whole of Liverpool and none was sited in the potential area to be covered by the study. Smoke -controlled areas were being established in Liverpool from the 1950s onwards and the programme was not completed until the 1980s. From the Medical Officer of Health's annual reports for the period, the intention was to make Fazakerley and Gillmoss smokeless zones by 1974, but this was deferred due to lack of finances. Therefore, this means that in 1970 there would be still a significant number of houses burning coal but no monitoring of particulates. Current particulate monitoring introduced 6 years ago is reliable and accurate, but only covers the period after the incinerator ceased operation.**

3.5.The Incinerators at Fazakerley

Preliminary inspection of the Medical Officer of Health's (MOH) Annual Reports between the period 1950 to 1973 (after which the post of MOH ceased and there was a fifteen year gap until Public Health Annual Reports were reintroduced with the creation of Director of Public Health) reveal a continuing concern with smoke abatement and atmospheric pollution in Liverpool. The major sources of pollution were the Clarence Dock Power Station, steam railway engines and the boilers and incinerators of Crown Properties with particular reference to the City's hospitals. With the last mentioned there appeared to be a number of reasons for this, poor working practices, insufficient management and out of date equipment. Crown Immunity meant however that the environment health officers had no powers of enforcement - a situation that did not change until the early 1990s when Crown Immunity was removed. The Annual Reports document numerous complaints from both the Medical Officer of Health and the Local Authority's Health Committee to Ministers of Health regarding the nuisance caused by hospital boilers and incinerators. Unfortunately the Annual Reports rarely document in detail specific incidents involving specific hospital sites - for instance, Fazakerley is not singled out. Most concern appears to be focused on the hospitals in the City centre.

Mr Laird former manager of the incinerator at Aintree University Hospital Trust provided the Incident Team with a report on incineration at the Fazakerley site between the period 1974 until the incinerator's closure in 1995.(2) The original simple incinerator was replaced in 1985 with a two stage pyrolytic process that was further upgraded to comply with interim emission standards in 1992. The incinerator was shut down in 1995 because it was unsuitable for further modification to meet the standards introduced under the Environment Protection Act. The incinerator was primarily used for clinical waste and confidential paper. This also included small amounts of very low level radioactive waste Carbon 14 from the BacTec laboratory process in the Public Health Laboratory at Fazakerley. The Laboratory has detailed records of the quantities involved. However, between 1989 and 1995, the hospital was incinerating material from other sources such as tobacco waste from Imperial Tobacco and materials (believed to be confiscated goods such as videos, illicit drugs and tobacco for HMS Customs and the Police). The hospital was not told the nature of these materials for reasons of public security but provided the hospital with a transfer note certifying that each load was safe to incinerate. It was established that HMS Customs are unlikely to have any detailed records of the nature of the goods destroyed.

The incinerator complied with all statutory regulations in place at the time and annual particulate emissions levels for 1992 to 1995 showed that the incinerator operated well within the interim guidance limits issued by the Department of Environment. However, in line with the rest of the UK, emission monitoring only covered smoke and solid particulate material. Dioxin and furan monitoring was not part of the statutory framework. Indeed limits for dioxins are set by the Environment Agency and apply to incinerators built since 1996 even though this is currently not required under the EU Municipal Waste Incinerator Directive. Most municipal waste incinerators are authorised or achieve 0.1ng/m^3 of dioxins and continuous or extractive monitoring used to demonstrate compliance.

3.6. Preliminary Toxicological Findings

The health impacts from waste incinerators are not well understood. The most complete studies carried out in the UK by Gatrell and Diggle of Lancaster University and Elliot of the Small Area Health Statistics Unit in London suggest that although initial investigation might reveal an excess of cancer deaths around incinerator sites that there is a strong association with socio-economic deprivation and that there is no evidence of decreasing risk with distance from the point source. However, the published research is problematic and results are inconclusive and inconsistent. Other studies have shown an association and increased risk particular types of cancer. The cancers that most often cited and for which there would appear to be a biological plausibility are laryngeal cancers, lung cancer, head and neck cancer and possibly some types of childhood cancers mainly leukaemia. Currently, that there is no published systematic review of the literature of waste incineration and cancer incidence. Most of the toxicological research on dioxins and cancer has been conducted on animal models but results have caused the US Environment Protection Agency (EPA) to request that dioxins are recognised as human carcinogens. No decision has been made on this yet and the UK Department of Health is considering the EPA's report.

4 HISTORICAL REVIEW OF AIRBORNE POLLUTION IN LIVERPOOL 1950-2000, WITH REFERENCE TO THE DEVELOPMENT OF THE FORMOSA DRIVE ESTATE, FAZAKERLEY

4.1. Methods and Sources

A variety of sources have been located for this review, both within Liverpool and nationally. The provision of historical context for contemporary issues is always limited by the survival of material.

THE ANNUAL REPORTS OF THE MEDICAL OFFICER OF HEALTH FOR LIVERPOOL (1950-1974).

The Medical Officer of Health (MOH) provided professional guidance to the city council on all matters relating to public health and wider environmental health concerns. The annual reports provide a range of useful statistics including air pollution monitoring data and the number of inspections and prosecutions made by the environmental health officers. The annual reports also document the introduction of measures under the 1956 Clean Air Act, specifically the creation of Smokeless Zones in parts of Liverpool from 1958.

ARCHIVE PAPERS FROM LIVERPOOL CITY COUNCIL COMMITTEES.

The MOH reported to the Health Committee of the City Council (352 MIN/HEA). The history of the development of public health services is illustrated in the transition of the committee systems. It reflects the split of health and environmental functions between the local authority and, after 1974, the Mersey Regional Health Authority. Between 1970 and 1974 a separate Environmental Health and Protection Committee functioned within the city council (352 MIN/ENV). This committee inherited responsibility for the introduction of Smokeless Zones. The committee minutes and reports have been scrutinsed and have provided useful commentary on the development of policy and practice on air pollution issues in Liverpool between 1970 and 1974. There do not appear to be any papers deposited yet at the Liverpool Record Office for the post 1974 period.

The Liverpool Record Office also operated a thematic newspaper cuttings service until it was curtailed by staff shortages in the 1980s. Comprehensive coverage of the introduction of Smokeless Zones between 1958 and the 1980s is to be found in the volume on air pollution (Hq 614.71 CUT).

THE PUBLIC RECORD OFFICE

The Public Record Office at Kew, London, holds all the official archives for government departments and advisory bodies. Official papers are subject to the 30-year closure rule. Therefore only papers dated before 1972 can be seen.
The following archives were identified as potentially useful for this research:

- Medical Research Council Air Pollution Unit. Papers from 1940s to 1974 (FD 1-23). The MRC began to take interest in air pollution particularly in relation to chronic bronchitis in the early 1950s. The Air Pollution Unit was established in 1955. It frequently served as an advisor to the Ministry of Health on pollution issues, in addition to providing a monitoring service for pollution levels in London.
- Ministry of Health (MH 166). This includes material on hospital building and management. These files were inspected for specific Liverpool references, but there was nothing that related to Fazakerley hospital. In 1968 a Committee of Inquiry was formed into Hospital Building Maintenance. This produced the Woodbine Parish Report in 1970. These papers, and others relating to hospital boiler plant installation and maintenance are closed until 01.01.06 (MH 166/627-698).
- Alkali Inspectorate (BT328). The Alkali Inspectorate, formed in 1863, acquired a wide range of duties relating to monitoring of industrial processes, with an emphasis on anti-pollution measures. It came under the various government health departments, known as Her Majesty's Inspectorate of Pollution (HMIP) from 1983 to 1987. In 1987 it became part of the Department of the Environment and, from 1996, part of the Environment Agency. The Alkali Order of 1958 placed all major heavy industries which emitted smoke, grit, dust and fumes under the supervision of the Inspectorate. Only one Register was found, which detailed premises in Liverpool in 1953. It is indicative of the general low-level of heavy industry in Liverpool in the 1950s. The Annual Reports of the Alkali Inspectorate from 1969 and the District Inspection files from 1981 are closed until 01.01.05 and 01.01.12 respectively.
- Warren Springs Laboratory (WSL). This was founded in Stevenage by the Department of Scientific and Industrial Research in 1958. It provided testing facilities and research on air pollution issues, particularly for HMIP, until its closure in 1994. There were a number of working parties on chimney heights and the dispersal of chimney gasses. The reports of WSL investigations were examined over the period 1958 to 1972, but there were no references to work on Merseyside (FV 12). WSL also produced the National Survey of Air Pollution 1961-71 (HMSO, 1972).
- Ministry of Housing and Local Government. Air Pollution and Smoke Abatement Files (HLG 55). Papers from the 1950s on the introduction of clean air legislation. Liverpool occurs occasionally in these papers,
- Royal Commission on Environmental Pollution 1974-76 (BT 328). Reports and correspondence relating to this are closed until 2007.

4.2. Historical Review of Air Pollution in Liverpool

Liverpool has been one of the most progressive urban areas in the development and implementation of public health policies. In part, this was in recognition of the fact that the town was one of the most 'insanitary' in early nineteenth century Britain. The quality of the local atmosphere has thus always been an integral component in the public health strategy of Liverpool City Council. The legislation through which air pollution has been monitored and controlled has developed from the mid-nineteenth century Nuisance Acts, through to more specific national legislation within Public Health Acts. The West Lancashire and Cheshire Smoke Abatement Committee, formed in connection with the Public Health Act 1936, provided a useful forum for discussion of local air pollution problems, supplementing the official local government investigations.

As in many large urban areas, Liverpool experienced increasingly severe 'smogs' in the first half of the twentieth century. Poor winter atmospheric conditions were exacerbated by smoke from domestic fires, shipping on the Mersey and assorted industrial activities. In 1937 a national Standing Conference on atmospheric pollution was formed. Liverpool Corporation was a member (along with 41 other municipal authorities), paying an annual subscription of £50, and sending two representatives to the annual meeting. The Standing conference members collected evidence from urban areas using deposit gauges, automatic filters and sulphur gas detectors. Professor W.H. Roberts, Liverpool City Analyst, was chairman of the Standing Conference between 1940 and 1943. Representatives from the Standing Conference were appointed to the Government Committee on Air Pollution (the Beaver Committee) formed in 1953. Liverpool was thus involved at first hand in the development of national legislation on air pollution.

By the late 1940s, Liverpool resumed its active interest in smoke abatement, which had been halted during the Second World War. The Public Health department arranged for two films on correct boiler and furnace operation to be shown to boiler stokers and firemen in the city. The Department now operated three standard deposit gauges and one Owen Automatic Air Filter to monitor pollution levels in the city. The 1955 Liverpool Corporation Act included a new code of conduct on the installation of approved furnaces.

Dr Frazer retired as Medical Officer of Health in 1953 and was replaced by Professor Andrew Semple, who served until 1974. Semple had a strong professional interest in air pollution and in 1952 he was appointed to the Medical Research Council committee to investigate the effects of atmospheric pollution on lung cancer. In 1955 he persuaded the City Council to hire an aircraft to fly above the Clarence Dock Power Station (coal-fired) to prove that the emissions were not just water vapour, but contained dust and sulphuric acid. Under Semple's leadership, Liverpool applied for the first Smokeless Zone using the 1956 Clean Air Act, and this was finally introduced to cover the city centre on 1 April 1958. Liverpool made significant progress towards improving air quality through its selective approach to smokeless zones. However, there were delays which were outside the control of the MOH and the City Council. The biggest planned zone – number 10 – which covered 17,000 acres of Aigburth, Allerton, Speke and Woolton was approved in 1960 but not implemented until 1963 because of shortages of smokeless fuel. The high cost of suitable fuel made the zones a politically sensitive issue, and the final target of a citywide scheme which had initially been planned for 1965, was not achieved until the early 1980s.

Fazakerley was one of the last parts of Liverpool to be designated a smokeless zone. The Smoke Control Order Number 26 was approved in 1971, but not completed until 1974.

In addition to implementing a smokeless zone policy – arguably one of the most efficient ways to combat the main culprit in Liverpool's air pollution problem – the domestic chimney – the City Council also actively pursued other lines of attack. Responsibility for monitoring the notorious Clarence Dock power station was transferred from the Alkali Inspectorate to Liverpool City Council (Public Health Department) under the Liverpool (Alkali Works) Order, 1960. Liverpool had specifically asked for this transfer under section 17 (1) of the 1956 Clean Air Act **(PRO archives BT 328/118)**. Liverpool's forms for approval of new furnaces were also adopted nationally for use under section 3 of the 1956 Act.

The Annual reports of the Medical Officer of Health, together with the minutes of the Health Committee and the Environmental Health and Protection Committee (EHPC) provide valuable evidence of the ongoing professional interest in air pollution in Liverpool. We conducted a review of selected annual reports of the MOH between 1956 and 1973. The Health Department of the City Council maintained a number of monitoring sites in different parts of the urban area. The data were used in setting smoke reduction targets and also forwarded to Warren Spring Laboratories for use in their regional surveys. In 1970 the EHPC authorised the purchase of five additional sets of equipment for pollution monitoring. Officers from the department were also delegated to attend various national and international conferences in pollution. Professor Semple (MOH 1954-74) noted in interview that he placed great emphasis on the staff of the Public Health Department maintaining their professional skills. Inspectors appear to have been able to give correct advice on furnace operation, chimney design and other issues relating to air pollution. The emphasis during this period (late 1970s) was very much on visible air pollution (smoke) rather than invisible gas emissions.

AIR POLLUTION FROM CROWN PROPERTIES

Under the 1936 Public Health Act, Crown Properties (which included local authority hospitals) were exempt from prosecution for nuisances, including air pollution. However, by the 1950s, with the increasing size of hospitals requiring additional boilers and incinerators, hospital air pollution was becoming a more pressing issue and the appropriate Ministers of State were approached to find solutions.
The Annual Report of the MOH for 1956 makes the following comment:

> *'There has been considerable smoke nuisance arising from the boiler chimneys at many of the hospitals in the city, giving rise to frequent complaints from the inhabitants of the premises in the neighbourhood of the various hospitals. As a result of a series of observations on the known offenders, it was established that a considerable smoke and grit nuisance existed at a number of hospitals….The Minister replied that a comprehensive scheme was in hand for the erection of new boiler houses at the various hospitals, and, in one case, it would take two-and-a-half years to complete the installation, although no date of commencement was given.' (pp182-83)*

In subsequent annual reports, Professor Semple notes that the Crown properties are generally amenable to advice on issues such as correct furnace operation. Evidence from the EHPC suggests that inspectors were closely involved in the planning for a number of hospital developments. See, for example, a report in 1973 on discussions with the Liverpool Regional Hospital Board for the extension of Sefton General Hospital **(Liverpool Record Office H 352 ENV 1/4 14.2.1973 minute 267)**.

The National Survey of Air Pollution, published in 1972, provided a detailed assessment of the North West region. Its main conclusion was that the region was one of the most polluted in Britain during the decade 1961-71, partly explained by the higher domestic coal consumption per head than in any other region. There had however, been reductions in rates of both smoke emissions and sulphur dioxide concentrations from the early 1960s. We have not been able to locate any local or national surveys of air pollution for the 1970s and 1980s.

4.3. Interview with Professor Andrew Semple, Medical Officer of Health for Liverpool 1953-1974.

The following are extracts from an interview conducted by Dr. Ardern and Dr Coffey with Professor Andrew Semple who was formerly the Medical Officer of Health for Liverpool between 1953 and 1974.

ON AIR POLLUTION IN LIVERPOOL IN 1950S

Professor Semple explained that air pollution problems in Liverpool were not a recent event but that there had been bad smogs in Liverpool in the 1950s.

> *"We had the Clean Air Act as a result of the 1950 smog in London which was a terrible thing, it was bad enough in Liverpool. I attended a meeting in town and when I came out, I drove my car into the Medical Institution car park and walked home here from Mount Pleasant because everything was stopped, buses and everything. Of course we had quite an influx of respiratory illness that we had to cope with.   A lot of people think it only happened in London - it certainly happened in Liverpool and it happened in Manchester too and one of the things that interested me was that people accepted this situation - these terrible Winter fogs. When I came for my interview in 1948 to Liverpool I was appalled walking into Dale Street because all these buildings were black - St George's Hall was black. Unfortunately, I wish then I'd taken photographs of them because, when I went into the Municipal Annexe in Dale Street, I was appalled at the blackness of the buildings."*

ON HOSPITALS AND COMPLIANCE WITH THE CLEAN AIR ACT IN 1950S

Professor Semple had considerable difficulty in getting NHS hospitals to comply with the Clean Act because the hospital boards were not obliged to do anything under the terms of Crown Immunity. Fazakerley at that time was an Infectious Diseases Hospital and it had been an old Corporation hospital - a municipal fever hospital and sanatorium. The two superintendents of the sanatorium were interested in the issue and tried to comply with the Clean Act . The hospital engineer at Walton Hospital was also interested and some of workers underwent local training on the correct management of incinerators and boilers. Professor Semple believed that Fazakerley Hospital put quite a lot of effort into overcoming atmospheric pollution.

ON AIR POLLUTION RESEARCH IN LIVERPOOL IN 1950S

Professor Semple and colleagues were involved in some research into air pollution and lung cancer cases in Liverpool in the 1950s funded by the MRC and the British Empire Cancer Fund. The primary focus of interest was in the Mersey tunnel and seeing whether the people who used it were more at risk It was hypothesised that pollution in the tunnel might be a factor and they took readings in the tunnel and watched tunnel users but found no connection.

What they did find was that Liverpool had the highest lung cancer rate in the country. Professor Semple believed that Liverpool's main industry of shipping was a factor.

> *"Liverpool was a sailor town and of course sailors were heavy smokers because they got them cheap - they were getting them Duty free on the ships. But talking to the chest surgeons at Broadgreen, they said that they saw as many lung cancer cases from the mining towns of Wigan and Widnes." But, once again, it was obvious it was cigarette smoking much more than atmospheric pollution because miners - as soon as they got out of the pit (because obviously they couldn't smoke in the pit) would light up a cigarette. Doll did the studies with the doctors showing that smokers had a higher incidence of lung cancer. Then of course it became obvious that Liverpool as a sailor town had large numbers of heavy smokers and, similarly, in the pit towns of Wigan and Widnes, so that explained that smoking was the main factor."*

Professor Semple observed that during the 1950s (unlike now) it was very unusual for women to contract lung cancer because prior to the Second World War very few women smoked.

> *"In 1950 in Broadgreen Hospital, they had a ward that always had about 5 or 6 men in with lung cancer. But, when they got a woman, they never knew what to do with her - they had to put her in a side room because women didn't smoke until the Second World War. "*

4.4. Development of the Fazakerley District of Liverpool

Liverpool's rapid growth in the nineteenth century was linked to the prosperity of the port. By 1911 the connurbation had a population of 1.17 million. The physical expansion of the urban area was facilitated by local authority investment in infrastructure, in particular the provision of suburban planning. The inter-war period witnessed increasing suburbanisation through the development of commercial and municipal housing schemes, the creation of new industrial zones and the systematic clearance of slum property in the city centre.

Liverpool City Council had built the first municipal houses in Britain in the 1870s. The city was committed to the provision of affordable housing, which also enabled the city centre slums to be demolished. The Fazakerley Estate was constructed in four phases by the Corporation between 1919 and 1945. Phase one was constructed under the 1919 Housing and Town Planning Act. The estate (originally called the Fazakerley Hall Estate) initially consisted of 62 non-parlour and 150 parlour houses (parlour houses, as the name suggests, had five rooms and better equipped, therefore more expensive to rent). The analysis of corporation housing between 1869 and 1945 conducted by Pooley and Irish provides socio-economic data on the occupants of the Fazakerley Estate during the period to 1945. It found that 52% of household heads in non-parlour households were engaged in skilled manual work (45% for parlour houses).

The Fazakerley Estate was further extended under the 1924 Housing (Financial Provisions) Act. An additional 1,228 houses were constructed, 968 non-parlour and 260 with parlour. The emphasis within the estate was therefore towards cheaper rental property for manual labour. The Estate was equipped with a small shopping centre, public hall, library, school and church. In 1930, 351 parlour and 930 non-parlour houses were added, and in by 1945 a further 410 parlour and 1030 non-parlour houses were constructed **(C.G. Pooley and S. Irish, The Development of Corporation Housing in Liverpool 1869-1945, Centre For North West Regional Studies Resource Paper, 1984, p.81).**

The demographic and economic structure of Liverpool changed considerably from the 1930s. The decline in port activities had repercussions for other aspects of the Merseyside economy. The population, which had been shifting outwards from the old core, began to show an absolute decline, with high levels of out-migration beyond the city boundaries to Knowsley, Kirby, St Helens and further afield. The City Council attempted to foster new economic activity – some of it in the vicinity of the Fazakerley Estate. In 1936 the Aintree Estate was founded under the 1936 Liverpool Corporation Act. It was primarily reserved for 'clean air' trades – including food processing and light engineering. In 1941 a wartime Ordnance factory was constructed at Fazakerley, adjacent to the corporation's estate. This was released for civilian use in 1961 and taken over by Joseph Lucas Ltd. By the 1970s there were 30 firms housed on the Aintree Estate, with some 20,000 employees **(J.A. Patmore and A.G. Hodgkiss (eds.), Merseyside in Maps, Longman, 1970, p41)**.

Conditions within the Fazakerley Estate began to decline from the 1950s. The Medical Officer of Health noted high levels of overcrowding in some of the council houses. As the economic recession grew deeper, Merseyside was designated as a 'Development Area' in 1949 by the Board of Trade. This generated some funds, but could not stimulate a full regeneration. Levels of unemployment increased and areas such as Fazakerley experienced proportionately higher levels of deprivation, exacerbated by the city council's problems in maintaining infrastructure and estate services such as public transport.

In 1968 the City Council commissioned the Liverpool Social Malaise Study, using census material to identify the most deprived parts of the city. This was followed in 1971 by a more rigorous Social Area Analysis which was undertaken by Hugh Wilson and Lewis Womesley and published as the **Liverpool Inner Area Study by the Department of the Environment in 1977**. This used a cluster analysis technique to identify similar socio-economic areas within Liverpool which were not necessarily geographically located in the same place. Fazakerley was placed within cluster 18 (there were a total of 25 clusters in the analysis), along with other outer council estates such as Cantril Farm, Norris Green and Speke. *A similar study by* **R.J. Webber, published as the Liverpool Social Area Study by the Centre for Environmental Studies in 1975** also placed Fazakerley in the same cluster. *The main findings for this cluster include:*

- 91% council tenants
- 41% of male occupants classed as skilled manual, 25% as semi-skilled
- Higher levels of economic deprivation than other outer council estates
- Largest incidence of educational sub-normality
- Largest incidence of children in care
- Largest incidence of supervision orders
- Largest incidence of possession orders
- Very high proportion of school-age children
- Very high proportion of 'very large' families
- Low rates of economic activity among married women
- High dependence on public transport for journeys to work
- Employment structure strongly oriented towards manufacturing industries
- Largest proportion of seriously overcrowded households
- Greatest disadvantage for accessibility – to work, shops, open spaces
- Highest rates of unemployed men of all the outer council estates

The Fazakerley Estate, as part of cluster 18 of outer council estates, was located on the very edge of the Liverpool Inner Urban Area. Under the Inner Urban Areas Act of 1978, three tiers of priority were created within 'designated districts' and Fazakerley came within the boundary of the Liverpool Special Partnership Area. The complexities of urban regeneration were increased in 1981 by further measures to stimulate economic and environmental regeneration, with the creation of the Merseyside Development Corporation (MDC) and the designation of two Enterprise Zones. Additional information on the more recent socio-economic status of the Fazakerley district can be obtained from the city council, and from the reports of the MDC.

5.1. Development of Air Pollution Control In The UK With Special Reference To Clinical Waste Incineration

ROLE OF LEGISLATION

The development of air pollution control in the UK, while no doubt driven by concerns about health, is essentially pragmatic, though the endorsement of (health derived) air quality standards (AQS) and the increasing use of health based risk assessments for new processes does suggest a recent change in approach.

The main air pollution legislation in the UK is shown in Table 5.[1]

Table 5.1: Air Pollution Legislation

Alkali etc Works Regulation Act 1906
Clean Air Acts 1956 and 1968
Control of Pollution Act 1974
Environmental Protection Act (EPA) 1990
Environment Act 1995
Pollution Prevention and Control Act 1999

It is instructive to consider the influences which led to the introduction in the legislation. In the middle of the 19th century there was severe pollution around industrial plants manufacturing alkalis e.g. NaOH. The Alkali Act of 1863 required emissions from such plants to be reduced by 95%; this act was extended to cover other processes to become the Alkali etc Works Regulation Act 1906, which was eventually superseded by the Control of Pollution Act 1974, and in turn by the Environmental Protection Act 1990. All these acts apply to the most potentially polluting or technologically complex processes (though both the Control of Pollution Act and EPA also apply to other sources of air pollution) which today covers around 2000 processes (termed scheduled processes). Pollution from these scheduled processes is enforced by the Environment Agency (since 1995), which subsumed Her Majesty's Inspectorate of Pollution (HMIP); HMIP (originally called the Alkali Inspectorate) had previously been under control of both the Department of Environment and the Health and Safety Executive. Initially HMIP were only responsible for air pollution from scheduled processes. However, following a report [2] of the Royal Commission on Environmental Pollution (RCEP), they now also regulate emissions to land and water from these processes, termed Integrated Pollution Control (IPC).

THE CLEAN AIR ACT 1956

The development of air pollution legislation has also been strongly influenced by the London smogs, in particular that of 1952 in which it is estimated that nearly 4000 people died as a result. The Beaver committee on air pollution was set up as a result of this incident with the Clean Air Act 1956 a result of its deliberations. The Clean Air Act 1956 granted local authorities the power to designate smoke control areas, including government grants to help reduce the costs of installations smoke-free heating systems. The Clean Air Act 1968 was passed to allow central government to force local government to introduce smoke free areas where there was regional resistance e.g. both monetary and political.

The Clean Air Acts were not only concerned with domestic smoke emissions but also industrial emissions from processes not covered i.e. non-scheduled processes, by the Alkali and subsequent acts. For non-scheduled combustion processes (presumably including waste incineration) the Clean Air Acts granted local authorities the right to require a minimum chimney height (following guidance issued by the Secretary of State but prepared by HMIP) so as to ensure that smoke (defined as dark smoke and black smoke measured using the Ringelmann chart), grit (>76 mm), dusts (1 - 76 mm) or fumes (<1 mm) emitted were not prejudicial to health or a nuisance. The Clean Air Acts also gave local authorities the power to require operators to use bpm to minimise grit and dust emissions (but not fumes) and require operators to monitor their emissions of grit and dust. However, because the Clean Air Acts refer specifically only to smoke, grit, dust and fume particles these controls were not considered as comprehensive as those applied to scheduled processes[3]. For non-scheduled processes not involving combustion processes there was no power of prior approval to reduce or limit emissions - the only powers available to deal with pollution incidents were those under the Public Health Acts 1936 and 1961[2].

RECENT LEGISLATION

- **The Control of Pollution Act 1974 was considered, with regards to air pollution, a codifying measure.**
- **The Environmental Protection Act (EPA) 1990 redefined the roles of HMIP and local authorities. Industrial processes were (and still are) assigned Schedule A (HMIP control) or Schedule B (local authority control) classification. While this system somewhat replaces the scheduled and non-scheduled system, it resulted in a net transfer of premises to local authority control. Schedule A processes are subject to Integrated Pollution Control (IPC), which as mentioned above is a more holistic approach to pollution control. A separate regime for controlling emissions to air alone is applied to schedule B processes termed Local Air pollution Control (LAPC).**

INTERPRETATION AND IMPLEMENTATION OF AIR POLLUTION LEGISLATION

What has not changed is the pragmatic approach adopted in regulating emissions. Operators of scheduled/non-scheduled (A or B) processes must apply *best practicable means (bpm)* to prevent or limit emissions of specified pollutants. *For example, the 1906 Alkali Act states:*

> ***"The best practicable means for preventing the escape of noxious or offensive gases by the exit flue of any apparatus used in the work, and for preventing the discharge, whether directly or indirectly, of such gases into the atmosphere, and for rendering such gases where discharged harmless and inoffensive."***

Neither the Alkali Act nor any subsequent act defines bpm though it is influenced by **local, economic** and **technological** factors. More recently an additional factor i.e. current knowledge of the effects of pollutants, has been added[3]. Under EPA 1990 the term *best available techniques not entailing excessive costs (BATNEEC)* has replaced *bpm*. Advice on *BATNEEC* is provided by both HMIP[4] and Secretary of State[5] Process Guidance (PG) notes (the latter are for local authority guidance). HMIP provide guidance in the formulation of both sets of PG notes, which promotes continuity in air pollution control between central and local control[2].

This bpm/BATNEEC approach has been criticised in that the approach is somewhat subjective and may not result in the setting of strict emission limits by HMIP, no doubt compounded by the close working relationship between HMIP and the industry it regulates leading to what some consider to be inadequate enforcement and prosecutions. It is worth noting that the RCEP has considered the merits of the *bmp/BATNEEC* approach and, with some caveats (such as wider consultation on *bpm/BATNEEC*, which is now the case), supports it[2].

Under EPA 1990 operators of processes apply for an authorisation to either HMIP or the local authority, which if granted sets conditions for the operation of the process so as to control potential air pollution. PG notes set out the factors that need to be considered, though it is up to HMIP/local authority to interpret the *BATNEEC* concept and decide what standards a plant should achieve. These authorisations are reviewed every four years, if necessary taking into account changes to the relevant PG note. PG notes are also subject to review. For example LAPC PG notes have already undergone their first 4 year review[7]. This approach follows the recommendations made by the RCEP as early as 1976[2] who were of the view that there was considerable room for improvement in the current regulatory framework, in particular that local authorities had much less regulatory control over non-domestic sources of pollution than the then Alkali Inspectorate, exacerbated by the often lack of technical expertise within the local authority and the wider remit (other than air pollution) of its public health officers. This issue about technical expertise was the subject of discussion in the RCEP 4th report[6]. Whether, and to what extent, this disparity was rectified prior to the EPA 1990 is unclear.

PUBLIC INFORMATION ON AIR POLLUTION FROM INDUSTRIAL PROCESSES

As part of their authorisation, both local authorities and HMIP require monitoring by the operators to demonstrate compliance with the conditions of the authorisation. Limited public access to the HMIP data was a previous criticism of the RCEP; this was rectified under EPA 1990 with emission data from A and B processes being held in a public register obtainable from the local authority[4].

In 1972 Warren Spring Laboratories published its national survey of air pollution[7] collected from 1200 sites nationally between 1961-1971, operated mainly by local authorities. Smoke and SO2 were the main pollutants monitored, showing a sharp decline in both smoke emissions and ground level concentrations with reductions in SO_2 less striking. The current UK air monitoring programme covers a far more comprehensive range of pollutants including toxic organic micropollutants (TOMPs) e.g. dioxins. This clearly reflects changing knowledge about the health effects of pollutants. Concern was initially directed at smoke and SO_2 concentrations (i.e. the products of coal burning), however, as these pollutants have been reduced attention has switched to other pollutants e.g. PM_{10}, VOCs, dioxins. With regards to air pollution control this problem of a moving target is not new (and is unlikely to go away, hence the addition of *current knowledge of the effects of pollutants* in deriving BATNEEC) and was noted by the RCEP[2] in 1976:

> ***"…people's standards seem to rise as pollution lessens so that, from the enforcing authorities viewpoint, expectations are always running ahead of what can be done (para 123)"***

LEGISLATION ON CLINICAL WASTE INCINERATION

Prior to the EPA 1990, HMIP only regulated chemical waste incineration plants; other forms of waste incineration were non-scheduled and were controlled by local authorities. Furthermore, up to 1991, when the exemption was removed[8] following a recommendation by the RCEP[9], **clinical waste incinerators operated by the NHS were exempt from pollution control** because they were regarded as being operated by the Crown Immunity. Under EPA 1990 waste incineration is now controlled by either IPC or LAPC depending on its rating; >1 tonne/hour is subject to IPC, < 1 tonne/hour to LAPC.Table 2 presents the emission standards (which are considered as indicative of *BATNEEC*) for specified pollutants described in HMIP and Secretary of State PG notes for clinical waste incinerators.[10]

Table 5.2: Standards for emissions to air from clinical waste incinerators

POLLUTANT	SCHEDULE A STANDARDS mg/Nm^3	SCHEDULE B STANDARDS mg/Nm^3	PRE-1985 EMISSIONS mg/Nm^3	POST-1985 EMISSIONS mg/Nm^3	MWI[d] EMISSIONS mg/Nm^3
Total particulate matter	30	100	210	140	500
Carbon monoxide	100	100	600	210	220
VOCs	20	20	NA[c]	14	NA
SO_2	300	300	60	16	340
HCl	30	100	460	23	690
hydrogen fluoride	2	-	NA	0.44	1.1
NOx	350	-	160	120	NA
Cadmium	0.1	-	0.7	0.65	0.6
Mercury	0.1	-	0.08	0.82	0.26
others	1[a]	5[b]	10	5	15
dioxins (ng TEQ/Nm3)	1	-	20	4.4	45

a Total of arsenic, chromium, copper, lead, managenese, nickle, and tin
b Total of all metals above except tin but plus cadmium and mercury
c data not available
d Municipal waste incinerator emissions

The standards set by the Secretary of State for smaller incinerators are less stringent than for those regulated by HMIP reflecting their smaller pollution load and economic constraints.

It was estimated that in 1991 there were as many as 700-800 clinical waste incinerators operating in the UK and that the annual throughput was in the range 300,000-400,000 tonnes. These were nearly all owned by NHS hospitals/trusts and located in hospital grounds.[10] The majority of these incinerators were unable to meet the new standards reported in Table 5.2 and so closed. Table 5.2 compares these standards to emissions measured in two pre-1985 and three post 1985 clinical waste incinerators (it does not appear that 1985 has any regulatory significance). Mean emissions from 20 municipal waste incinerators (MWIs) operating before the introduction of the new emission limits is shown alongside for comparison. This indicates that for these pollutants emissions from clinical waste incinerators were comparable/less-than emissions from the local authority regulated MWIs.

As part of its review of waste incineration in 1993 the RCEP[10] stated that **"the present proliferation of small and inadequately staffed plants for clinical waste should be replaced by a smaller number of large plants with expert advice"**.

INCORPORATION OF HEALTH CONCERNS INTO CURRENT AIR POLLUTION LEGISLATION

What should be clear from the above is that air pollution control in the UK has been built up stage by stage in response to particular, primarily health-related, problems. Its development has been essentially pragmatic with the regulator working closely with the regulated to prevent/minimise emissions taking into account local, economic, and technological factors. An improvement in health has been an implicit assumption throughout this development, though any assumed improvement has been qualitative rather than quantitative.

Recently there has been a change in the emphasis. Air Quality Standards (AQS) form an integral part of the recent UK National Air Quality Strategy.[10] AQS are objective health based standards i.e. concentrations of specific pollutants in air that are not considered a risk to health. The Strategy sets out AQS for eight main air pollutants and objectives for their achievement throughout the UK by 2005. However, these AQS will not replace the existing IPC/LAPC (which has now been replaced by the new Pollution Prevention and Control [PPC] regime that implements EC directive 96/610); rather it is anticipated that PPC and other relevant legislation will achieve these health-based standards.

While not a regulatory requirement it is becoming more commonplace for applicants as part of the process of seeking an authorisation to carry out a health based risk assessment to demonstrate that there will be no unacceptable impact on the surrounding population. An example of this is the HMIP risk assessment of dioxin emissions from MWIs[11].

On the basis of the limited emission data presented in Table 5.2 it is reasonable to draw comparisons regarding potential exposure to emissions between residents living near MWIs and those living near clinical waste incinerators.

5.2 IMPLICATIONS OF PAST MONITORING POLICY

- It is generally agreed that there has been a significant improvement in emission standards as time has progressed and that subsequently it is likely that earlier incinerators will have had higher emissions than now currently permitted although they would have met the standards in force at the time.
- The lack of monitoring data prior to 1990 means we can therefore only speculate on the chemicals present in those emissions. Epidemiological studies, including recent ones examining the association of adverse health effects with proximity to municipal and clinical waste incinerators refer to exposures (of uncertain duration) experienced before the introduction of prescribed chemical levels and enforcement of monitoring. Hazards have been viewed in terms of proximity to the source. The plume distribution of the gases emitted might have been overlooked, thus excluding focus on potentially vulnerable populations from study. The older incinerators are likely to have emitted greater concentrations of pollutants than is currently permitted.
- The Committee on Carcinogenicity was asked during 1993-4 to comment on the Small Area Health Statistics Unit study investigating the cancer incidence of over 14 million people living near 72 solid waste incinerators. The Committee agreed that the observed excess of cancers was not associated with emissions from the incinerators and may have been due to socio-economic confounding factors. Furthermore the Committee agreed that any potential risk of cancer due to residency of ten years or more near to municipal solid waste incinerators was low and probably not measurable by modern epidemiological studies.[12]
- Compliance with more stringent standards applied only from 1996. The IEH predicts two possible effects: first pollutants not prescribed for monitoring may form a greater proportion of the emissions in the future; second, the new limits apply only to stack emissions so that other pathways may assume greater importance. The relative importance of different pollutants may change.[13]
- In recent years there has been a reduction in levels of emissions from pollutants in the UK. The detection, if any, of an increased relative risk in cancers causally associated with incinerator emissions would refer predominantly to accumulated exposures before the implementation of stricter controls took effect.[14]

5.3 THE FAZAKERLEY INCINERATOR

EFFECT OF CHANGING REGULATIONS AND LEGISLATION

In 1991 clinical waste incineration became a prescribed process under the Environmental Protection (Prescribed Processes and Substances) Regulations 1991, thereby removing Crown Immunity that had previously exempted hospital incinerators from the requirement of pollution and planning control. Under Crown Immunity, all hospital incinerators were administered by the NHS estates managers. While Crown Immunity applied, monitoring was frequently minimal or even absent. For example, prior to 1992, there is restricted data on what substances were burned and there are no monitoring records for the Fazakerley hospital incinerator. Between 1992-95 the incinerator was tested three times within the requirements of the Authorisation and was found to be well within the particulate emissions allowed (500mg/m^3), no chemical analysis of emissions were performed as there was no requirement for it. The absence of data allows the experts [now the Environment Agency (EA), in 2000, previously Her Majesty's Inspectorate for Pollution (HMIP)] to make only a speculative guess at what chemicals were present in the emissions.

Prior to the Environmental Protection (Prescribed Processes and Substances) Regulations 1991, there was no requirement for keeping records or testing, other than the rudimentary requirements of the Clean Air Acts. Record keeping and monitoring would not have been required even in the case of commercial incinerators. Thus it is the lack of legislative requirement rather than the Crown Immunity, which may be important.

After 1991 legislation provided for more stringent pollution control and environmental protection. Incinerators burning less than 1 tonne/hour were the responsibility of the Local Authority while those burning in excess of 1 tonne/hour fell under HMIP, the predecessor of the Environment Agency. The legislation did not apply with immediate effect but allowed for the phased gradual implementation of the new performance standards and emission limits, so that existing incinerators only had to conform to the new standards by 1 December 1996.

In respect of the phased introduction of the 1991 legislation:

- Only incinerators, which complied with or were upgraded to meet the interim standards contained in (PG)5/1(95) were allowed to continue in operation in the short term
- The original date given by the Secretary of State by which these had to comply with the full emission requirements was 1 October 1995. It is understood that this was later extended in some cases. Fazakerley Hospital closed its incinerator in advance of the original date and did not require to take advantage of this extension in time.

Many small-scale hospital incinerators were unable to comply with the stricter emission standards so ceased operation at this time. Department of the Environment Process Guidance Note (PG)5/1(91) opened the way for private investment in large scale operations which would accept large quantities of clinical waste from a wide area incorporating several health authorities.(15) The number of major incinerators treating clinical waste has reduced from 700 in the late 1980s to 37 in the 1990s, the majority operated by private specialist companies and only a minority on hospital sites.(16)

Under the Environmental Protection Act 1990, Part I, enforcing authorities have control over waste incineration. Standards for incineration processes rated at less that 1 tonne per hour are set out in the Department of Environment Guidance note PG5/1(95) and for those rated 1 tonne per hour or more, in PG5/1(92). Incinerators rated at more than 1 tonne per hour are subject to Integrated Pollution Control (IPC) Part A which covers all emissions. Part B sets standards for flue emissions to air only, from incinerators rated at less than 1 tonne per hour and are regulated by local authorities under Local Authority Air Pollution Control (LAAPC) or in Scotland by the Scottish Environment Protection Agency (SEPA).

Under the Environmental protection Act 1990, operators are required to use the Best Available Techniques Not Entailing Excessive Costs (BATNEEC) which aims "to prevent the release of prescribed substances, and where that is not possible, to reduce and render harmless unavoidable releases".(17)

The environmental performance of incinerators has improved over the past decade. Many newer incineration plants now permit energy recovery, using a variety of incinerator systems. At the same time the European-proposed Waste Incineration Directive (WID) will include tight controls on emissions and ensures more efficient operation of incinerators.

TYPES OF INCINERATORS

There are a number of incinerator types, each with advantages and disadvantages. Material may be lost during any stage of the process from receipt of waste onwards as solid, liquid or gas waste streams and this may occur as part of the process or accidentally. Release may occur to air, to water or to land.

According to information received(18) the original incinerator at Fazakerley, provided circa 1974, was very basic, and was of a design currently in use throughout the NHS at the time. In 1985 this was replaced by a pyrolytic incinerator complying with the latest British Standard and guidance and this was substantially upgraded in 1992. Throughout the period of interest the hospital invested substantially in providing an up-to-date clinical waste incinerator plant that was operated and maintained in accordance with all standards relevant at the time. During this period, the bottom ash (the residue of the incineration process) was removed regularly to sealed containers and disposed of as sterile ash by licensed waste contractors in approved landfill sites nationally.

Clinical incineration is regarded as the best option for processing hazardous clinical waste; for lower grades of infectious waste other methods may be used following autoclaving or microwaving and landfill sites may be used.(19) Municipal incinerators are generally much larger than clinical waste incinerators, are more efficient and can achieve greater economies of scale. However they are only licensed to deal with certain types of low-risk non-infectious waste.(20)

5.4 Review of the Current Research on the Possible Links between Long-term Exposure to Emissions from Incinerators and Cancers in the Population

PROBLEM IDENTIFICATION AND DEFINITION OF TASK

Incineration can be defined as the "controlled burning of waste products".(21) There is public concern about incinerators because of reports of possible health effects on people living near them. Waste incineration is known to result in emission of potentially toxic pollutants, in particular dioxins, acid gases, nitrogen oxides, heavy metals, particulates and polyaromatic hydrocarbons. "Small quantities of a multitude of other products may be formed, depending on the chemical composition of the waste and the combustion conditions encountered".(22)

The greatest amount of scientific and public concern has been given to dioxin-like compounds, which are a subset of chemicals, formed as by-products during various combustion, industrial and biological processes and widely distributed in the environment.(23) Waste incineration is the largest known source of emissions of dioxins and furans to air in Europe. The most toxic dioxin, 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), was classified as a human carcinogenic substance by the International Agency for Research on Cancer in 1997 mainly on the basis of evidence from epidemiological studies of highly exposed humans and laboratory animal studies.(24) A subsequent consideration of the evidence by the UK Committee on Carcinogenicity of Chemicals in Food, Consumer Products and the Environment concluded that there was insufficient data to conclude a causal link with cancer in humans but that it would be prudent to consider TCDD as a "probable weak human carcinogen."(25)

As one component of the multidisciplinary investigation, Dr Emer Coffey was asked to review the evidence from epidemiological studies of an association between chronic environmental exposure to incinerators and risk of cancers in the population. There are different types of studies that assess the health effects of incinerators. Human exposure studies look at evidence of exposure to pollutants from incinerators. Risk assessment studies calculate life-time probabilities of health effects for individuals with estimated exposure levels using chemical specific emissions data.(22) Epidemiological studies have advantages over both exposure studies and risk assessment studies in that they study real life scenarios, measuring actual health outcomes in addition to accounting for exposure to all pollutants, measured or unmeasured. It was thus decided to focus this review on epidemiological studies because they may be considered the most valid.(26)

The primary purpose of this review was to examine the epidemiological evidence of excess cancer risk in populations living around the incinerator. Studies of workers with chronic occupational exposure to incinerators were examined separately because this type of exposure is different.

SEARCH STRATEGY

The main source of studies for this review was a search of MEDLINE and TOXLINE databases in November 2000 for relevant articles using the search terms for the period early 1980s to 2000, using the following search strategy:

(neoplasm or neoplasms or cancer or cancers) and (incineration or incinerator or incinerators); (neoplasm* or cancer* or sarcoma* or lymphoma*) and (incinerat*); these databases were also searched using terms (incinerat*) and (health or adverse) not (cancer*or neoplasm) and (human).

World Health Organisation, European Union and International Agency for Research on Cancer websites were examined for relevant material in September 2000. Reference lists from pertinent articles were screened and contact was made with experts and Greenpeace UK to identify further relevant studies. An attempt was made to obtain non-english language references.

Titles and abstracts of all articles identified by the methods described in the search strategy were assessed to identify potentially relevant studies. Full-text versions of these papers were obtained and assessed to see if they met the inclusion criteria.

SELECTION CRITERIA

Types of studies:

Epidemiological studies of adverse health outcomes in people living near or working at incinerators were considered for inclusion in this review according to the following weighting:

A: Prospective cohort studies.

B: Case control studies / retrospective cohort studies.

C: Other/Ecological studies.

Types of participants:

Studies which looked at either residents living near incinerators and people working at incinerators were considered. Attention was paid to whether the distance of the residence from the incinerator was reported - the nearer to the incinerator the better. If age was specified children and adults were considered separately.

Types of exposure:

Studies of all types of incinerator were considered. Note was taken, if reported, of the type of incinerator studied, for example municipal waste or clinical waste, and of environmental or biological measurements of contaminants or biological markers of such contamination if reported.

The types of exposure included were living near an incinerator or working at an incinerator preferably compared with no exposure. Studies that examined the health effects of acute high dose exposures, for example as the result of an accident or explosion, were not included because they were not considered relevant. Exposure to an acute high dose of a pollutant is different to potentially being exposed to chronic low doses in the environment and it may have different effects on the body.

Note was taken of whether account was taken of factors other than exposure to an incinerator which may have affected the incidence of adverse health events, for example, socio-economic status and smoking.

Types of outcomes:

The outcomes included were:

- The occurrence of cases of cancer. All measures of occurrence were included, for example, incidence of cancer and death from cancer. Both studies that reported the occurrence of cancer at all sites and cancer at specific sites, for example, lung cancer, were included;
- The occurrence of an adverse health outcome other than cancer, however measured.

Attention was paid to whether the lag-time or latency time between people being exposed to incinerators and the measurement of health outcomes was appropriate, i.e. whether the studies were long enough to detect possible long-term effects of exposure.

QUALITY

Studies were assessed for their quality in terms of their design, potential biases, measurement of exposure and control of confounders. Specific quality issues of each study are considered separately after the description of the study. General quality issues are considered in the discussion.

RESULTS: STUDIES OF CANCER IN GENERAL POPULATION

1. Small Area Health Statistics Unit (SAHSU) 1992 study of waste solvents and oil incinerators[27]

This study was carried out in response to reports of a possible cluster of cases of laryngeal cancer near an incinerator at Charnock Richard in Lancashire in the UK. The Charnock Richard study had found no more cases of laryngeal cancer than expected (58 cases) but a statistically significant effect of distance on the incidence of cancer of the larynx; this was highly dependent on a cluster of 4 cases close to the incinerator.[28] In order to investigate this association, a bigger study was carried out which measured the incidences of cancer of the larynx and lung in the population living within 10km of 10 incinerators of waste solvents and oil in Great Britain, which began operation before 1979, using lag-periods of 5 and 10 years. Cancer-registry records were used to measure the cancer incidence. The rates observed were compared with expected values based on national cancer incidence rates adjusted to take account of regional differences in reporting and incidence rates. When the data were adjusted to take account of socio-economic deprivation, no statistically significant excess incidence of cancer was found. There was also no trend of decreasing risk of cancer with distance from the incinerators.

The authors noted that their study was limited by the likelihood of sub-regional differences in completeness of cancer-registration, reliance on census data for population estimates which did not take migration into account, short lag-time used and the approximate nature of the exposure measured which was a simple distance model.

2. SAHSU 1996 study of municipal waste incinerators[29]

A much bigger study was subsequently done in the UK by the Small Area Health Statistics Unit. The cancer incidence of over 14 million people living near 72 municipal waste incinerators was examined, from 1974-86 (England), 1974-84 (Wales) and 1975-87 (Scotland) and compared with regionally adjusted national cancer incidence rates as before. The study was done in two stages as a quality-check to protect against finding a positive association by chance; the first involved a stratified sample of 20 incinerators and the second considered the remaining 52 incinerators.

Overall, the risk of all cancers combined, and stomach, colorectal, liver and lung cancers was found to decrease significantly with distance from incinerators. In the second stage of the investigation, allowing for a 10 year lagtime, the excess risk in people living less than 1 kilometre from an incinerator was found to be 4% for colorectal cancer, 6% for all cancers combined, 14% for stomach cancer, 14% for lung cancer and 37% for liver cancer. This excess risk of 37% for liver cancer was calculated to mean 23 extra cases within 1 km.

Despite attempting to adjust the data for socio-economic deprivation, the authors reported that residual socio-economic confounding was likely to explain the findings for all cancers combined, stomach and lung cancer and to explain at least part of the excess risk of liver cancer.

3. Review of SAHSU municipal solid waste incinerators study 2000 (30)

The Committee on Carcinogenicity of the Department of Health requested that a further investigation of liver cancer cases be carried out because of the possibility of residual confounding and the possibility of misdiagnosis of secondary liver cancers as primary cancers.(31) A histological and case-note review of the liver cancer cases was subsequently carried out. 119 cases of liver cancer were selected from the 235 cases previously identified, using criteria of availability of histopathological material and/or medical records. The 235 cases included all 87 cases at less than 1km from municipal waste incinerators and random samples of 74 from each of the distance ranges of 1 to 7.5km and greater than 7.5km. Histopathological material was available for 94 cases and medical records only were available for a further 25 cases. Primary liver cancer was confirmed in 66/119 cases (55%); and 21/119 cases(18%) were considered to be definitely secondary; 26 cases could not be distinguished between primary and secondary cases; no cancerous material was found in 6 cases. Assuming that the primary liver cancer is the correct diagnosis in 55% of all registered cases means that the number of excess cases of liver cancer at less than 1km from municipal solid waste incinerators is reduced from 23 cases to between 12.6 and 18.8 cases depending on whether possible primaries are included.

In its overall evaluation of the SAHSU studies, the Committee on Carcinogenicity agreed the excess of all cancers and cancers of the stomach, lung and colo-rectal were caused by socio-economic confounding. They agreed that although the excess of primary liver cancer near incinerators was not readily explained by confounding, that residual socio-economic confounding could not be excluded as the cause of this excess because the risk of primary liver cancer has been found to be more than double in the most deprived compared with the most affluent areas.(31)

4. Hoglund 1993 study of a waste incinerator(32)

This was a Swedish cohort study of cancer incidence of over 4000 residents of an area "polluted" (as defined by a computer simulation) by a waste incinerator, compared with residents of a "non-polluted" area. No difference in cancer incidence was reported.

It was not possible to appraise the quality of this study because the results were just available as a short abstract.

5. Biggeri 1996 study of industrial/urban pollution(33)

This was a retrospective case-control study, with geographical analysis, set in an industrial area in Italy. 938 cases of histologically confirmed lung cancer in men living in the province of Trieste, who had died between 1979 and 1981 or between 1985 and 1986 were identified from the cancer registry. Age was not specified. 755 cases for whom it was possible to find the next of kin were selected. The cases were compared with a random selection of 755 men from the same province, who died within the same six-month period at the same age plus or minus two years. Men who had died from chronic lung disease, cancer of the upper aero-digestive tract, urinary tract, pancreas, liver or gastrointestinal tract were excluded from the comparison group. Information on smoking habit and occupation was obtained from the next of kin for both cases and controls. The distance from four sources of environmental pollution (shipyard, iron foundry, incinerator and city centre) was measured. Spatial models, based on distance from source, were used to investigate the effects of different sources of air pollution on lung cancer after adjustment for age, smoking, likelihood of exposure to occupational carcinogens and levels of air particulates.

The study found that the risk of lung cancer was highly related to the city centre. When each source was considered separately, there was no statistically significant excess risk at the incinerator. However, an excess risk related to the incinerator was found if the data are adjusted for the effects of the city centre as well as the above confounders.

A major limitation of this study is that it studied industrial/urban air pollution rather than just incinerator emissions. The authors mention the possibility of residual confounding due to other unmeasured exposure, possibility of incomplete case-ascertainment and bias due to change in residence.

6. Michelozzi 1998 study of industrial pollution(34)

This was an observational study set in a suburb of Rome. Mortality from cancer of the liver, larynx, lung, kidney, lymphatic and haematopoietic systems, as defined by death-certificates, was evaluated for 341,389 males and females living within a 10km radius of an industrial area between 1987 and 1993. The industrial area contained a waste incinerator plant in addition to a waste disposal site and an oil refinery plant. All plants had been in operation since the 60s but the incinerator had been shut down in 1985. Distance from the industrial area was divided into bands: 0-3km, 3-8km and 8-10km The rate of observed death from different types of cancers was compared with the rate expected for men and women of different age. No significant excesses of cancer death were found within 3km of the industrial area or within the distance range of 8-10km. Within the distance range 3-8km, observed deaths of cancer of the kidney in women were higher than expected. The authors noted that it was possible that this positive finding was found by chance because so many tests were carried out.

There was no significant decline in mortality with distance from the industrial area when socio-economic factors were taken into account.

A major limitation of this study was that it looked at industrial pollution rather than differentiated sources of pollution. The likelihood of incompleteness of case-ascertainment due to reliance on death-certificates to define outcome is also mentioned by the authors.

7. Viel 2000 study of a municipal waste incinerator(35)

This study analysed 110 incident cases of soft-tissue sarcoma and 803 cases of non-Hodgkins lymphoma in France in an area with 485,000 inhabitants over 16 years from 1980–1995. A municipal solid waste incinerator with high dioxin emissions was operating since 1971 in the area. Dioxin emission concentrations were reportedly 16 times the 1994 European Union standard. The number of cases observed was compared with the expected number and adjusted for age and the pattern of occurrence of the cancers was examined for the presence of clusters. For the clustering analysis, cases of Hodgkin's disease were measured as a comparison to examine for selection bias. Hodgkin's disease was chosen because the authors noted that it has not been consistently associated with exposure to dioxins.

A significant geographic cluster of soft-tissue sarcomas was found in an area around the incinerator composed of 2 cantons or wards (p=0.004). This cluster was dependent on an excess of 14 cases more than the 31 expected. When the data was adjusted for sex, the cluster was only significant for males. A significant cluster in the same 2 ward location was also found for non-Hodgkin's lymphoma (p=0.00003) which was dependent on an excess of 61 cases more than the 225 expected.

This study has a number of limitations. The location of the cluster could not be measured at sub-ward level. It is reported that cantons are electoral wards varying in size from 50-325 square km with population size varying from 2,900 to 123,000. The size of the cantons around the incinerator are not described other than a description of one being semi-rural and another densely populated. Also, the effect of migration was not considered. The data were not adjusted for socio-economic status that may be a confounder although the authors report that "the majority of mortality and incidence data for lymphomas and connective tissue cancer have shown no clear association with social class". Urbanisation is a possible confounder although the authors report that there is no conclusive evidence of an association. The fact that no cluster of Hodgkin's disease was found was cited as evidence that the pattern of cases observed was not due to varied pattern of referral.

The authors recommended that their findings needed to be confirmed by further investigation, for example, by a case-control study in which dioxin levels were measured in the body before clusters of soft-tissue sarcoma and non-Hodgkin's lymphoma could be ascribed to dioxin release from the incinerator.

8. Knox 1997 study of industrial processes(36)

This ecological study was based on the premise that childhood leukaemias and cancers occur in small geographical clusters that reflect persistent local hazards. The study aimed to examine the association between the addresses at birth and at death of children dying from cancer and the location of different types of "potential environmental hazards". A broad range of potential hazards was considered including factories, oil refineries, gasworks, airfields, crematoria, nuclear installations, TV transmitters, railways and motorways. All

22,458 children who died from cancer before their 16th birthday between 1953 and 1980 in England, Scotland and Wales were included. The observed numbers of births and deaths at different radial distances from the hazards were compared with the numbers expected. Relative excesses of leukaemia and solid cancers were reported within 5 kilometres of a range of industrial processes. A stronger association was reported for birth addresses than death addresses. The authors concluded that childhood cancers are geographically associated with two main types of industrial atmospheric effluent: (1) petroleum derived volatiles, and (2) kiln and furnace smoke and gases, and effluents from internal combustion engines.

This study was reviewed by the Committee on Toxicity, Mutagenicity, Carcinogenicity of chemicals in Food, Consumer Products and the Environment at the request of the Department of Health.(37) *The Committee found that it was not possible to draw any conclusions from this study in view of the following limitations:*

- **Absence of reliable data on total number of children at risk in each postcode area.**
- **Imprecise identification of hazards and lack of information about potential chemical exposures.**
- **No consideration of specific cancers.**

The Committee also felt that there was no need to carry out further work on this study.

9. Knox 2000: further analysis of municipal waste incinerators, hospital incinerators and toxic waste landfill sites[38]

A subgroup of these children who had moved at least 0.1 km between birth and death was further analysed using a method that did not involve demographic data. The design was based on the principle that a cancer-initiating source operating shortly before or after birth would be more closely geographically associated with the birth address of a dead child than with the child's address at death. The "potential hazards" considered in this study were 70 municipal waste incinerators, 307 hospital incinerators and 460 toxic waste landfill sites.

9,224 children were identified as having migrated between birth and death. The analysis was chiefly based on 4,385 children or 47.5% of "migrating cancers" for whom it was possible to accurately identify both birth and death addresses.

The distance between the birth and death addresses of each child and the nearest "hazard" was measured. The source closest to each of the paired (birth/death) addresses was selected. The closest source at birth was usually the same source as at death but where the sources differed, the distances to the separate sources were differenced. The outcome was measured as the ratio between numbers of children migrating away from or towards their nearest source. Different distance-bands were chosen to investigate possible different exposure-gradients. Significant excesses of migrations away from municipal and hospital incinerators were found. The asymmetry depended largely on 10 of the 70 municipal waste incinerators; 9 of which were in operation before 1945 and all of which were in industrial areas. No asymmetry of migration was found around landfill sites.

Although a different method is used in this study than the previous Knox study, limitations (b) and (c) which the Committee on Carcinogenicity felt applied to the previous study also seem to apply to this study.[37] The authors also note that social, demographic and other selective effects cannot be excluded.

STUDIES OF SUBGROUP OF OCCUPATIONALLY EXPOSED INCINERATOR WORKERS

1. GUSTAVSSON 1989[39]

This retrospective cohort study in Sweden compared the mortality rates of 183 male workers at an incineration plant with general population rates over 34 years from 1951-1985. Selected workers had been working in the plant for at least 1 year. 22 cancer deaths and 9 lung-cancer deaths were observed. Information about smoking was obtained through interviewing An excess of lung cancer was found when compared with national rates was 355; (95% CI=162-675). However, when compared with the local rates which seems like a more appropriate comparison, the standardised mortality ratio decreased to 197 (95% CI=90-374), which means that there was no significant excess in lung cancer.

This study is limited by the small numbers involved as demonstrated by the wide confidence intervals attached to the ratios. In addition, latency periods were inadequate for some workers because workers were exposed for different duration's from as little as 1 year.

2. Rapiti 1997[40]

This retrospective cohort study in Rome compared the mortality rates of 532 male workers employed at 2 municipal recycling and incinerating plants with regional rates over 30 years from 1962-1992. No significant excess was found in death rates from gastric cancer, lung cancer or all cancers.

This study was again limited by small numbers leading to wide confidence intervals. Local rates would be a better comparison than regional rates because mortality rates can vary greatly within a region. There is a lack of details about work activities so that workers are not graded according to the likelihood of exposure. There is inadequate information about confounders. Latency period is inadequate.

CHARACTERISTICS OF EXCLUDED STUDIES

The following studies were excluded because they did not fit the inclusion criteria.

ATSDR 1992[41] This study found an excess number of neuroblastomas in the area around a hazardous waste incinerator. There are concerns about the biological plausibility of linking neuroblastomas to environmental agents (this last point is not relevant to carcinogenesis but other non-cancer endpoints).

SCARLETT 1990[42] This study of occupational exposure looked at urinary mutagens as an outcome rather than actual cancer outcomes.

RELEVANT STUDY WHICH COULD NOT BE ASSESSED FOR INCLUSION/EXCLUSION

ARKANSAS 1987[43] An attempt has been made to locate the study which has been unsuccessful to date. Study of population of Union County, Arkansas; site of location of hazardous waste incineration facility. Observed deaths due to cancer compared with US mortality rates. Observed number found to be equal to the expected number.

DISCUSSION

The epidemiological studies which examine the association between cancer risk and living near or working at incinerators are difficult to summarise because the studies are very different, for example, in terms of size, design, exposure measured and quality. The findings across studies were not consistent.

Two of the studies of cancer risk in the population living around incinerators had no positive findings.[27, 32] Although the other studies reported positive associations, there were many limitations which weaken the evidence.

Two of the studies which reported positive findings,[33,34] were really studies of industrial air pollution rather than incinerators. An authoritative UK body, the Committee on Carcinogenicity found it was not possible to draw conclusions from the first study of Knox 1997;[36, 37] the further analysis of this study, Knox 2000,[38] also seems to have some of the same limitations. The study by Viel 2000[35] provides weak evidence due to its limited spatial analysis design and possibility of confounding.

The biggest studies by far were those carried out by the Small Area Health Statistics Unit (SAHSU) on people living near municipal waste incinerators in the UK.[29,30] This seems to be the most relevant study to consider in terms of addressing local concerns in Liverpool. It is based in the UK and focuses on incinerators rather than industrial air pollution.

Although it found an excess of some cancers, deprivation was found to be the likely explanation for all cancers, stomach and lung. Deprivation could not be excluded as the explanation for the small increase in primary liver cancer found.[31]

Therefore, overall the evidence of an increased risk of cancer in the general population living near incinerators is weak. However, the lack of evidence does not rule out the possibility that health may be affected by living near incinerators because "the effects if any are delayed, non-specific and weak"[44] and because of the difficulties in designing and interpreting studies to measure these effects. Many of the studies were small and lacked the power to detect weak effects.

Only two epidemiological studies of cancer outcomes in chronically occupationally exposed incinerator-workers.[39,40] Neither of these found a statistically significant excess occurrence of cancer. However, both of these studies were limited by their size.

Other issues which are pertinent for the interpretation of epidemiological studies of health effects of incinerators are discussed briefly below.

Selection of controls:

Choosing the most appropriate comparison group can be difficult and the limitations of the comparison used should be considered in interpretation. Using the general population as a standard may underestimate any true increased or decreased risk because there are both exposed and non-exposed individuals in the general population.

Exposure data:

Measuring exposure to incinerator emissions is not easy. The studies reviewed contained little information about measured levels of emissions, either in the environment or biologic tissues. There are many problems with measuring emissions from incinerators. The levels may vary daily. Quantities emitted may be minute and difficult to measure. There may be uncertainty about what constitutes a safe level of a exposure and thus what levels need to be measured. Incinerator emissions consist of many chemicals and there is difficulty in selecting which chemicals to measure. There is a lack of information about the toxic cocktail effect of the multitude of pollutants acting together.

Instead of directly measuring exposure data, most studies used measures of distance of residence from an incinerator as a proxy for exposure. A problem with using distance is that the height of the stack of the incinerator, the local geology and weather will all influence the area that is exposed. Plume-modelling, which attempts to define a "polluted" area by taking these factors into account, was not used in any of the studies.

Unrecorded migration may lead to misclassification of exposure. This would lead to a dilution of any effect of exposure, for example, an exposed person migrating out of the study area before the disease occurs or an unexposed person moving into the study area.

Pathways and levels of exposure are different in occupational studies. A limitation of the studies of occupational exposure reviewed was a lack of differentiation of workers in terms of their likelihood of exposure. It is likely that some workers are potentially more highly exposed to pollutants than others.

Confounders:

Even if an association is found between incinerators and risk of cancer, this does not necessarily mean that the association is causal. Other factors, which may be difficult to identify and adjust for, may confound the association. Many factors are recognised to contribute to the development of cancer. The causes and their relative importance are not fully understood.

Potential confounders considered in the reviewed studies to different extents were: age, sex, smoking, occupation, socio-economic status, urbanisation, patterns of medical referral, level of particulates, background radiation, use of herbicides, other risk factors for specific diseases e.g. cirrhosis and liver cancer.

Socio-economic factors are particularly important to consider. The majority of cancers are more common in people with lower socio-economic status, for example, liver and lung cancers. Residential areas around industrial plants like incinerators tend to be disadvantaged and therefore the people living in these areas tend to have a higher risk of cancer anyway.

Latency:

Latency periods must be adequate to measure outcomes. The lag-period between exposure to a carcinogen and diagnosis of cancer may be very long and also may be variable. Latency period for childhood cancer will be at the shorter end of the range. The latency periods in the studies were not always clear due to lack of information about length of operation about incinerators, length of residence of people, age of people. In some studies, the length of latency period varied.

Lag-times of 5 and 10 years were used in the SAHSU studies.[29,30] However, Elliott 1992 noted that 10 years was a relatively short lag-time for the epidemiology of solid tumours.[27]

Outcomes:

Often routine data are used to measure outcomes. Routine data have their limitations in terms of accuracy which may introduce biases, for example, death-certificates may be inaccurate; Elliott 2000 found that cancer registration data overestimated the numbers of primary liver cancer cases.[30]

Both cancer deaths and cancer occurrence were used as measures of cancer outcomes. Data on deaths is a poor measures of cancers with a long survival time. Advantages of using data on cancer occurrence are that it is not affected by differential survival between areas[35] and because it alleviates migration effects because the time from exposure is shorter.

Applicability of findings:

The time context of the studies needs to be considered. Emissions from the incinerators studied are likely to have been higher than from modern incinerators. Over the years, standards for pollution control have changed, for example, new European Union standards imposed in 1996, resulted in the closure of many older incinerators.[45] It may not be appropriate to extrapolate from evidence from exposure to older incinerators to modern incinerators although it may be appropriate when considering historical exposure.

In general, it may not be appropriate to extrapolate from the results of any one incinerator study to other incinerator scenarios because local conditions may differ[45] for example:

- **Waste (e.g. type and volume). Clinical waste contains a relatively high amount of chlorinated plastics compared with municipal waste. Metal emissions from hazardous waste or municipal solid waste incinerators have been reported to be from 2 to 20 times higher than from sewage-sludge incinerators.[22] Volume of waste may be highly variable from day to day as well as from incinerator to incinerator.**
- **Incinerator (e.g. location, size, structure, technology, pollution control methods, management).**
- **Other sources of exposure to pollutants. For example, in this review, two of the studies did not specifically study incinerator emissions but industrial emissions to which incinerators contributed.[33, 34]**
- **Geographical and meteorological conditions**
- **The pathways of exposure (e.g. the food chain)**
- **Activities of exposed population**
- **Susceptibility of exposed population (e.g. the young, the old, the infirm, genetic susceptibility).**

Knox 2000 looked at children under 16.[38] Age was not specified in the other studies. There is a lack of data about the potential effects of chronic exposure to incinerators and the development of childhood cancer or the effects of perinatal exposure on development of cancers later in life. Rapidly growing foetal tissues may be more susceptible to the effects of exposure.[38]

Males and females may respond differently to emissions-exposure. Biggeri 1996 looked only at males.[33] All other studies looked at both males and females.

Research possibilities

Most studies about the effects of incinerator emissions are animal studies or studies of acute high exposure, for example, due to accidents. There have been very few epidemiological studies published of health effects of people living near or working at incinerators.

There is a need for further and better quality data about human exposure to pollutants.[46,33,35]

In March 2000, in the light of the findings of the SAHSU study, the Committee on Carcinogenicity in the UK concluded that "any potential risk of cancer due to residency (for periods in excess of 10 years) near to municipal solid waste incinerators was exceedingly low and probably not measurable by the most modern epidemiological techniques" and that there was no need for further epidemiological investigations of cancer incidence near municipal solid waste incinerators "at the present time". [31]

If epidemiological studies are carried out in the future, in order to obtain good quality information, the studies need to be large enough and long enough to detect small increases in risk over the longer term and also need to address possible confounding factors. Ideally, they would be combined with better measures of exposure to pollutants, i.e., biological and environmental emissions data. These principles apply to both studies of the general population living near incinerators and workers at incinerators.

6.1. Environmental Background

Much of the environmental background has already been covered in section 4 on the history of air pollution in Liverpool. In the course of pursuing this investigation, contact was made with the Environment Agency North West regarding any historical major industrial processes in the Fazakerley area and also with Knowsley and Liverpool Local Authorities Environmental Health Departments regarding any known potential local environmental hazards. Both Local Authorities are currently compiling their Contaminated Land Registers and were able to review the study area for possible land contamination.

One problem encountered early on in the study was that the incinerator under consideration no longer existed as it had been demolished following ceasing operation in September 1995. This meant that it was not possible to carry out test firings and record dispersion plumes. Prior to its closure, the incinerator was fully tested on three occasions (April 1992, December 1993 and February 1995) by Liverpool City Council in accordance with the interim standards set out in the 1990 Environmental Protection Act.

6.2. Liverpool City Council's Investigation into Complaints from Local Residents

During the period from October 1993 until the incinerator's closure, the plant (both incinerator and the hospital's boilerhouse) was subject to considerable scrutiny from Liverpool City Council's Environmental Health Department[1] due to the concerns of local residents. ***It is clear from the report and from the complaints of local residents that the smut deposition was mainly from emissions from the hospital boilers rather than the incinerator. However, there has been considerable confusion between the two in both official reports and media coverage.***

A report[1] on this was prepared for the members of the Environmental Strategy and Development Control Sub-Committees by the Head of the Environmental Health Service in April 1994.The purpose of the report was to advise the committees on the monitoring of the emissions from the incinerators and boilers and whether the hospital management should be asked to carry out additional monitoring.

MONITORING OF SMUT DEPOSITION BY ENVIRONMENTAL HEALTH 1993-1994

Following complaints from local residents in the summer of 1993, the Environmental Health Department had carried out some basic monitoring using contact plates (these are 5 inch square sticky pads which collect particulate deposits). The plates were sited at two houses on Lower Lane between 6th and 23rd August 1993. They were then sent to the Public Analyst's laboratory for examination. The Analyst's report stated that:

"the deposits on the plates were typical of an urban airborne deposit and were not excessive. There was, however, some evidence of acid smuts, comprising agglomerates of coked oil droplets."[1]

The Analyst's opinion was that the coked oil droplets were probably derived from oil fired boilers.

The Environmental Health Department carried out an inspection of the Fazakerley Hospital boilers on 23rd September 1993 which concluded that the boilers were operating correctly and there was no obvious reason why the plant should be making abnormal emissions. The hospital engineers in the meantime had sought advice from SAAKE, the manufacturers of the boilers, on the efflux velocity of gases leaving the chimney as this could effect smut formation within the flue. Environmental Health also requested that the manufacturers should advise on the most effective way of firing -up the stand-by boilers to prevent "lift-off" of smuts from the flue lining.

The information from the manufacturers confirmed that the efflux velocities were within acceptable ranges. (16ms^{-1} at high fire and 5ms^{-1} at low fire). A minimum of 6ms^{-1} at high fire was required to prevent ingress of cold air (inversion) at the chimney stack exit.

SAAKE advised the hospital engineers to carry out the following additional measures:

- Check for air leaks in the ducting between the boiler and the chimney. This was carried out and found to be satisfactory.
- Not to continuously operate the main boilers at low fire rate while frequently firing -up the stand-by boilers to maintain pressure. The hospital engineers investigated firing-up the stand-by boiler once a day on both burners to ensure enough heat input to maintain pressure (as opposed to the practice at the time of firing it up four or five times a day).

Environmental Health advised that the most vulnerable time for a boiler plant to emit smuts was at low fire rate or during firing-up from cold. The hospital engineers took steps to prevent smut emissions in those circumstances that included the use of a fuel additive to prevent or minimise the formation of acid smuts in the first place by neutralising the acid gases in the chimney-stack.

Following these measures, the Environmental Health Department carried out further contact plate monitoring at two houses on Lower Lane between 24th September and 11th October 1993. Analysis showed typical urban deposits. *From late November 1993 until the time of the report to Committee (April 1994), weekly monitoring of smut emissions in the vicinity of the boiler-house and incinerator was carried out using contact plates at three locations:*

- Two houses on Lower Lane
- 1 local school (Holy Name)

The results showed no evidence of excessive emissions from either the boiler or the incinerator.

The Environmental Health Department, in their 1994 report, advised that:

"Section 10 of the Clean Air Act 1993 makes provision for a Local Authority to require measurement of grit, dust and fumes emitted from a chimney to assess compliance with the specified emissions standards and, if necessary, require the installation of arrestment plant. However the Clean Air (Arrestment Plant) (Exemption) Regulations 1961 specifically exempt oil-burning furnaces from the requirement to fit arrestment plant."(1)

The report pointed out that under the then legislation, the existing plant was permitted an emission rate of **14 .0 lb/hr of grit and dust**. It was considered that this level was not being exceeded but the hospital engineer had been asked to consider whether the best practicable means practicable were being used to minimise emissions from the boiler chimney.

PARTICULATE MONITORING UNDER THE INTERIM STANDARDS OF THE ENVIRONMENTAL PROTECTION ACT 1990

The Hospital Management were required, under the terms of their authorisation under the Environmental Protection Act, to monitor particulate emissions annually. A first emissions test was carried out in September 1992 and showed an average annual concentration of **140mg/m^3** as compared to then limit value of **500mg/m^3** as set down in clause 9 of the Interim Standards of the Secretary of State's guidance on Clinical Waste Incineration Processes under 1 tonne per hour. *(**NB**. Fazakerley's incinerator had a capacity of 250kg per hour).*

A second particulate emissions test was carried out in December 1993 and showed an average annual concentration of **185mg/m^3**. The final particulate emissions test carried out in February 1995 showed an average annual concentration of **229mg/m^3**.

Clause 13 of the Interim Standards referred to above stated that:

"the frequency of testing should be approved by the Local Authority and should depend on the nature of the emissions and the size of the incinerator."(1)

The report to Committee pointed out that the Local Authority was obliged to ensure that the incineration of clinical waste was carried out using BATNEC (Best Available Techniques, Not Entailing Excessive Costs) - particulate sampling at the time (1994) cost approximately £2,000. As previous sampling had shown that concentrations were well within the emissions limits set out in the Interim Standards, the Environmental Health Department advised that more frequent monitoring would be **"excessive"**. The Environmental Health Department's report concluded that the weekly monitoring programme since November late 1993 had failed to provide any evidence which would justify the City Council requesting additional or more frequent monitoring of emissions from either the hospital incinerator or boilers.

Indeed the report pointed out that:

"As far as the incinerator is concerned, if the authorisation was varied to include more frequent monitoring (than annual), the hospital authorities (Aintree Hospitals NHS Trust) would have just cause to appeal against this condition under Section 15(b) of the Environmental Protection Act 1990 and the Local Authority would have little chance of winning given the current guidance issued by the (then in 1994) Department of Environment.."(1)

ATTEMPTS TO CLOSE THE FAZAKERLEY HOSPITAL INCINERATOR

A further report[2] on the incinerator was presented by the Environmental Health Department to Liverpool City Council's Policy and Resources Committee on 16th April 1995. The purpose of this was twofold:

- To advise city councillors of the possible financial implications of a resolution approved by the Environmental Services and Consumer Protection Committee on 13th March 1995.
- To inform city councillors on the powers available to the City Council to force the closure of the incinerator.

Following the meeting on 13th March 1995, the Head of Environmental Services had met with the Maintenance and Technical Services Manger of Aintree Hospitals to clarify the financial implications which would have been involved in taking action to close the incinerator. Legal advice had also been sought by the Head of Legal Services from Counsel.

The implications are summarised below:

- The Trust would have sought to recover any additional costs it would have incurred in disposing of waste if the Local Authority had closed the incinerator and then the Trust won on appeal. **A notice served requiring closure from the beginning of June 1995 would have resulted in a four month notice period up to the 1st October 1995 when the incinerator was scheduled to close anyway under the conditions set out in the Environmental Protection Act.**[2] The additional costs would have been between £80,000 to £200,000.
- The Trust had sought legal advice and was advised that it should appeal to the Secretary of State for the Environment had the Council served a closure notice.
- A number of other agencies were, at that time, contracting with the Trust to use the incinerator to dispose of clinical waste from domestic collections. Notably, the Merseyside Waste Disposal Authority which disposed of clinical waste from Sefton and Knowsley Metropolitan Borough Councils and Liverpool City Council in this way. In the year 1994-1995 the Trust had disposed of approximately **857 tonnes** from Liverpool City Council's own domestic collection.
- There were, at the time, no other incinerators (either new which were capable of meeting the full BATNEEC standards or those that could meet the interim Environmental Protection Act requirements) in Merseyside that had sufficient spare capacity to be able provide an alternative in the event of the Fazakerley incinerator closing ahead of schedule.
- Merseyside Waste Disposal Authority was concerned about the financial and environmental implications of early closure.
- The Trust had confirmed in writing to the Environmental Health Department that it was ***"planning to contract for the incineration of clinical waste effective from 1st October 1995 and plans to close down the Fazakerley incinerator at that time."***[2]

The report concluded with the Head of Environmental Services recommending that the City Council should not proceed with action to force the early closure of the incinerator. He did recommend that the Trust inform the City Council as soon as a contract for alternative disposal had been signed and to confirm the closure date of 1st October 1995. His concern at the time was that the Secretary of State for the Environment could have chosen to extend the deadline by which incinerators like that at Fazakerley could be upgraded or closed down. The City Council would have found such a course of action unacceptable because of the degree of public concern. In the event this did not happen and the closure of the incinerator went ahead as planned in September 1995.

6.3. Basic Modelling of Emissions from the Fazakerley Hospital Incinerator

The Environmental Health Department were aware of the continuing debate about the potential of harmful emissions from the incineration of clinical waste and, in addition to the weekly contact plate monitoring, had also considered the issue of gaseous emissions. In 1994, they had investigated the ground level concentrations for sulphur dioxide around the incinerator chimney.[1] This was one of the pollutants produced by incineration for which the Air Quality Standards of the time **(Air Quality Standards Regulations 1989)** had specified a limit value of **0.131 parts per million** (98 percentile of daily means throughout one year).

Environmental Health had used the modelling facilities of the Merseyside Emergency Resource Management and Information Database (MERMAID) to predict ground level concentrations of sulphur dioxide from the incinerator.[1] This system was primarily (and is still) used for the emergency management of chemical incidents where a crude plume dispersal model of accidental releases is helpful in making decisions about advice to local residents, need for evacuation, notifying neighbouring authorities etc. It is a crude system by present day modelling standards but was considered to be state of the art. The model produced for the Fazakerley incinerator took into account varying atmospheric conditions and used the data obtained from the December 1993 emissions test. Based on this information, it showed (deliberately) a worst case scenario - i.e stable atmospheric conditions, with low wind speed, where the plume would reach ground level quickly - and the direction of the plume across local residential property. The worst case scenario produced by this model showed that the highest concentration of sulphur dioxide (SO_2) at ground level was **0.012-0.024 parts per million**. These concentrations were found to exist at greater distance from the source than the properties on Lower Lane and complied with all the then current air quality standards. Indeed, the Environmental Health Department point out the highest concentration were almost identical to the then European guide value for sulphur dioxide of 15 to 23 parts per billion which was, at the time, a target level to be aimed for. The exercise was repeated for hydrogen chloride and particulate material and showed ground concentrations to be well within limit values.

The Environmental Health Department concluded that, in the context of the then Air Quality Standards, the emissions from the Fazakerley Hospital Incinerator were well within the limits specified.

ADVICE ON FURTHER MODELLING

As part of the current investigation being conducted by the Fazakerley Incident Team, advice was sought on whether further emission modelling from the incinerator stack (based on the annual particulate concentrations and the MERMAID model) was viable from Dr Adrian Watson and colleagues at the Atmospheric Research Information Centre based at the Department of Environmental and Geographical Sciences at Manchester Metropolitan University.

Dr Watson reviewed the reports from Liverpool City Council's Environmental Health Department[1,2] and the MERMAID "worst case" dispersion plume. He concluded that it was unlikely that any substantially new evidence could be provided by further modelling. The reasons for this were:

- The scarcity of the emission data.
- The incinerator no longer physically exists so cannot undertake test firings.
- The absence of meteorological data.

Dr Watson went on to explain that whilst current dispersion modelling techniques are advancements on MERMAID, they still operate on the same gaussian plume type approach and, in the absence of detailed meteorological data, would produce a similar dispersal pattern for the emissions as MERMAID. He also concluded that even with the "worst case" scenario produced by the MERMAID model the maximum pollutant concentration levels were indicative of the incinerator operating within regulatory limits.

7 TOXICOLOGY REVIEW

7.1 Role of Chemical Incident Response Service

The Chemical Incident Response Service (CIRS) at Guys and St.Thomas' Hospital London were asked to contribute to the investigation. Their findings are set out in this section.
They were asked to:

- Define the term "clinical waste".
- Identify the types of waste burnt at this incinerator and what the possible combustion products might be.
- Discuss the pollution control legislation applicable at the time, how it is changing and its implications for future emissions.
- Describe the toxicology of the identified individual and combined combustion products.

7.2. Methods and Sources of Information

SEARCH STRATEGY

A search was undertaken of Medline/Toxline bibliographic databases using MESH headings 'incinerat*', 'clinical incineration', 'adverse health effects', 'cancer'. Several environmental websites were searched including the UK Environment Agency, the US Environment Protection Agency, the Department for the Environment, Transport and Regions, the Health and Safety Executive using keywords 'waste incineration' and 'clinical waste incineration'. Colleagues at the Environment Agency and University Hospital, Aintree assisted in accessing grey literature and government documentation on regulation of incineration processes. Local reports and documents relating to the Fazakerley hospital incinerator were consulted. Experts on incineration based at the Environment Agency and academic institutions were consulted for their expert opinion and to discuss issues regarding past monitoring policy. (Table 7.1)

Table 7.1: Organisations consulted

ORGANISATION	PERSON CONSULTED
UK Environment Agency	Dr Amin Anjum *National Adviser on Waste Management*
University Hospital Aintree *(Previously Fazakerley Hospital)*	Mr Jim Laird *I.T. Services Manager Aintree Hospitals NHS Trust (previously Maintenance and Technical Services Manager)*

SELECTION CRITERIA

Articles were assessed for inclusion based on the following criteria:

- Definition and content of clinical waste incineration and their products of combustion.
- Association of waste incineration products with cancer and other adverse health effects in humans.
- Statutory regulation of incinerator emissions in the UK between 1990 and 2000. (see Section 5)
- Expert panel advice on permitted levels of emissions.

LIMITATIONS OF THE METHODS

- Many of the reviews and epidemiological studies, which pertain mainly to municipal waste incinerators, reflect absent or minimal monitoring of chemical emissions. Where monitored the emissions measured were limited to filter sampling analysis and did not take account of vapour phase test data and plume modelling, so that the potential path or extent of dissemination of the emissions was not known.
- Most studies referred to municipal rather than to clinical waste incinerators and there may be differences in the quantity and effects of specific emissions. There is little in the literature on monitoring; those chemical emissions monitored might produce a biased picture because they are perceived to be related to a specific problem. Those chemicals not monitored might turn out to be equally or more hazardous. Hazards have been viewed in relation to proximity to the incinerators.
- The adverse effects on human health ascribed to the pollutants from incinerator emissions may not represent an excess over the effects of contamination and pollution from surrounding industries, and/or the influence of socio-economic factors on human health from which they are not easily separated.
- Owing to Crown Immunity and lack of strict regulation prior to 1995, there is little data on the monitoring of emissions from this size of incinerator (larger incinerators were required to monitor more extensively than Part B incinerators in the period between 1992-1996 (suggest explain part b and interim EPA). The composition of the emissions is therefore only speculative and based on information about the nature of the waste. Plume modelling was not undertaken, nor was routine monitoring of incinerator staff to assess occupational health exposure. In the absence of plume modelling, risk assessment by proximity to the hospital or by postcodes may be misleading as the postcodes may fall outside the area of plume fallout and deposition.
- It is difficult to obtain clinical waste incineration monitoring data prior to 1995. Data were not held centrally, were incomplete and were held only by the Local Authority.

7.3.Clinical Waste

DEFINITION OF CLINICAL WASTE

The current accepted definition of clinical waste based on The Controlled Waste Regulations 1992[(1)] is:

(a) ***"any waste which consists wholly or partly of human or animal tissue, blood or other body fluids, excretions, drugs or other pharmaceutical products, swabs or dressings, or syringes, needles, or other sharp instruments which unless rendered safe may prove hazardous to any person coming in contact with it."***

(b) ***"any other waste arising from medical, nursing, dental, veterinary, pharmaceutical or similar practice, investigation, treatment, care, teaching or research, or the collection of blood for transfusion, being waste which may cause infection to any person coming into contact with it."***

Technical guidance on disposal of clinical waste is complex and proper segregation of different types of waste is critical to safe management with consideration given to risk assessment, handling and packaging, labelling, transport and treatment and disposal.[(2,3)]

CLINICAL WASTE GENERATION

A study for the Environment Agency in 1998 estimated that NHS Trusts in the UK produced 193,000 tonnes of clinical waste per year, with other sources including private hospitals, GPs, dentists and nursing homes producing a similar quantity.[4]

According to the hospital records it is estimated that the annual throughput of the incinerator under consideration was <5,000 tonnes per year - records show it burnt around 300,000 bags per year and a bag is unlikely to contain more than 10-15kg.

About 85% of hospital waste is general refuse, while infectious waste represents about 10% of the waste stream. Hospital wastes may contain a high proportion of plastics (20-30%), compared with only 3-7% in municipal solid waste.[5] The volume of plastics in hospital waste is increasing with the introduction of plastic packaging and disposable equipment.

CLINICAL WASTE DISPOSAL

Clinical waste has been divided into five categories, which provide a basis for risk assessment.[2] The level of risk varies within the groups. "Special waste" is subject to controls under the Special Waste Regulations 1996[6] and includes ACDP hazard Group 4 pathogens e.g. viral haemorrhagic fever pathogens, and prescription only medicines.

Table 7.2 below shows the five designated categories and the proposed options suitable for their treatment or disposal adapted from Safe disposal of clinical waste.[2]

Table 7.2 Waste categories and options for treatment/disposal

CATEGORY OF WASTE	TREATMENT OR DISPOSAL OPTION
Group A ▪ Includes the following items: Identifiable human tissue*, blood, animal carcasses and tissue from veterinary centres, hospitals or laboratories. ▪ Soiled surgical dressings, swabs and other similar soiled waste. ▪ Other waste materials, for example from infectious disease cases, excluding any in groups B-E.	**Clinical waste incinerator** ▪ *All identifiable human tissue is clinical waste and the only appropriate treatment is incineration. ▪ Maceration and heat or chemical treatment may be appropriate if waste does not contain ACDP Hazard Group 4 biological agents, waste from laboratories level 3, cultures of ACDP Hazard Group 2 biological agents.
Group B Discarded syringe needles, cartridges, broken glass and any other contaminated disposable sharp instruments or items.	**Clinical waste incinerator** Maceration and heat or chemical treatment may be appropriate if waste does not contain ACDP Hazard Group 4 biological agents, waste from laboratories level 3, cultures of ACDP Hazard Group 2 biological agents.
Group C Microbiological cultures and potentially infected waste from pathology departments and other clinical or research laboratories.	**Disposal Options** ▪ Autoclaved group C - Clinical waste incinerator ▪ Unautoclaved Group C - Maceration and heat or chemical treatment. Residues may be incinerated or sent to landfill
Group D Drugs or other pharmaceutical products.	**Clinical waste incinerator**
Group E Items used to dispose of urine, faeces and other bodily secretions or excretions which do not fall within Group A. This includes disposable bed pans or bed pan liners, incontinence pads, stoma bags and urine containers.	**Disposal Options** ▪ Clinical waste incinerator, municipal waste incinerator (which conforms to requirements for incineration of clinical waste), maceration and heat or chemical treatment. ▪ Residues may be incinerated or sent to landfill. ▪ Macerated group E- e.g.excreta may be disposed of in sewer.

At the time the Fazakerley incinerator was in operation maceration and heat and chemical treatment were not approved. The only legal alternative to incineration was deep burial at licensed landfill sites, which had been disbanded by the early 1990s.

7.4. Chemical Pollution Associated with Incineration of Clinical Waste

POSSIBLE POLLUTANTS PRODUCED BY BURNING CLINICAL WASTE

Most of the pollutant emissions from clinical waste will be the same as those found in municipal incineration as the bulk of clinical waste is generally refuse. However the relatively high percentage of plastics in clinical waste (derived from syringes, disposable instruments, plastic plates, bedpans, urine bags respiratory devices, dialysis equipment, packaging, etc.) containing PVC and other halogenated polymers and co-polymers, makes it distinctive.[5,7] Hospital refuse also contains chlorinated pharmaceuticals, chlorinated salts and aromatic organic compounds. Inefficient combustion of plastics and hazardous pharmaceuticals can generate toxic dioxin/dibenzofuran emissions that potentially pose a health hazard. Waste pharmaceuticals are hazardous and cytotoxic drugs may require temperatures as high as 2000°F (1093°C) to ensure complete degradation. Hospital incinerators of older design did not have the capability of complete destruction of these compounds. Incomplete combustion of these substances may therefore produce emissions of hydrochloric acid, chlorine gas, dioxins and furans among others.

POLLUTANTS RESULTING FROM INCINERATION

A study undertaken by the Institute for Environment and Health (IEH)[8] has identified a list of over 60 pollutants, or groups of pollutants which are generated from incineration of municipal, clinical, chemical waste and sewage sludge. This report and others such as the Royal Commission on Environmental Pollution report in 1993[9] have identified the main pollutants from incineration as particulates, metals and acidic and combustion gases and organic compounds (table 7.3).

Table 7.3: All Identified Pollutants from Incineration

POLLUTANT TYPE	IDENTIFIED INDIVIDUAL POLLUTANTS	
Particulates	Smoke, soot dust, PM10, total particulates.	
Metals	Aluminium, antimony, arsenic, barium, beryllium, cadmium, chromium, cobalt, copper, iron, lead, manganese, mercury, nickel, selenium, strontium, thallium, tin, titanium, vanadium, zinc.	
Acid and combustion gases	Carbon monoxide, carbon dioxide, hydrogen chloride, hydrogen fluoride, oxides of nitrogen, and sulphur oxides.	
Organic compounds	1,1,1-trichloroethane 1,1,2,2-tetrachloroethane 1,1,2-dichloroethane 1,1-dichloroethane 1,2-dichloroethane 1,3-butadiene 2,4-dinitrophenol acetonitrile acrolein acrylonitrile benzene bis(2-ethylhexyl)phthalate bromomethane butan-2-one carbon tetrachloride chlorobenzenes chloroform	chloromethane clorophenols dichlorodifluoromethane dichloromethane diethyl phthalate ethylbenzenes formaldehyde hexachloroethane phenol polybrominated dibenzodioxins/furans polychlorinated biphenyls polychlorinated dibenzodioxins/furans polycyclic aromatic hydrocarbons tetracholroethene toluene trichloroethene vinyl chloride xylenes

TOXIC EFFECTS OF COMMON POLLUTANTS

The IEH evaluated ten pollutants produced by incineration and considered to be important in terms of their effect on human health. As with much of toxicology, data on health effects generally relates to occupational, accidental or deliberate high dose exposures. Furthermore there is little published on the effects of low-dose exposure to mixtures of chemicals as occurs in incinerator emissions, rather than exposure to a single chemical.[8] While chemicals may be regarded as carcinogenic, based on animal or epidemiological studies, studies on their effects at environmental levels are not easily found. Many of the chemicals are present in the environment and it may not be possible to establish the excess levels of exposure due to incinerator emissions. For example, in 1989 the Department of the Environment estimated that municipal waste incinerators operating in the UK contributed approximately a third of dioxins in air.[10] In this report, human intake of dioxins is thought to be almost wholly attributable to intake from food. Furthermore little evidence existed to show that exposure to dioxins occurred at concentrations likely to be produced by incineration plants that would cause adverse health effects considering evidence following the Seveso disaster.

The ten pollutants considered by the IEH were selected on the likelihood of their being produced by waste incineration, in amounts likely to exceed short-term (SAQS) and long-term (LAQS) air quality standards, bioaccumulation, environmental persistence and on their inherent toxicity. The report does emphasise that these ten are not the only pollutants that should be considered in a formal risk assessment, as others may be of greater importance depending on the circumstances. While concerned primarily with municipal waste emissions the IEH list does include emissions from clinical waste incineration and other sources.
A summary of this information with additional data has been included in Appendix A3.

TYPE OF WASTE INCINERATED AT FAZAKERLEY HOSPITAL

- Most of the waste incinerated at Fazakerley was clinical waste[12]. It should be noted that in line with earlier guidance issued by the DOH the hospital did not segregate waste. All waste arising from wards and clinical departments was treated as clinical waste and incinerated.
- During the period up to 1989 no records exist but it is known that there was a small amount of waste from non-clinical agencies (less than 0.1%) including the Police and HM Customs and Excise. From 1989 to 1995 significant amounts of waste from sources outside the hospital were burned. Most clinical waste derived from other hospitals, domestic collections and other healthcare activities. The nature of the material burned would have varied: that originating in hospitals was likely to have had a high plastics content while that from nursing homes would be likely to have had a high moisture content, e.g. incontinence pads.
- In addition there was incineration of waste paper, tobacco for a local manufacturer from 1990-93 (at that time classified as general waste), date-expired drugs from the hospital pharmacy (although probably not cytotoxics for which they were licensed). Small quantities of low level radioactive Carbon 14 waste were disposed of which originated in the Public Health laboratory situated at the hospital. The site was licensed under the Radioactive Substances Act 1960 for the disposal of incineration of very low radioactive waste. Detailed records are still held of the quantities of Carbon 14 incinerated.
- As waste was not segregated into different streams, this would have increased the amount of general waste, so diluting the effect of the plastics and chlorinated substances in the waste being incinerated.

7.5. Current Government Policy on Waste Disposal

Currently the government supports waste minimisation, segregation, re-use and recycling where this is possible. It is committed to setting and enforcing high environmental protection standards and also to using waste as a renewable energy source, known as energy from waste (EfW) technologies. Extracting energy from waste would reduce consumption of fossil fuels and of methane production in landfill sites. However there is much public concern about the hazardous effects to human health due to the combustion products of the incineration process. The concern exists despite the recent more stringent regulations imposed by the government on location, operation and permitted emissions of incinerators.

THE CURRENT STANDARDS

New regulations require standards for planning and siting, testing and monitoring of chemical emissions, operator training and qualification and waste management plans. (Tables 7.5, 7.6, 7.7)

Table 7.5: Release limits for non-continuous monitoring: further guidance is given for interpretation of limits on continuous monitoring

EMISSIONS	CONCENTRATION *PG5/1(91)* <1 tonne per hour	CONCENTRATION *PG IPR 5/2(92)* >1 tonne per hour	CONCENTRATION *PG5/1(95)* <1 tonne per hour	Recommended frequency of monitoring
Total particulate matter	100mg/m^3	30 mg/m^3	30 mg/m^3	Continuous
Hydrogen chloride	100mg/m^3	30 mg/m^3	30 mg/m^3	Every 6 months
Hydrogen fluoride		2 mg/m^3		
Sulphur +- dioxide	300mg/m^3	300 mg/m^3	300 mg/m^3	Every 6 months
Carbon monoxide	100mg/m^3		50 mg/m^3 daily average	Continuous
NOx		350 mg/m^3		
VOCs		20 mg/m^3	20 mg/m^3	Every 6 months
Dioxins		1 ng/m^3 TEQ Should aim to achieve a guide TEQ value of 0.1ng/m^3	1 ng/m^3 TEQ	Once a year
Cadmium		0.1 mg/m^3	0.1 mg/m^3	Once a year
Mercury		0.1 mg/m^3	0.1 mg/m^3	Once a year
Other heavy metals (arsenic, chromium, copper, lead, manganese, nickel, tin) taken together	5mg/m^3 Total of cadmium, mercury, arsenic, chromium, copper, lead, manganese, nickel expressed as metal	1.0 mg/m^3	1.0 mg/m^3	Once a year

Emissions should be free from offensive odour and from visible smoke

Table 7.6: Standards for incinerator emissions in the EU

EMISSION	DAILY AVERAGE (mg/m^3)	HOURLY AVERAGE (mg/m^3)	4-HOUR AVERAGE (mg/m^3)
Total dust	5	10	
Total organic carbon	5	10	
Chlorine compounds	5	10	
Fluorine compounds	1	2	
Sulfur oxides as SO_2	25	50	
Nitrogen oxides as NO_2	100	200	
Carbon monoxide	50	100	
Mercury	-	-	0.05
Cadmium and thallium	-	-	0.05
Lead, chromium, copper, manganese	-	-	0.5
Nickel and arsenic	-	-	0.5
Antimony, cobalt, vanadium, tin	-	-	0.5
Dioxins and furans	-	-	0.1
Oxygen content		At least 6% at any moment	

Table 7.7: Prescribed substances from the incineration process (1 tonne or more per hour) which should be identified and quantified by the applicant

FOR AIR	FOR WATER	FOR LAND
Particulate matter	Mercury and its compounds	Organo-metallic compounds
Metals, metalloids and their compounds	Cadmium and its compounds	Polychlorinated dibenzo-p-dioxins (PCDDs)
Oxides of sulphur and other sulphur compounds		Polychlorinated dibenzofurans (PCDFs)
Oxides of carbon		Polychlorinated diphenyls (PCBs)
Halogens and their compounds		Alkaline earth metal oxides
Organic compounds and partial oxidation products		

Research undertaken for The Public Acceptability of Incineration by the National Society for Clean Air and Environmental Protection project suggests that, given the new stringent emission limits for incinerators, the potential for impacts on the health of local populations is extremely small.(13) Even where this is accepted, however, regulators need to develop compliance procedures, which can demonstrably enhance public confidence.

7.6. Conclusions of the Toxicology Review

There are a number of lessons to be learned from the investigation of the Fazakerley incinerator incident:

- **Regulation**

With the advantage of hindsight and more recent legislation in place, it should be remembered that when the Fazakerley incinerator closed down in 1995, it met or was better than the standards, which were in place at the time.(14) Provisions in the legislation and the existence of Crown Immunity prior to 1990 did not require emissions from hospital incinerators to be regulated or monitored. Current UK legislation will in future strictly regulate permitted levels of emissions from incinerators, and the Environment Agency will be required to co-consult with Health Authorities. The latter are expected to anticipate and assess the health impact of emissions on their local populations.

- **Need for Monitoring**

The lack of regulatory legislation and monitoring requirements for older incinerators prior to 1990 means that little data exists on the chemical emissions or their dispersion paths. Studies associating incinerators with specific disease entities are frequently based on proximity to the incinerators; associations cannot be accurately attributed to specific emissions or assessed for the effects of different concentrations of a chemical. This investigation points to the need for environmental monitoring and control in order to provide the necessary scientific tools to interpret public health concerns adequately.

- **Health Impact Assessment**

In the case of existing and proposed future incineration plants, all agencies with statutory responsibility will need to have knowledge of pertinent legislation and regulations. In the event of future planning of waste incineration plants, specialist multidisciplinary, multi-agency knowledge should influence the decision of whether incineration is the best option. Environmental and health impact assessments will increasingly be required to be undertaken involving the Environment Agency, Local Authority, Health Authorities, CIRS and attention given to health impacts on populations.(15)

- **Addressing Public Concern**

Public concern about the hazards of emissions existed for many years but there was little acknowledgement of this. There has been a sea change in attitudes to the public concerns about environmental impact on health. The government has called for multi-agency consultation and public participation assessing health impacts of new major developments(16) and review of existing industries by either the Environment Agency or the Local Authority under Pollution and Prevention Control legislation. It should be emphasised that the health impacts from waste incineration are still not well understood(17,18,19,20) and the Environment Agency is working with the Department of Health to prepare national protocols and standards to assess health impacts.

8 REVIEW OF CANCER INCIDENCE IN FAZAKERLEY 1974 TO 1998

8.1. The Role of the Merseyside and Cheshire Cancer Registry

Merseyside and Cheshire Cancer Registry (MCCR) has been used as the main source of information about cancer cases because it holds comprehensive computerised register of cancer cases for Merseyside and Cheshire residents. Data come to the cancer registry from a variety of sources including, pathology reports, death certificates, hospital records, and other cancer registries. MCCR also receives information from specialised tumour registries, including the National Childhood Cancer Registry with which data are regularly cross-checked.

MCCR was asked to undertake a study into possible cancer risks associated with emissions from the incinerator operating at Aintree NHS Hospital Trust between 1974 and 1995. The study has focussed upon the Fazakerley and Gillmoss (L9, L10 and L11) areas of Liverpool, as those would be the most likely to have been affected by emissions. The study area has a population of around 78,000 (1991) and accounts for approximately 16% of the population of Liverpool Health Authority (LHA).

MCCR were asked to try to answer the following question:

Are the cancer incidence rates in the study area significantly different from those in LHA and Merseyside and Cheshire as a whole?

The incidence rate (or incidence) which has been used extensively in the analyses, is generally expressed as the number of cases per 100,000 of the population.

8.2. Methods

All cases of primary cancer occurring in Merseyside and Cheshire residents between 1974 and 1998 inclusive were selected for the study.

Postcode was used to assign cases to the three comparison areas:

The study area (postcode districts: L9, L10 and L11)

Liverpool Health Authority

Merseyside and Cheshire

Additional postcodes were manually obtained from address details held on the MCCR database, thereby completing the case finding for the three areas. Cases from subsidiary sources including occupational health records and newspaper reports were only included if they were already registered with MCCR.

Cancers have been selected for inclusion in this study based upon the results of the literature search (see Section 5) and the specific concerns expressed by local residents.

The cancers of interest are shown in Table 8.1 below:

Table 8.1. Cancers of Interest Included in the MCCR Study

ADULT CANCERS	CHILDHOOD CANCERS
Lung (including trachea and bronchus)	Leukaemias
Liver	Lymphomas
Leukaemias	Other sites combined
Lymphomas	All cancer sites
Larynx	
Nasopharynx	
Soft and connective tissue (about 90% of which are sarcomas)	
All cancer sites	

The following cancers were included in the analyses for comparison purposes, because neither are reported in the literature to be associated with emissions from incinerators:

- Colon
- Rectum

Data items for each case selected from the MCCR database included, gender, age at diagnosis, postcode, health district of residence, year of diagnosis, tumour site, and smoking status at diagnosis.

ANALYSIS

In order to calculate incidence rates, population data were needed for the three comparison areas: the study area, LHA, and Merseyside and Cheshire. Population data for the study area were only available for 5-year age bands for census years 1981 and 1991 within the overall study period. Census year populations were available for enumeration districts, which were combined into the postcode districts L9 - L11, to form the population for the study area. The two known census year populations were then used to estimate the study area population over time, focussing on the two five year periods 1979-83 and 1989-93, for which the census year was the mid-point. The 1981 census data were also used, in further analyses, to estimate the population for the years 1974-86, and 1991 census data were used to estimate the population for the years 1987-98. Rather than using mid-year population estimates for LHA and Merseyside and Cheshire available from the Office for National Statistics, populations for these two areas were estimated in the same way as for the study area to minimise bias in subsequent calculations.

Most of the analyses relate to the 5-year periods 1979-83 and 1989-93, for which the population estimates are most accurate. In addition, especially within the study area and for rare cancer sites, numbers of cases for individual years are too small to permit meaningful analyses. For the rarest sites, analyses have used the whole period 1974-98. Rates for the other 5 year periods are included within the tables in this report for completeness in the Statistical Appendix, but caution should be used when attempting to interpret these rates due to the difficulties already described with estimating the population.

Three types of incidence rate have been calculated:

- Crude Rate is simply the number of cancers per 100,000 of the population, and does not account for any differences in the age structure of the population. It is not appropriate to compare crude rates between geographical areas where the age structure of the population differs, as here. Crude rates have been included in the tables in the Statistical Appendix for completeness, but were not therefore used to compare between Merseyside and Cheshire, LHA and the study area.
- Cumulative Rate (0-74 years) is equivalent to the probability of developing cancer before the age of 75 years and accounts for differences in age structure in a population, thereby allowing meaningful comparisons between populations with different age structures.
- Age Standardised Rates are rates that account for different age structures within populations and include all ages. Details of how these rates are calculated can be found in the Statistical Appendix.

Socio-economic group was assigned as Super Profile group[1] based on enumeration district (ED) for 1991 for cases and populations for each of the three comparison areas. Cancer rates for each socio-economic group and area were calculated and adjusted for age, where the number of cases permitted.

NOTES ON POPULATION ESTIMATES

The EDs in existence in 1991 do not map exactly to postcode districts. For the EDs that cut across a postcode district, the proportion of the ED population within the relevant postcode has been used to adjust the population estimate. These proportions were provided by the Merseyside Information Service.

A direct link between the EDs in existence in 1981 and the postcode district is not available. Population estimates for 1981 were therefore converted into 1991 EDs using a link provided by Manchester Information and Associated Services (MIMAS). The EDs were then converted into postcode districts using the link between 1991 EDs and postcode districts described above.

8.3. Results

Using the postcode on the cancer registry database, a total of around 10,490 cancers were initially identified by MCCR as occurring in residents of the study area. Manually generating postcodes from address details yielded a further 3,000 potential cases of which 100 were found on further checking to be from the study area. Thus, **10,590** cancers were diagnosed between 1974-98 in individuals resident in the study area, of which 5,406 (51%) occurred in males and 5,184 (49%) in females.

Table 8.2 shows the number of cases by site and 5 year period for the study area for males and females separately.

For the study area, analyses for the following rare cancers are shown throughout for the whole period 1974-98:

- Nasopharynx
- Liver
- Hodgkins lymphoma
- Soft and connective tissue
- Female laryngeal cancers

Total includes 10,085 primary cancers and 505 known secondary cancers with an unknown primary site.

Table 8.2. The Number Of Cancer Cases in L9, 10 and 11 (Males And Females) by site and 5Year Period

MALES

SITE	1974-1978	1979-1983	1984-1988	1989-1993	1994-1998	1974-1998
All malignant cancers	1083	1042	1050	1075	1156	5406
Trachea, bronchus And lung†	399	340	312	279	269	1599
Larynx†	20	16	17	16	21	90
Nasopharynx†	0	2	1	0	3	6
Liver†	4	7	6	11	12	40
Leukaemias†	16	16	25	15	23	95
Non-Hodgkins lymphoma†	16	10	19	20	26	91
Hodgkins lymphoma†	4	7	4	8	3	26
Soft andconnective Tissue†	5	3	7	7	6	28
Colon†	53	61	58	75	67	314
Rectum†	63	50	53	41	63	270

† Primary cancers only

FEMALES

SITE	1974-1978	1979-1983	1984-1988	1989-1993	1994-1998	1974-1998
All malignant cancers	953	991	1048	1057	1135	5184
Trachea,bronchus And lung†	117	114	152	185	202	770
Larynx†	7	2	6	9	6	30
Nasopharynx†	0	1	0	1	0	2
Liver†	7	0	4	8	11	30
Leukaemias†	17	12	9	14	23	75
Non-Hodgkins lymphoma†	10	9	21	26	21	87
Hodgkins lymphoma†	5	5	4	2	2	18
Soft and connective tissue†	5	3	5	4	6	23
Colon†	68	84	84	54	65	355
Rectum†	54	39	51	44	39	227

† Primary cancers only

Table 8.3 shows the total number of cases of childhood cancer (aged 0-14 years) within the study area. **Again, the numbers of cases by site for childhood cancers are too small to enable meaningful analyses**. Thus in the following analyses, all childhood cancer sites have been combined, and will be looked at for the whole period 1974-98. The numbers of childhood cancers are so small overall (n=45) in comparison to the numbers of adult cancers (n=10,545), that data presented for all ages (0-74 years) are almost identical to those presented for adults (15-74 years) separately.

Table 8.3: The Number of Childhood Cancers within L9, 10 and 11 by site and 5 year period

MALES

SITE	1974-1978	1979-1983	1984-1988	1989-1993	1994-1998	1974-1998
All malignant cancers	3	3	5	7	9	27
Leukaemias†	1	1	3	2	5	12
Non-Hodgkins lymphoma†	0	0	1	1	0	2
Hodgkins lymphoma†	0	1	1	0	2	4

† Primary cancers only

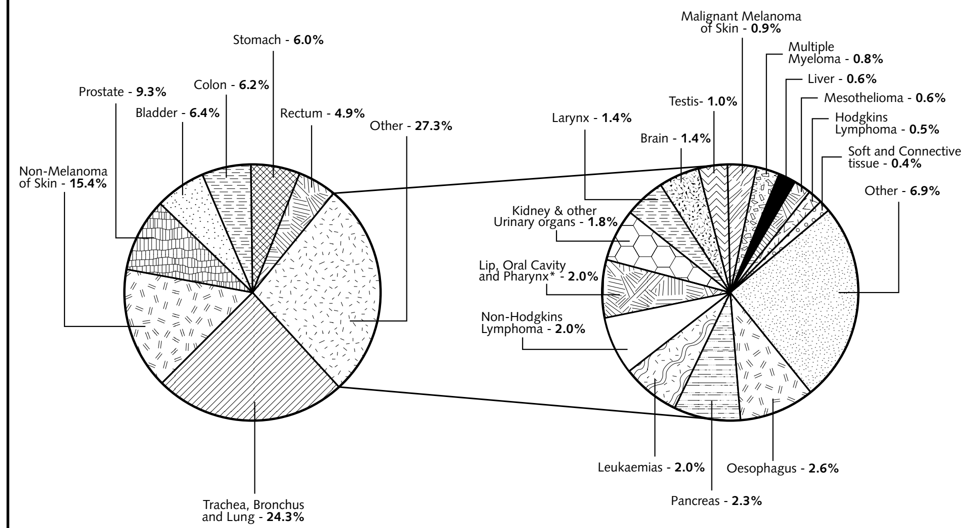
FEMALES

SITE	1974-1978	1979-1983	1984-1988	1989-1993	1994-1998	1974-1998
All malignant cancers	5	2	3	3	5	18
Leukaemias†	2	1	0	0	1	4
Non-Hodgkins lymphoma†	0	0	0	1	1	2
Hodgkins lymphoma†	0	0	0	0	0	0

† Primary Cancers only

Figures 8.1 and 8.2 show for males and females respectively, the distribution of cancers by site within Merseyside and Cheshire for 1974-98. Although not shown, the distribution for both the study area and LHA was not significantly different from this, with the exception of lung cancer, for which there were higher proportions in males and females in the study area (30% in males, 15% in females) and LHA (28% in males and 14% in females). It can also be seen that some of the cancers included in this study eg leukaemias (2%) are comparatively rare.

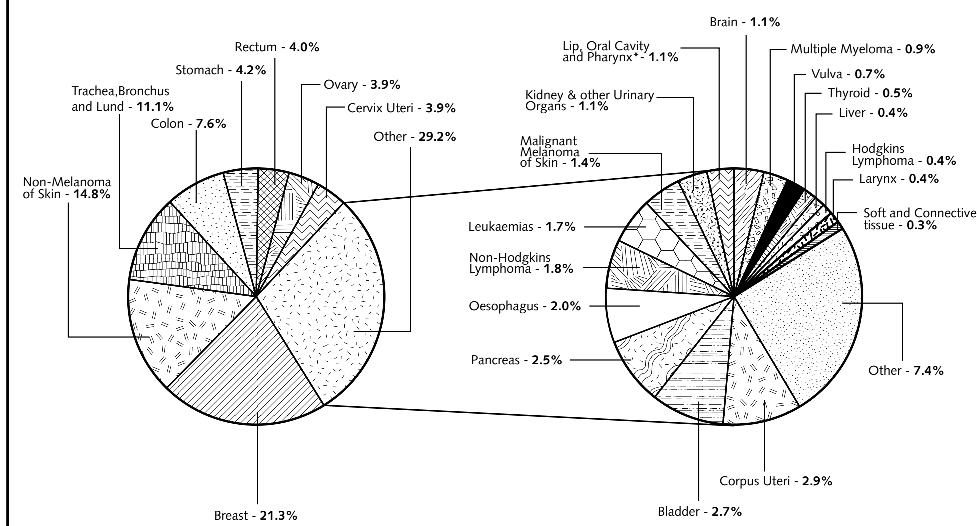
Figure 8.1: Merseyside and Cheshire Cancers 1974-1998, all ages, by site - MALES



*includes Nasopharanx

Merseyside and Cheshire Cancer Registry - August 2001

Figure 8.2: Merseyside and Cheshire Cancers 1974-1998, all ages, by site - FEMALES



*includes Nasopharanx

Merseyside and Cheshire Cancer Registry - August 2001

Figure 8.3 shows that the number of cases of cancer occurring in the study area has remained reasonably constant, at about 400 each year over the period 1974-98, and that a little over half of these occurred in men.

Figure 8.3: Number of cancers within the study area by year of diagnosis and gender 1974-1998

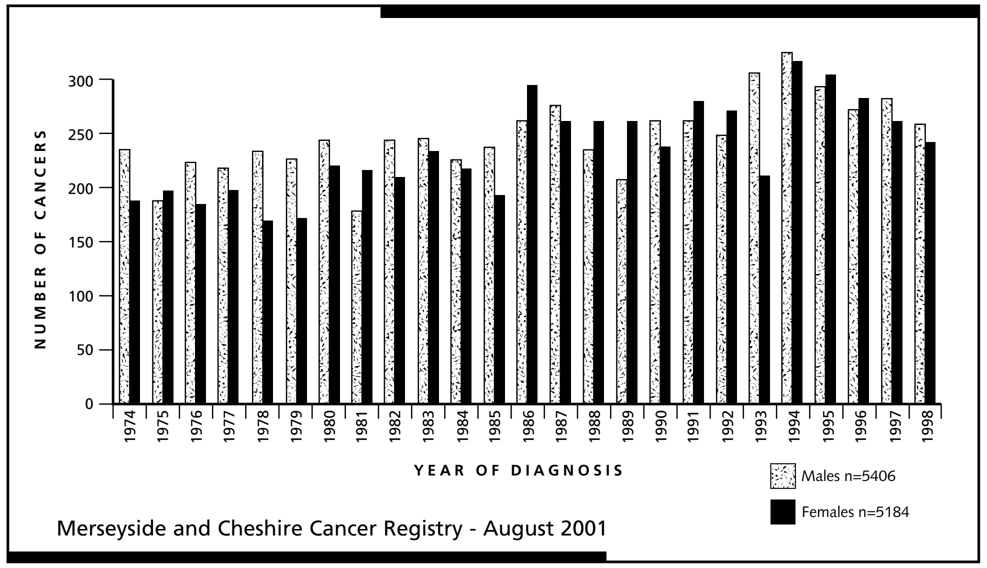


Figure 8.4 shows that the study area accounts for about 16% (1493/9280 in 1989-93) of the cancers in LHA, and that the risk of developing cancer between 15-74 years in the study area for the period 1989-93 is 1 in 3.0 and 1 in 3.5 for males and females respectively.

N.B. (The error bars shown in this and successive figures, represent the uncertainty around the rates due to small numbers, and show the range within which the true rate might lie; the lower the number of cases the larger the error bar, and conversely, the higher the number of cases the smaller the error bar. The error bars tend therefore to be narrowest for Merseyside and Cheshire rates and widest for study area rates, with LHA occupying a middle position in general. Overlapping error bars indicate that rates are not significantly different).

Male cancer rates are significantly higher than female cancer rates for both 5 year periods and in all three comparison areas. The study area and LHA have significantly higher rates of cancer overall compared with Merseyside and Cheshire for both time periods for males and for the earlier time period 1979-83 for females. In 1989-93, in females the study area had similar rates compared with Merseyside and Cheshire. In both time periods, the study area rates however are not significantly different from those of LHA for either males or females.

Figure 8.4: Cumulative rate for all adult cancers (aged 15 - 74 years) by area and gender for 1979-1983 and 1989-1993

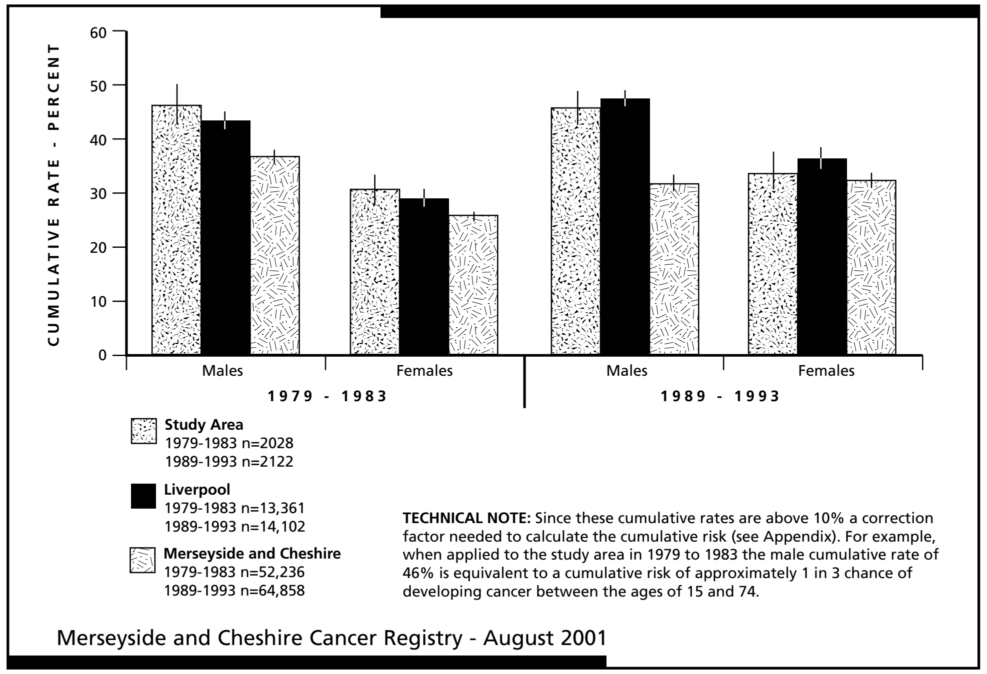


Figure 8.5 shows that the study area contributes about 17% (45/263) of LHA's childhood cancers, and that the risk of developing cancer by the age of 15 years for individuals in the study area is 1 in 500, and 1 in 1,000 for males and females respectively. Males rates are higher than female rates in all three comparison areas, although this gender difference is significant only in Merseyside and Cheshire as a whole. There are no significant differences between the comparison areas for childhood cancers taken altogether.

Figure 8.5: Cumulative rate for all childhood cancers (aged 0-14 years) by area and gender for 1974-1998

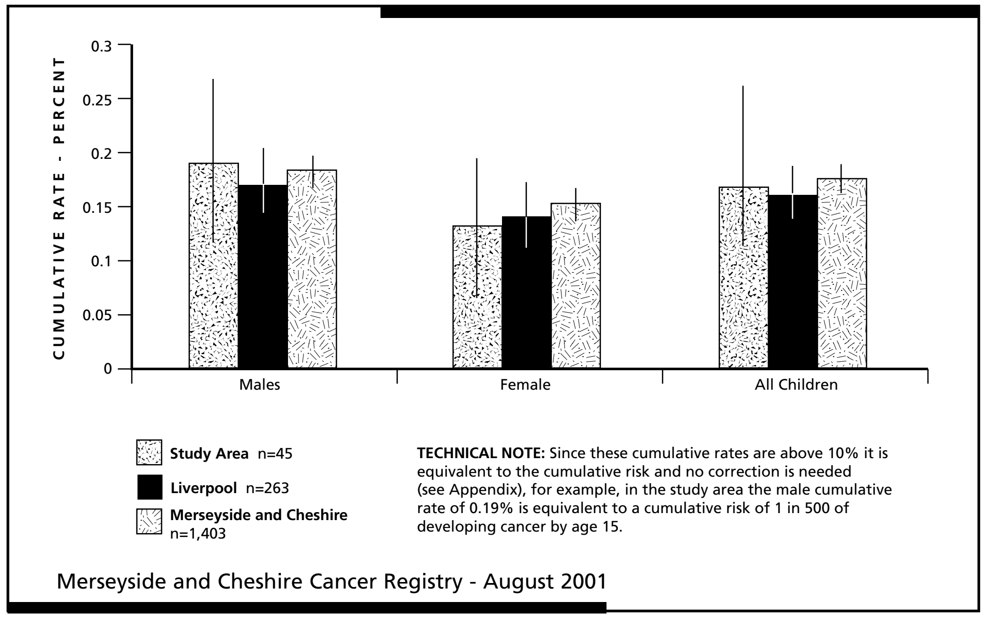


Figure 8.6 shows that in 1979-83, in males, for leukaemias, non-Hodgkins lymphoma, and cancers of the larynx, colon and rectum, there are no significant differences between the three comparison areas. The exception to this general pattern is lung cancer, which has significantly higher rates than the other selected cancers, and for which both study area and LHA rates are significantly higher than Merseyside and Cheshire. The study area and LHA are however not significantly different from one another. Figure 8.7 shows that this general pattern is still evident in the later period 1989-93, although actual levels have changed slightly.

Figure 8.6: Cumulative rate for male cancers, aged 0-74 years, by common selected sites and area 1979-1983

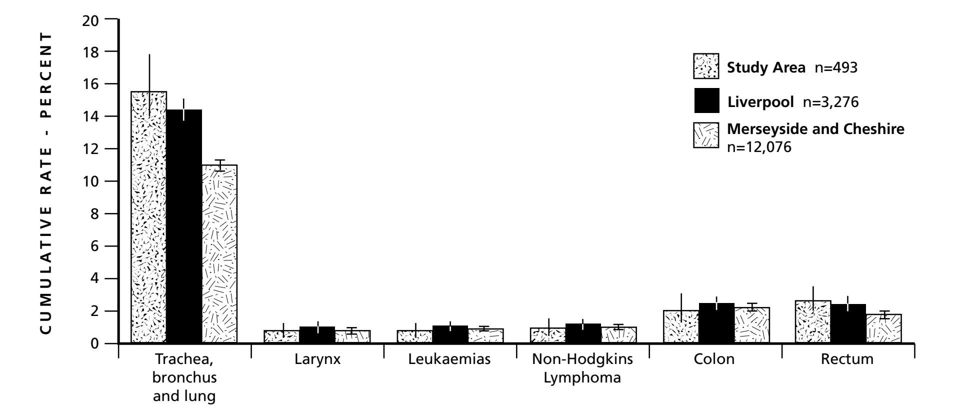


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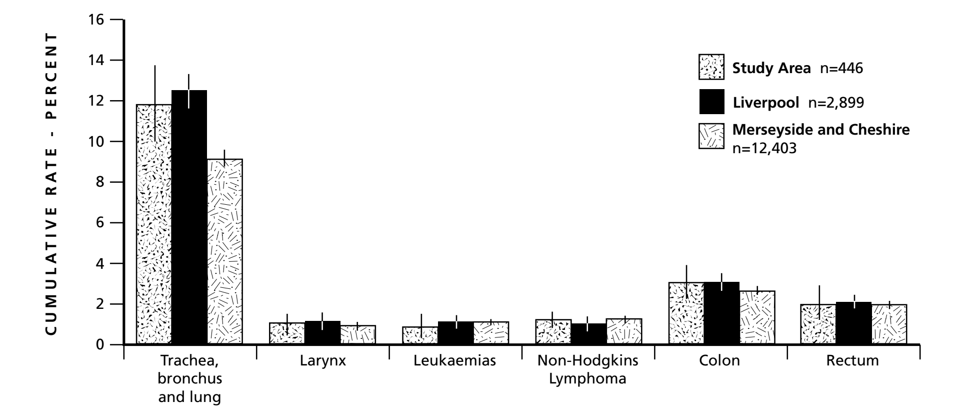
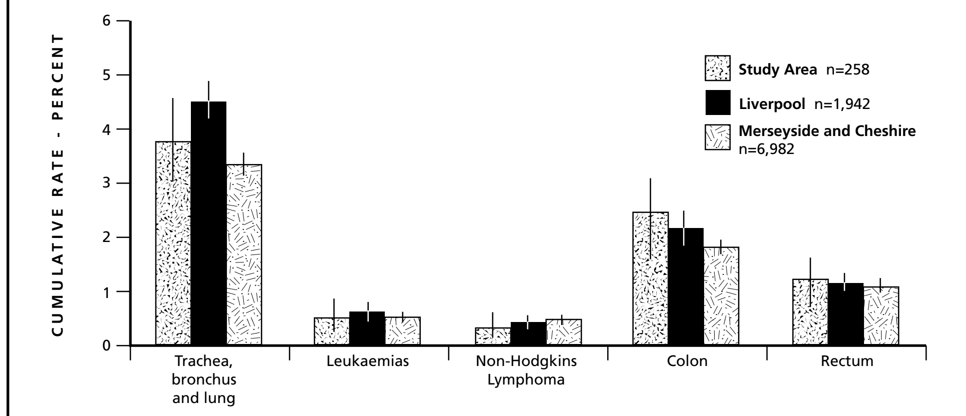
Figure 8.7: Cumulative rate for male cancers, aged 0-74 years, by common selected sites and area 1989-1993



Merseyside and Cheshire Cancer Registry - August 2001

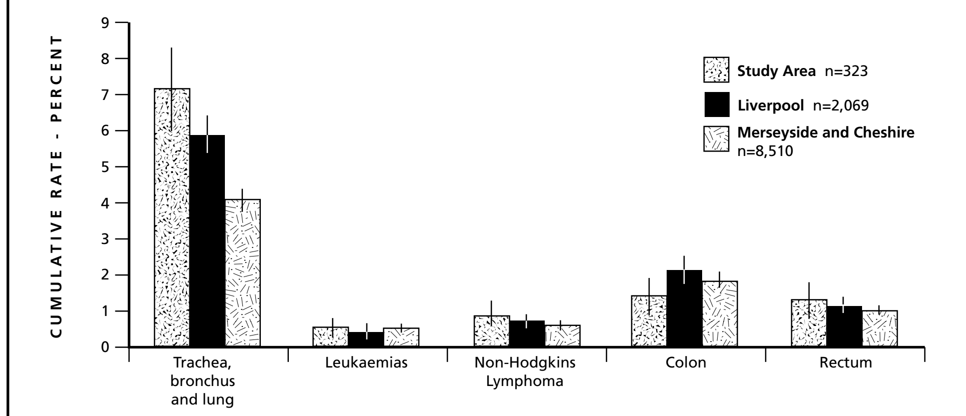
Figure 8.8 shows that in 1979-83, in females there are no significant differences between the three comparison areas for: leukaemias, non-Hodgkins lymphoma, and cancers of the colon and rectum. As for males, lung cancer rates are significantly higher than those of the other selected sites. Lung cancer in females is significantly higher in LHA overall compared with Merseyside and Cheshire as a whole. Rates in the study area are lower than in LHA, although this finding is not significant. Figure 8.9 shows the same general pattern appearing in the later time period (1989-93). It is noteworthy that female lung cancer rates in the study area now exceed those in LHA, although this finding is not significant. Lung cancer rates for both the study area and LHA are significantly higher than rates for Merseyside and Cheshire as a whole.

Figure 8.8: Cumulative rate for female cancers, aged 0-74 years, cy common selected sites and area 1979-1983



Merseyside and Cheshire Cancer Registry - August 2001

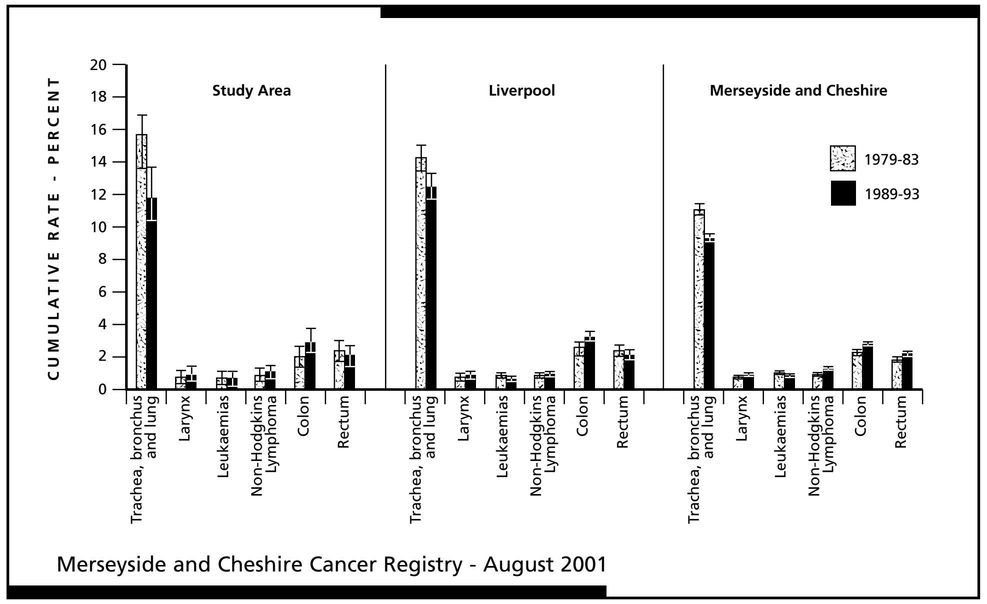
Figure 8.9: Cumulative rate for female cancers, aged 0-74 years, cy common selected sites and area 1989-1993



Merseyside and Cheshire Cancer Registry - August 2001

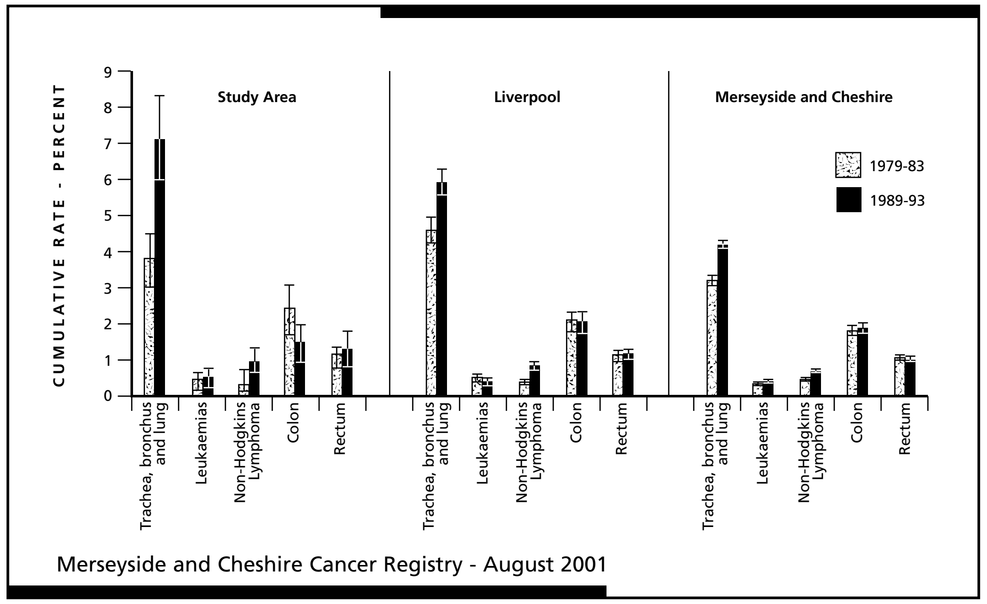
Figures 8.10 and 8.11 compare for males and females respectively, the two 5 year periods and three areas for the commoner selected cancers. In both figures, the predominance of lung cancer is evident, with rates significantly higher than the other selected sites. In Figure 8.10 overall the pattern of cancer between the time periods is similar in the three comparison areas. For example, in all areas, lung cancer in males has declined significantly between 1979-83 and 1989-93 time periods. In contrast, in all areas colon cancer in males has increased, significantly in LHA and Merseyside and Cheshire as a whole. Figure 8.11 also shows similarities in the way in which the site-specific rates vary between the two 5 year periods for the three areas. Although, lung cancer in females has increased significantly in all three comparison areas, the most striking increase is to be found in the study area, where rates have increased significantly from 3.8% (95% confidence intervals 3.0-4.6) to 7.1% (95%CI 6.0-8.3).

Figure 8.10: Cumulative rate for male cancers, aged 0-74 years, by common selected sites and area for 1979-1983 and 1989-1993



Merseyside and Cheshire Cancer Registry - August 2001

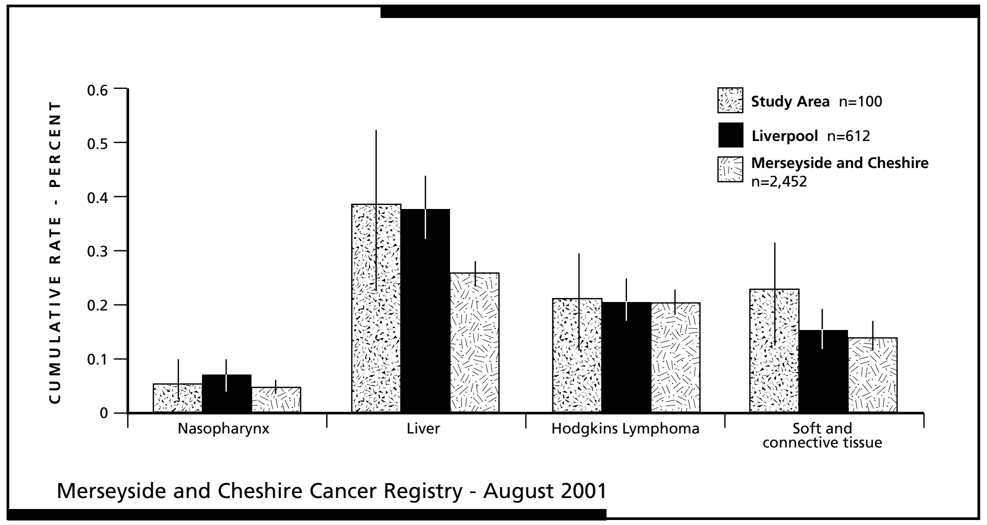
Figure 8.11: Cumulative rate for female cancers, aged 0-74 years, by common selected sites and area for 1979-1983 and 1989-1993



Merseyside and Cheshire Cancer Registry - August 2001

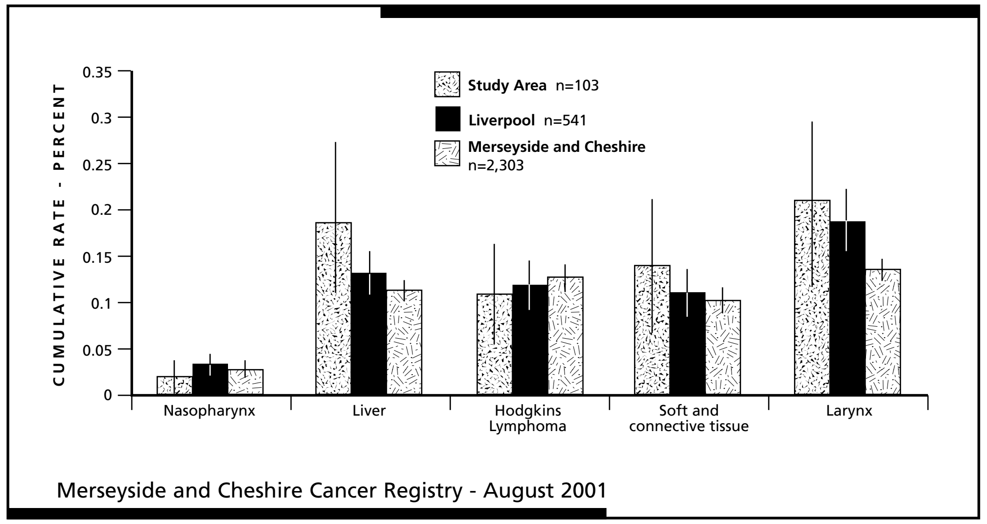
Figures 8.12 and 8.13 show how rates vary between areas for the rare cancers included in the study, for males and females respectively. There are no significant differences between LHA and the study area for rates of cancers of the nasopharynx, liver, Hodgkins lymphoma, and soft and connective tissue, for males or females, and for laryngeal cancer in females. In general, rates for Merseyside and Cheshire are also not significantly different from LHA or the study area. The two exceptions to this are, liver cancer in males, and laryngeal cancer in females for both of which the LHA rate is significantly higher than that for Merseyside and Cheshire as a whole.

Figure 8.12: Cumulative rate for male cancers, aged 0-74 years, by rare selected sites and area 1974-1998



Merseyside and Cheshire Cancer Registry - August 2001

Figure 8.13: Cumulative rate for female cancers, aged 0-74 years, by rare selected sites and area 1974-1998



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The detailed data on the number of cases, cumulative rates and age standardised rates by each 5 year period between 1974-98 for the commoner sites selected for this study appears in the Statistical Appendix

Table 8.4 shows that the study area and LHA have similar socio-economic profiles, for males and females and that both areas are more deprived compared with Merseyside and Cheshire as a whole.

Table 8.4: Comparison of 1991 population estimates by socio-economic group (S/E group)

DESCRIPTION	S/E GROUP	MALES 1991 POPULATION			PROPORTION WITHIN THE S/E GROUP		
		STUDY AREA	LIVERPOOL	MERSEYSIDE & CHESHIRE	STUDY AREA	LIVERPOOL	MERSEYSIDE & CHESHIRE
Affluent achievers	1	0	9519	135941	0%	4%	12%
Thriving greys	2	859	10461	101394	2%	5%	9%
Settled suburbans	3	2569	15926	162104	7%	7%	14%
Nest builders	4	7585	29442	200091	20%	13%	17%
Urban venturers	5	778	15108	22452	2%	7%	2%
Country life	6	0	0	14458	0%	0%	1%
Senior citizens	7	545	10094	58730	1%	4%	5%
Producers	8	2344	14975	140002	6%	6%	12%
Hard pressed families	9	6048	30228	108167	16%	13%	9%
Have nots	10	16595	95884	225339	44%	41%	19%
Not known	11	0	464	656	0%	0%	0%
		37322	232101	1169335	100%	100%	100%

DESCRIPTION	S/E GROUP	FEMALES 1991 POPULATION			PROPORTION WITHIN THE S/E GROUP		
		STUDY AREA	LIVERPOOL	MERSEYSIDE & CHESHIRE	STUDY AREA	LIVERPOOL	MERSEYSIDE & CHESHIRE
Affluent achievers	1	0	10373	143291	0%	4%	11%
Thriving greys	2	904	12230	112451	2%	5%	9%
Settled suburbans	3	2693	17247	170131	7%	7%	14%
Nest builders	4	8091	31296	205594	20%	13%	16%
Urban venturers	5	759	14224	21653	2%	6%	2%
Country life	6	0	0	14375	0%	0%	1%
Senior citizens	7	687	11246	68542	2%	5%	5%
Producers	8	2623	16734	153497	6%	7%	12%
Hard pressed families	9	6486	31776	113090	16%	13%	9%
Have nots	10	18116	103389	243437	45%	42%	20%
Not known	11	0	133	395	0%	0%	0%
		40358	248648	1246456	100%	100%	100%

* Socio-economic group is based on Super profile category

Comparisons between the three study areas by socio-economic group were performed only for lung cancer because numbers were too small for the other cancers selected to permit meaningful analysis and interpretation. Figures 8.14 and 8.15 show for males and females respectively, that there is a socio-economic gradient for lung cancer in the three comparison areas, with group 1 (most affluent) having lower rates of lung cancer than group 10 (most deprived). There are no significant differences in lung cancer rates between the study area and LHA for either males or females, although error bars are wide, especially for the study area, due to small numbers of cases in some socio-economic groups. With the exception of group 7 in males, in which statistical significance is achieved, there are no significant differences between the study area and Merseyside and Cheshire as a whole. Lung cancer rates in females in the study area are however higher in most socio-economic groups for which there are sufficient numbers of cases, compared with LHA and Merseyside and Cheshire, although these findings are not significant. For males, this relationship is less straightforward. Merseyside and Cheshire lung cancer rates are significantly lower than rates in LHA only for males and females in group 8 (deprived). Tables 8.5a and b show for males and females respectively, lung cancer data for the three comparison areas and Figures 8.14 and 8.15.

Table 8.5a: Trachea, bronchus and lung cancers, 1989-1993, all ages, by area and socio-economic group (S/E group)

Males:

S/E GROUP	STUDY AREA				LIVERPOOL				MERSEYSIDE AND CHESHIRE			
		95% CI				95% CI				95% CI		
	N	RATE	LOWER	UPPER	N	RATE	LOWER	UPPER	N	RATE	LOWER	UPPER
1	**0**				**39**				**523**			
Crude rate		0.0	0.0	0.0		81.9	56.2	107.7		76.9	70.4	83.5
cumulative rate		0.0	0.0	0.0		6.5	3.8	9.1		5.3	4.7	5.9
ASR		0.0	0.0	0.0		72.8	49.6	96.0		65.7	60.0	71.4
2	**6**				**80**				**553**			
Crude rate		139.8	27.9	251.6		152.9	119.4	186.5		109.1	100.0	118.2
cumulative rate		8.4	1.4	15.4		10.5	7.5	13.4		6.3	5.6	6.9
ASR		86.7	9.0	164.4		124.8	97.2	152.5		77.2	70.7	83.8
3	**12**				**68**				**698**			
Crude rate		93.4	40.6	146.3		85.4	65.1	105.7		86.1	79.7	92.5
cumulative rate		6.2	1.5	11.0		5.7	4.0	7.5		6.8	6.1	7.4
ASR		78.7	33.2	124.1		75.2	57.0	93.5		84.5	78.1	90.9
4	**33**				**136**				**776**			
Crude rate		87.0	57.3	116.7		92.4	76.9	107.9		77.6	72.1	83.0
cumulative rate		8.5	4.9	12.2		9.3	7.4	11.2		8.4	7.7	9.1
ASR		113.9	74.2	153.6		104.5	86.7	122.3		105.1	97.5	112.6
5	**2**				**90**				**134**			
Crude rate		51.4	-19.8	122.6		119.1	94.5	143.8		119.4	99.2	139.6
cumulative rate		9.0	-4.0	21.9		14.9	11.3	18.5		14.1	11.2	17.0
ASR		66.7	-25.8	159.2		162.9	128.3	197.4		162.3	134.2	190.3
6	**0**				**0**				**55**			
Crude rate		0.0	0.0	0.0		0.0	0.0	0.0		76.1	56.0	96.2
cumulative rate		0.0	0.0	0.0		0.0	0.0	0.0		6.0	4.0	7.9
ASR		0.0	0.0	0.0		0.0	0.0	0.0		65.1	47.7	82.4
7	**15**				**105**				**509**			
Crude rate		550.5	271.9	829.1		208.0	168.2	247.8		173.3	158.3	188.4
cumulative rate		27.5	12.0	43.0		14.1	11.0	17.2		10.7	9.6	11.9
ASR		312.4	133.6	491.1		155.2	123.4	187.0		118.9	107.8	130.0
8	**26**				**180**				**1167**			
Crude rate		221.8	136.5	307.1		240.4	205.3	275.5		166.7	157.1	176.3
cumulative rate		16.6	9.4	23.7		14.8	12.2	17.3		10.3	9.6	11.0
ASR		167.9	100.7	235.1		168.2	142.4	194.0		119.7	112.6	126.9
9	**42**				**223**				**752**			
Crude rate		138.9	96.9	180.9		147.5	128.2	166.9		139.0	129.1	149.0
cumulative rate		10.1	6.2	13.9		11.9	9.9	13.8		12.2	11.1	13.2
ASR		143.2	99.0	187.5		153.9	133.2	174.6		147.2	136.3	158.0
10	**139**				**800**				**1674**			
Crude rate		167.5	139.7	195.4		166.9	155.3	178.4		148.6	141.5	155.7
cumulative rate		13.2	10.6	15.9		14.1	12.9	15.2		13.8	13.0	14.6
ASR		171.7	141.2	202.2		171.2	159.0	183.5		164.0	155.9	172.1

Table 8.5b: Trachea, bronchus and lung cancers, 1989-1993, all ages, by area and socio-economic group (S/E group)

Females:

S/E GROUP	STUDY AREA				LIVERPOOL				MERSEYSIDE AND CHESHIRE			
		95% CI				95% CI				95% CI		
	N	RATE	LOWER	UPPER	N	RATE	LOWER	UPPER	N	RATE	LOWER	UPPER
1	**0**				**18**				**235**			
Crude rate		0.0	0.0	0.0		34.7	18.7	50.7		32.8	28.6	37.0
cumulative rate		0.0	0.0	0.0		2.3	0.8	3.7		1.8	1.5	2.2
ASR		0.0	0.0	0.0		23.4	11.7	35.1		21.8	18.9	24.7
2	**5**				**46**				**283**			
Crude rate		110.6	13.7	207.6		75.2	53.5	97.0		50.3	44.5	56.2
cumulative rate		4.4	-0.1	8.9		3.8	2.3	5.3		2.3	1.9	2.6
ASR		67.9	2.1	133.8		48.5	33.1	63.9		27.0	23.6	30.4
3	**6**				**44**				**392**			
Crude rate		44.6	8.9	80.2		51.0	35.9	66.1		46.1	41.5	50.6
cumulative rate		3.7	0.7	6.6		2.5	1.4	3.5		3.2	2.8	3.6
ASR		37.4	7.1	67.8		32.7	22.5	42.8		35.9	32.3	39.6
4	**21**				**91**				**415**			
Crude rate		51.9	29.7	74.1		58.2	46.2	70.1		40.4	36.5	44.3
cumulative rate		5.6	3.1	8.1		5.2	4.0	6.4		3.9	3.5	4.4
ASR		62.0	35.1	88.8		56.1	44.1	68.1		43.2	38.9	47.4
5	**1**				**47**				**66**			
Crude rate		26.4	-25.3	78.0		66.1	47.2	85.0		61.0	46.3	75.7
cumulative rate		5.9	-5.7	17.5		6.1	3.9	8.4		5.6	3.9	7.4
ASR		47.3	-45.4	140.1		75.4	51.9	99.0		66.0	48.6	83.4
6	**0**				**0**				**30**			
Crude rate		0.0	0.0	0.0		0.0	0.0	0.0		41.7	26.8	56.7
cumulative rate		0.0	0.0	0.0		0.0	0.0	0.0		2.7	1.5	3.9
ASR		0.0	0.0	0.0		0.0	0.0	0.0		31.7	19.8	43.6
7	**10**				**67**				**323**			
Crude rate		291.0	110.6	471.3		119.2	90.6	147.7		94.2	84.0	104.5
cumulative rate		11.3	3.1	19.5		5.8	4.1	7.6		4.4	3.7	5.0
ASR		109.5	29.0	189.9		58.1	41.0	75.3		48.6	41.8	55.3
8	**19**				**114**				**605**			
Crude rate		144.9	79.7	210.0		136.3	111.2	161.3		78.8	72.5	85.1
cumulative rate		7.7	3.4	11.9		7.1	5.5	8.7		4.1	3.7	4.5
ASR		84.6	42.5	126.7		82.5	65.2	99.8		45.9	41.8	49.9
9	**25**				**119**				**376**			
Crude rate		77.1	46.9	107.3		74.9	61.4	88.4		66.5	59.8	73.2
cumulative rate		6.9	4.0	9.8		5.5	4.3	6.7		5.2	4.5	5.8
ASR		66.6	39.4	93.8		61.4	49.8	73.0		55.3	49.5	61.1
10	**96**				**480**				**979**			
Crude rate		106.0	84.8	127.2		92.9	84.5	101.2		80.4	75.4	85.5
cumulative rate		7.9	6.1	9.8		6.9	6.1	7.6		6.5	6.0	7.0
ASR		85.9	67.9	104.0		73.4	66.5	80.4		70.1	65.5	74.8

NB: All rates are based on 1981 and 1991 populations (1981 population has been used for the years 1974-1986 and 1991 population has been used for 1987-1991)

Figure 8.14: Trachea, bronchus and lung cancers in males by socio-economic group and area 1989-1993

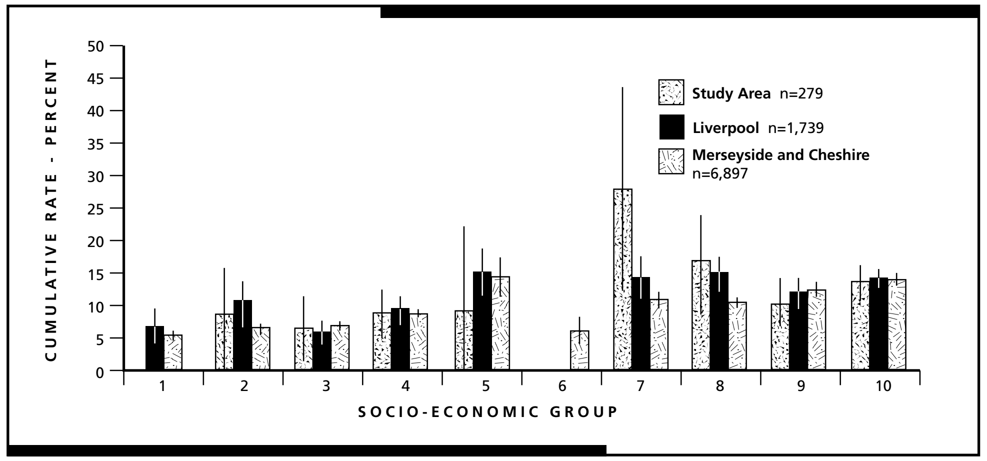


Figure 8.15: Trachea, bronchus and lung cancers in females by socio-economic group and area 1989-1993

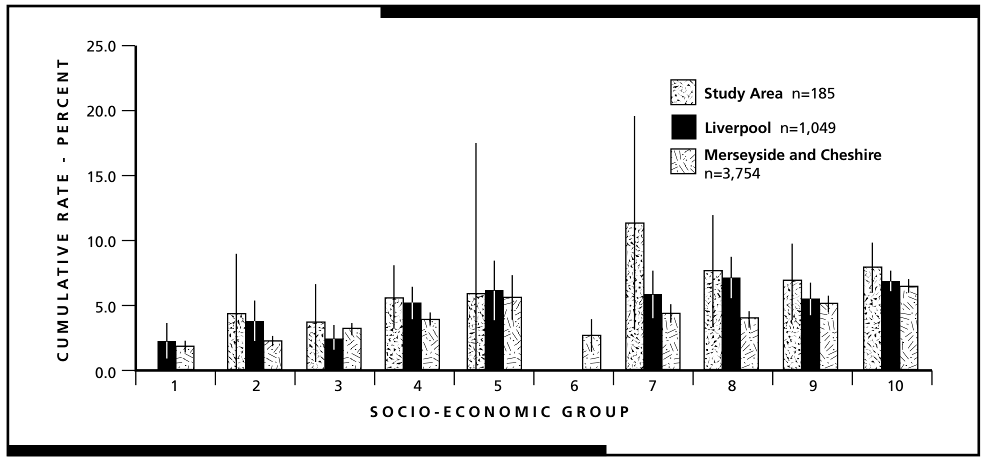


Table 8.6 below shows that the proportion of smokers amongst cases diagnosed with lung cancer is significantly higher in the study area and LHA than in Merseyside and Cheshire for both males and females for 1989-93.

Table 8.6: Proportion of smokers (male and female) diagnosed with Lung cancer (study area, LHA and Merseyside and Cheshire) between 1989-93

POPULATION GROUP	L9,L10,L11			LIVERPOOL			MERSEYSIDE AND CHESHIRE		
	NUMBER	PROPORTION	CONFIDENCE INTERVAL	NUMBER	PROPORTION	CONFIDENCE INTERVAL	NUMBER	PROPORTION	CONFIDENCE INTERVAL
Males	212 (279)	76%	70-82%	1291 (1739)	74%	72-77%	4620 (6897)	67%	66-68%
Females	128 (185)	69%	61-77%	687 (1049)	65%	62-69%	2226 (3754)	59%	57-61%
Persons	340 (464)	73%	69-78%	1978 (2788)	71%	69-73%	6846 (10651)	64%	63-65%

The number in brackets in the numbers column is the total number of lung cancers in each population group.

8.4. Discussion

Please see also Section 10 for summary of main findings and detailed discussion

METHODS USED

The problems associated with estimating the study area population have been minimised by limiting the main analyses to the 5 years around the census years 1981 and 1991 for which the population is known. These two periods (1979–83 and 1989–93) cover a 15 year period, including a period soon after the incinerator was first built in 1974. For solid tumours and, to a lesser extent, cancers of the lymphatic system and haematopoietic systems a lag period should be assumed between first exposure to a suspected cancer inducing agent and development of clinical disease. [2] In previous studies [2,3] lag periods of 5 and 10 years have been reported. Examination of the latter period (i.e. 1989–93) thereby allows for a lag of almost twenty years between exposure and potential health effect.

Given the small number of cases for certain sites, it was necessary to aggregate over the full time period to produce rates based on as large a number of cases as possible thereby reducing the uncertainty surrounding them. However, aggregation over a 25-year period has still resulted in some rates that have wide confidence intervals, reflecting the rarity of such cancers, and making interpretation more problematic.

LUNG CANCER IN FAZAKERLEY: ROLE OF SMOKING

The high rate of lung cancer in Liverpool has been known for some time and reported in several publications produced by MCCR. [4,5] Lung cancer has a strong association with deprivation,[3] which is associated with higher rates of smoking in more deprived areas. Smoking is known to be the major cause of lung cancer and is thought to account for 90% of cases.[3] **Higher deprivation and smoking rates account for the higher rates of lung cancer seen in Liverpool and similarly could account for the higher rates in the study area.**

Data on smoking status at diagnosis have been routinely collected by MCCR since 1988. Information on smoking is recorded when case notes are abstracted at hospital. A person is recorded as being a smoker if there is any information in the hospital case notes that states that they are a current or ex-smoker (regardless of the amount or type of tobacco they smoked). **Smoking data recorded by MCCR are dependent on hospitals recording this information, and as such are likely to underestimate the true proportions**. It is unlikely that bias, if present, would account for the higher levels recorded in the study area and LHA. Although population smoking rates would have been ideal, these are not available, nevertheless, the higher proportions of smokers amongst lung cancer cases in the study area and LHA, may act as a marker of higher smoking levels in these populations.

8.5 Study area and control area comparison

BACKGROUND

In addition to the comparative analysis of cancer data described above, Merseyside and Cheshire Cancer Registry in collaboration with colleagues at the Department of Civic Design at the University of Liverpool conducted a comparison between the L9, L10 and L11 study area and a control area, which has a similar socio-economic and age profile but is not believed to be affected by incinerator emissions.

METHODS

In selecting a control group, account was taken of confounding variables, such as social class, age and sex, which are important determinants of cancer. It was also important to minimise, as far as possible, the likelihood that the control area was affected by incinerator emissions since this was the exposure under consideration. The criteria for the selection of the control area were:

- Closely comparable with the study area for social class profile.
- Closely comparable with the study area for age profile.
- From a geographical area not within 3 kilometres of a hospital incinerator.
- From a geographical area served by Merseyside and Cheshire Cancer Registry.

In order to identify potential control areas, the socio-economic profile of the study area was first defined. This was done using 40 different types of neighbourhood or small area based on data from the Census of Population. Each census Enumeration District (ED) in the country (typically 150 households) is assigned to one of the 40 Super Profile Target Market area types.

The pattern of assignment of the ED population to Target Markets in the study area then served as the profile used to identify wards, elsewhere in Merseyside, matching this as closely as possible. Once these best matching wards had been identified, the search was refined by eliminating EDs within the ward that were not in the target profile.

Merseyside was searched to identify a suitable control area located as far away from an incinerator as possible. In order to meet the criteria of having a similar socio-economic profile as the study area and being of a suitable size to enable comparisons, the maximum distance from an incinerator that a control area could be identified was 3km. Within these constraints, it was not possible to identify a contiguous control area, therefore the area selected is a collection of small areas spread across Merseyside.

The final control area selected includes EDs in the following eight wards:

- Ford
- Tranmere
- St Marys
- St Oswalds
- St Gabriel
- Halewood West
- Halewood South
- Part of the Gillmoss ward not included in the study area

Case selection was as previously reported, ie all cases of primary cancer occurring in Merseyside and Cheshire residents between 1974 and 1998 inclusive. Cases were assigned to the control area on the basis of postcode within the selected EDs. Cancers were selected for inclusion in this study based upon the results of the literature search as previously reported. Data items were as described previously.

Population data for the control population were only available for 5 year age bands from the 1991 census (see **Statistical Appendix** for details). The population estimates from the 1991 census for the selected EDs within the control area were aggregated to produce a population estimate for the control area. These analyses therefore focus on the five year period 1989-93, for which the 1991 census year was the mid-point. The 1991 census data have also been used in further analyses to estimate the population for the 10 year period 1989-98.

Three types of incidence rates have been calculated, as described in 8.2: Crude rates, cumulative rates and age standardised rates.

RESULTS

The analyses compare cancer incidence rates in the study and control areas.

The control area has a population of around 64,000 (1991) and has a very similar distribution by age, sex and social class profile to the study area (**Statistical Appendix**). About 40% of the control area population are in the wards furthest away from a hospital incinerator.

As previously reported, 10,590 cancers were diagnosed between 1974-98 in individuals resident in the study area, of which 5,406 (51%) occurred in males and 5,184 (49%) in females. In the control area, a total of 9,698 cancers were initially identified by MCCR. In a final check of 600 missing or unknown postcodes, none was found to be in the control area. Of the 9,698 cases in the control area 5,127 (53%) occurred in males and 4,571 (47%) occurred in females.

Tables 8.7a and 8.7b show the number of cases by site and 5 year period for males and females separately for the study area and control area respectively. For liver cancer cases, and for cases of Hodgkins lymphoma, analyses are shown throughout over the 10 year period 1989-98 for males and females combined, due to the small number of cases in the control area. For the following sites, the number of cases within the control area even across the 10 year period, was very small and results should therefore be interpreted with caution:

- nasopharynx
- soft and connective tissue
- female laryngeal cancers

Table 8.7a: Study area - number of cancers by selected sites and quinquennia (all ages)

MALES

SITE	1974-1978	1979-1983	1984-1988	1989-1993	1994-1998	TOTAL
All malignant neoplasms	**1083**	**1042**	**1050**	**1075**	**1156**	**5406**
Trachea, bronchus and lung	399	340	312	279	269	1599
Larynx	20	16	17	16	21	90
Nasopharynx	0	2	1	0	3	6
Liver	4	7	6	11	12	40
Leukaemias	16	16	25	15	23	95
Non-Hodgkins lymphoma	16	10	19	20	26	91
Hodgkins lymphoma	4	7	4	8	3	26
Soft and connective tissue	5	3	7	7	6	28
Colon	53	61	58	75	67	314
Rectum	63	50	53	41	63	270

FEMALES

SITE	1974-1978	1979-1983	1984-1988	1989-1993	1994-1998	TOTAL
All malignant neoplasms	**953**	**991**	**1048**	**1057**	**1135**	**5184**
Trachea, bronchus and lung	117	114	152	185	202	770
Larynx	7	2	6	9	6	30
Nasopharynx	0	1	0	1	0	2
Liver	7	0	4	8	11	30
Leukaemias	17	12	9	14	23	75
Non-Hodgkins lymphoma	10	9	21	26	21	87
Hodgkins lymphoma	5	5	4	2	2	18
Soft and connective tissue	5	3	5	4	6	23
Colon	68	84	84	54	65	355
Rectum	54	39	51	44	39	227

Table 8.7b: Control area - number of cases by site and quinquennia (all ages)

MALES

SITE	1974-1978	1979-1983	1984-1988	1989-1993	1994-1998	TOTAL
All malignant neoplasms	**902**	**975**	**1010**	**1055**	**1185**	**5127**
Trachea, bronchus and lung	320	324	308	297	283	1532
Larynx	13	17	17	18	20	85
Nasopharynx	3	0	0	0	1	4
Liver	12	1	3	8	6	30
Leukaemias	21	20	20	25	16	102
Non-hodgkins lymphoma	17	14	23	21	26	101
Hodgkins lymphoma	3	6	7	3	5	24
Soft and connective tissue	2	3	2	3		10
Colon	43	85	59	54	61	302
Rectum	49	40	43	68	56	256

FEMALES

SITE	1974-1978	1979-1983	1984-1988	1989-1993	1994-1998	TOTAL
All malignant neoplasms	**825**	**862**	**902**	**949**	**1033**	**4571**
Trachea, bronchus and lung	93	135	141	126	199	694
Larynx	9	3	3	3	4	22
Nasopharynx	2	0	1	0	1	4
Liver	1	7	2	5	10	25
Leukaemias	19	15	17	15	10	76
Non-hodgkins lymphoma	6	11	18	19	17	71
Hodgkins lymphoma	9	3		3	3	18
Soft and connective tissue	5	2	6	2	3	18
Colon	59	70	52	66	55	302
Rectum	34	30	36	36	23	159

Tables 8.8a and 8.8b show the total number of cases of childhood cancer (aged 0-14 years) within the study and control areas respectively. Again, the numbers of cases by site for childhood cancers are too small to enable meaningful analyses for cancer subgroups. Thus, as in the main report, in the following analyses, all childhood cancer sites have been combined, and were looked at for the 10 year period 1989-98 for males and females combined.

The study and control areas had similar proportions of cancers by site (1974-98) for males and females (all ages - data not shown). The study area had similar proportions to LHA and both also had a higher proportion of lung cancers compared with Merseyside and Cheshire as a whole for males and females.

Table 8.8a: Study area - number of cancers by selected sites and quinquennia Children (aged under 15)

MALES

SITE	1974-1978	1979-1983	1984-1988	1989-1993	1994-1998	TOTAL
All malignant neoplasms	**3**	**3**	**5**	**7**	**9**	**27**
Leukaemias	1	1	3	2	5	12
Non-Hodgkins lymphoma	0	0	1	1	0	2
Hodgkins lymphoma	0	1	1	0	2	4

FEMALES

SITE	1974-1978	1979-1983	1984-1988	1989-1993	1994-1998	TOTAL
All malignant neoplasms	**5**	**2**	**3**	**3**	**5**	**18**
Leukaemias	2	1	0	0	1	4
Non-Hodgkins lymphoma	0	0	0	1	1	2
Hodgkins lymphoma	0	0	0	0	0	0

Table 8.8b: Control area - number of cancers by site and quinquennia Children (aged under 15)

MALES

SITE	1974-1978	1979-1983	1984-1988	1989-1993	1994-1998	TOTAL
All malignant neoplasms	**9**	**2**		**13**	**3**	**27**
Leukaemias	4			1		5
Non-Hodgkins lymphoma	2					2
Hodgkins lymphoma				2		2

FEMALES

SITE	1974-1978	1979-1983	1984-1988	1989-1993	1994-1998	TOTAL
All malignant neoplasms	**6**	**5**	**8**	**2**	**5**	**26**
Leukaemias	5					5
Non-Hodgkins lymphoma					1	1
Hodgkins lymphoma	0	0	0	0	0	0

Figure 8.16 shows that the number of cases within the control area over time has fluctuated between around 300 - 500 cases per year, and that the overall pattern is similar to the study area. This compares with an average over the same period of 400 in the study area reported previously.

Figure 8.16: Number of cancers within the control area by year of diagnosis and gender 1974-1998

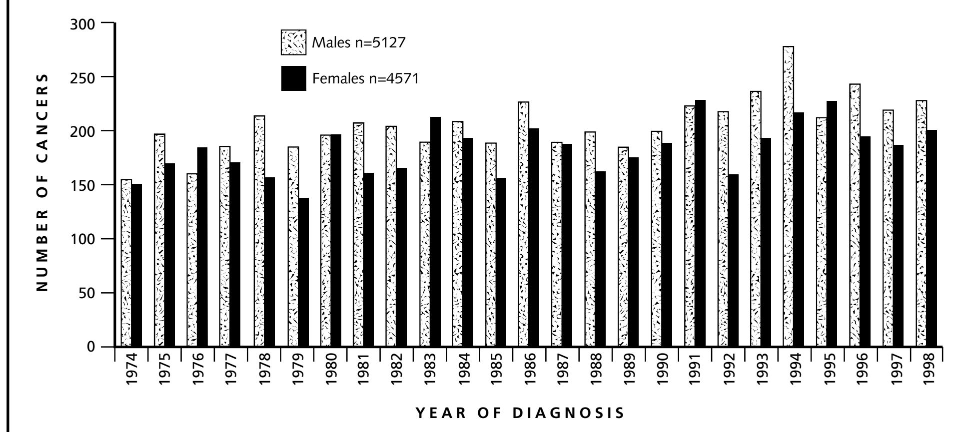


Figure 8.17 shows that the overall cancer rate in males and females in the study area is significantly lower than the rate in the control area.

Figure 8.17: Cumulative rate for all adult cancers (aged 15 to 74) by area and gender for 1989-1993

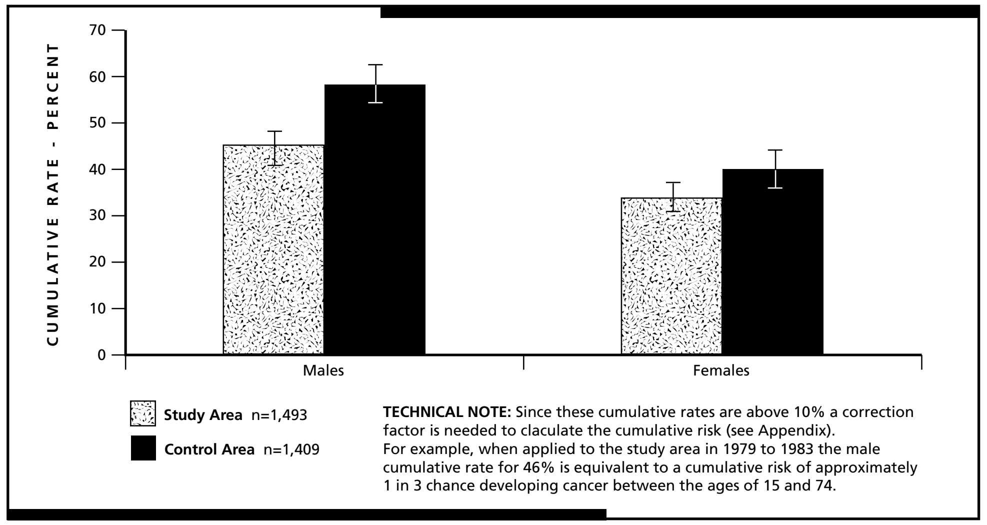
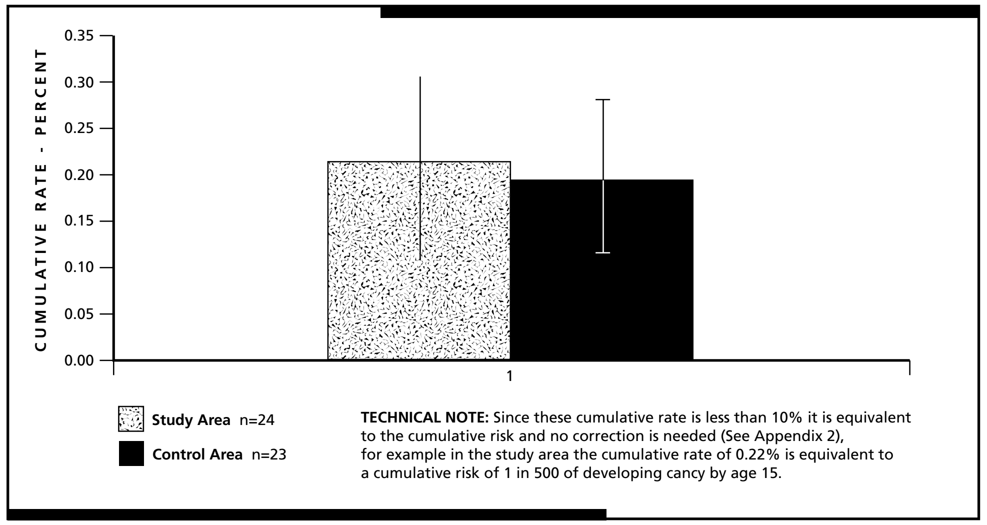


Figure 8.18 shows that there are no significant differences between the study and control areas for childhood cancers when taken altogether.

Figure 8.18: Cumulative rate for all childhood cancers (aged under 15) by area for 1989-1993



Figures 8.19 and 8.20 show that in 1989-93, for leukaemias, non-Hodgkins lymphoma and colon cancers (both males and females), for rectal and lung cancers (females only), and for laryngeal (males only) there were no significant differences between the study and control areas. Exceptions to this general pattern were found in lung and rectal cancers in men, for which study area rates were significantly lower than control area rates.

Figure 8.19: Cumulative rate for male cancers, aged 0-74, by common selected sites and area 1989-1993

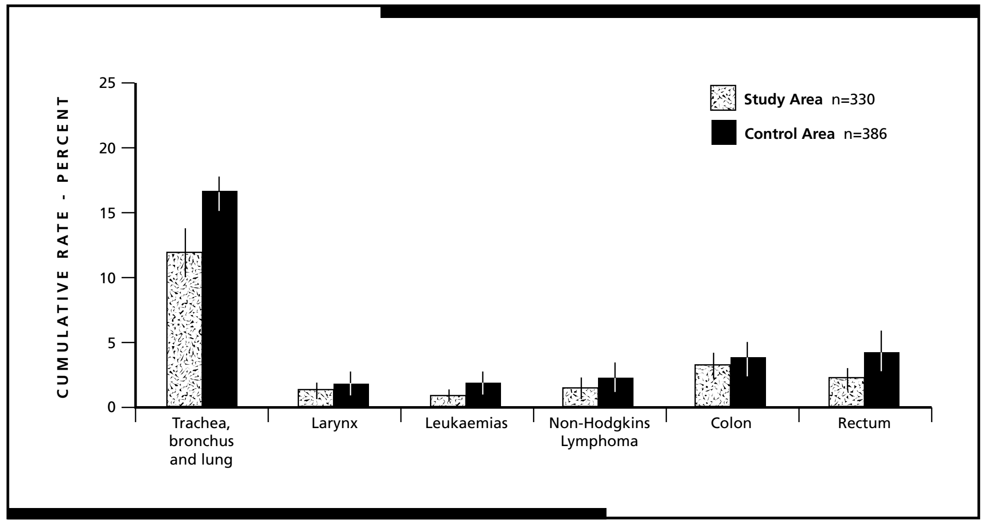


Figure 8.20: Cumulative rate for female cancers, aged 0-74, by common selected sites and area 1989-1993

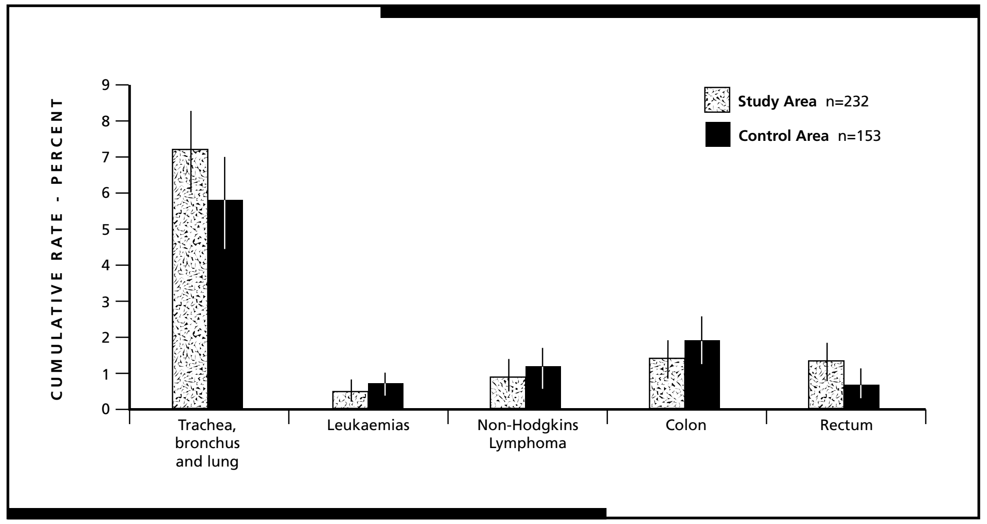
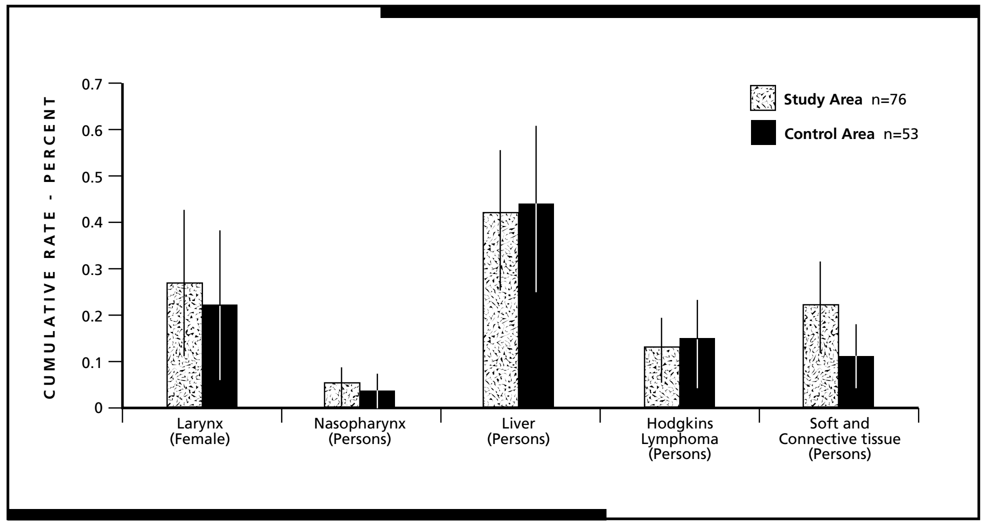


Figure 8.21 shows how rates vary between areas for the rare cancers included in the study. There are no significant differences between the study and control areas in 1989-98 for rates of Hodgkins lymphoma, liver cancer, nasopharyngeal cancer, and soft tissue sarcomas for males and females combined, nor for laryngeal cancers in females. Interpretation of these figures should be cautious in view of the small numbers of cases and the uncertainty indicated by the wide error bars.

Figure 8.21: Cumulative rate for selected rare cancers in persons aged 0-74, for control and study areas 1989-1993



Tables 8.9a and 8.9b show detailed data on the number of cases, cumulative rates and age standardised rates for the 10 year period between 1989-98 for the rare sites (0-74 years), selected for study, and for childhood cancers (0-14 years) respectively.

Table 8.9a: 1989-1998 cancers by selected rare sites and area

	STUDY AREA*				CONTROL AREA*			
	95% CI				95% CI			
	n	RATE	LOWER	UPPER	n	RATE	LOWER	UPPER
Female Larynx cancers	15				7			
Crude rate		3.72	1.84	5.60		2.12	0.55	3.69
Cumulative rate (aged 0-74)		0.27	0.11	0.43		0.22	0.06	0.38
Age Standardised Rate		3.07	1.41	4.72		2.40	0.58	4.22
Nasopharynx	4	2						
Crude rate		0.51	0.01	1.02		0.31	-0.12	0.75
Cumulative rate (aged 0-74)		0.05	0.00	0.09		0.03	-0.01	0.08
Age Standardised Rate		0.56	0.00	1.11		0.42	-0.17	1.01
Liver	42	29						
Crude rate		5.41	3.77	7.04		4.53	2.88	6.18
Cumulative rate (aged 0-74)		0.41	0.26	0.56		0.43	0.25	0.60
Age Standardised Rate		4.80	3.29	6.31		4.30	2.70	5.90
Hodgkins lymphoma	15	14						
Crude rate		1.93	0.95	2.91		2.19	1.04	3.33
Cumulative rate (aged 0-74)		0.12	0.05	0.19		0.15	0.06	0.23
Age Standardised Rate		1.84	0.89	2.78		1.98	0.92	3.04
Soft and connective tissue	23	8						
Crude rate		2.96	1.75	4.17		1.25	0.38	2.12
Cumulative rate (aged 0-74)		0.21	0.11	0.31		0.10	0.03	0.18
Age Standardised Rate		2.86	1.66	4.07		1.31	0.38	2.24

Table 8.9b: Childhood cancers (aged 0-14 years), 1989-1998 by area

	95% CI			
	n	RATE	LOWER	UPPER
Study area*	24			
Crude rate		14.22	-0.94	19.91
Cumulative rate (aged 0-14)		0.22	0.13	0.30
Age Standardised Rate		3.14	1.88	4.40
Control area*	23			
Crude rate		13.63	7.46	19.20
Cumulative rate (aged 0-14)		0.20	0.12	0.28
Age Standardised Rate		3.01	1.78	4.24

* To calculate rates for the study and control area population data from the 1991 census have been used as the population estimates for 1989 to 1998

Table 8.10 shows detailed data on the number of cases, cumulative rates and age standardised rates for the two 5 year periods between 1989-98 for adult (15-74 years) cancers overall. Similar detailed data on the common sites selected for study are shown in Tables 8.11 to 8.16 (0-74 years).

Table 8.10: All adult cancers (aged 15 and over) by area and quinquennia

MALES

	1989-1993				1994-1998			
	95% CI				95% CI			
	n	RATE	LOWER	UPPER	n	RATE	LOWER	UPPER
Study area*	1068				1147			
Crude rate		742.3	697.8	786.8		797.2	751.1	843.3
Cumulative rate (aged 0-74)		45.5	42.2	48.8		51.1	47.5	54.7
Age Standardised Rate		583.0	546.8	619.1		632.6	594.6	670.6
Control area*	1042				1182			
Crude rate		892.0	837.9	946.2		1011.9	954.2	1069.6
Cumulative rate (aged 0-74)		58.2	54.0	62.5		65.5	60.9	70.1
Age Standardised Rate		726.6	680.9	772.3		825.3	776.6	874.1

FEMALES

	1989-1993				1994-1998			
	95% CI				95% CI			
	n	RATE	LOWER	UPPER	n	RATE	LOWER	UPPER
Study area*	1054				1130			
Crude rate		658.1	618.4	697.8		705.6	664.4	746.7
Cumulative rate (aged 0-74)		34.2	31.7	36.8		36.9	34.2	39.6
Age Standardised Rate		427.6	400.2	454.9		449.9	422.2	477.7
Control area*	947				1028			
Crude rate		727.9	681.6	774.3		790.2	741.9	838.5
Cumulative rate (aged 0-74)		40.1	36.9	43.2		47.0	43.5	50.4
Age Standardised Rate		505.2	471.4	539.0		562.0	526.1	597.9

* To calculate rates for the study and control area population data from the 1991 census have been used as the population estimates for 1989-1998

Table 8.11: Trachea, Bronchus and Lung cancers, all ages by area and quinquennia

MALES

	1989-1993				1994-1998			
		95% CI				95% CI		
	n	RATE	LOWER	UPPER	n	RATE	LOWER	UPPER
Study area*	279				269			
Crude rate		149.5	132.0	167.1		144.1	126.9	161.4
Cumulative rate (aged 0-74)		11.9	10.2	13.5		12.1	10.3	13.8
Age Standardised Rate		149.4	131.3	167.5		148.0	129.6	166.4
Control area*	297				283			
Crude rate		191.5	169.7	213.2		182.4	161.2	203.7
Cumulative rate (aged 0-74)		17.2	14.8	19.5		17.1	14.7	19.5
Age Standardised Rate		200.0	176.6	223.3		198.0	174.0	221.9

FEMALES

	1989-1993				1994-1998			
		95% CI				95% CI		
	n	RATE	LOWER	UPPER	n	RATE	LOWER	UPPER
Study area*	185				202			
Crude rate		91.7	78.5	104.9		100.1	86.3	113.9
Cumulative rate (aged 0-74)		7.1	6.0	8.3		7.4	6.1	8.6
Age Standardised Rate		76.1	64.6	87.6		77.2	66.0	88.3
Control area*	126				199			
Crude rate		76.4	63.0	89.7		120.6	103.8	137.4
Cumulative rate (aged 0-74)		5.8	4.5	7.0		10.2	8.6	11.8
Age Standardised Rate		64.6	52.9	76.4		105.2	90.1	120.3

* To calculate rates for the study and control area population data from the 1991 census have been used as the population estimates for 1989-1998

Table 8.12: Cancer of the Larynx, all ages, by area and quinnquennia

MALES

	1989-1993				1994-1998			
		95% CI				95% CI		
	n	RATE	LOWER	UPPER	n	RATE	LOWER	UPPER
Study area*	16				21			
Crude rate		8.6	4.4	12.8		11.3	6.4	16.1
Cumulative rate (aged 0-74)		0.8	0.4	1.3		1.1	0.6	1.6
Age Standardised Rate		8.5	4.2	12.7		10.5	6.0	15.0
Control area*	18				20			
Crude rate		11.6	6.2	17.0		12.9	7.2	18.5
Cumulative rate (aged 0-74)		1.0	0.5	1.6		1.4	0.8	2.1
Age Standardised Rate		13.5	6.9	20.1		14.1	7.9	20.4

FEMALES

	1989-1993				1994-1998			
		95% CI				95% CI		
	n	RATE	LOWER	UPPER	n	RATE	LOWER	UPPER
Study area*	9				6			
Crude rate		4.5	1.5	7.4		3.0	0.6	5.4
Cumulative rate (aged 0-74)		0.3	0.1	0.6		0.2	0.0	0.4
Age Standardised Rate		3.8	1.2	6.4		2.4	0.3	4.4
Control area*	3				4			
Crude rate		1.8	-0.2	3.9		2.4	0.0	4.8
Cumulative rate (aged 0-74)		0.2	0.0	0.4		0.2	0.0	0.5
Age Standardised Rate		1.7	-0.2	3.7		3.1	0.0	6.1

* To calculate rates for the study and control area population data from the 1991 census have been used as the population estimates for 1989-1998

Table 8.13: Leukaemias, all ages, by area and quinnquennia

MALES

	1989-1993				1994-1998			
		95% CI				95% CI		
	n	RATE	LOWER	UPPER	n	RATE	LOWER	UPPER
Study area*	15				23			
Crude rate		8.0	4.0	12.1		12.3	7.3	17.4
Cumulative rate (aged 0-74)		0.6	0.2	1.0		1.2	0.6	1.7
Age Standardised Rate		8.3	4.0	12.5		12.2	7.0	17.3
Control area*	25				16			
Crude rate		16.1	9.8	22.4		10.3	5.3	15.4
Cumulative rate (aged 0-74)		1.4	0.7	2.1		0.9	0.3	1.4
Age Standardised Rate		18.1	10.7	25.6		11.4	5.6	17.2

FEMALES

	1989-1993				1994-1998			
		95% CI				95% CI		
	n	RATE	LOWER	UPPER	n	RATE	LOWER	UPPER
Study area*	14				23			
Crude rate		6.9	3.3	10.6		11.4	6.7	16.1
Cumulative rate (aged 0-74)		0.5	0.2	0.8		0.6	0.2	0.9
Age Standardised Rate		5.6	2.5	8.7		8.0	4.6	11.5
Control area*	15				10			
Crude rate		9.1	4.5	13.7		6.1	2.3	9.8
Cumulative rate (aged 0-74)		0.7	0.2	1.1		0.4	0.0	0.7
Age Standardised Rate		8.1	3.8	12.4		5.1	1.9	8.4

* To calculate rates for the study and control area population data from the 1991 census have been used as the population estimates for 1989-1998

Table 8.14: Non-Hodgkins lymphoma, all ages, by area and quinnquennia

MALES

	1989-1993				1994-1998			
		95% CI				95% CI		
	n	RATE	LOWER	UPPER	n	RATE	LOWER	UPPER
Study area*	20				26			
Crude rate		10.7	6.0	15.4		13.9	8.6	19.3
Cumulative rate (aged 0-74)		0.9	0.5	1.4		1.1	0.6	1.7
Age Standardised Rate		10.4	5.8	15.0		14.1	8.6	19.6
Control area*	21				26			
Crude rate		13.5	7.7	19.3		16.8	10.3	23.2
Cumulative rate (aged 0-74)		1.4	0.8	2.1		1.7	1.0	2.4
Age Standardised Rate		13.8	7.8	19.8		18.6	11.2	26.0

FEMALES

	1989-1993				1994-1998			
		95% CI				95% CI		
	n	RATE	LOWER	UPPER	n	RATE	LOWER	UPPER
Study area*	26				21			
Crude rate		12.9	7.9	17.8		10.4	6.0	14.9
Cumulative rate (aged 0-74)		0.9	0.5	1.3		0.6	0.2	0.9
Age Standardised Rate		11.7	7.0	16.5		8.8	4.8	12.8
Control area*	19				17			
Crude rate		11.5	6.3	16.7		10.3	5.4	15.2
Cumulative rate (aged 0-74)		1.1	0.6	1.6		0.7	0.3	1.1
Age Standardised Rate		11.2	5.9	16.5		8.3	4.2	12.3

* To calculate rates for the study and control area population data from the 1991 census have been used as the population estimates for 1989-1998

Table 8.15: Colon Cancer, all ages, by area and quinnquennia

MALES

	1989-1993				1994-1998			
			95% CI				95% CI	
	n	RATE	LOWER	UPPER	n	RATE	LOWER	UPPER
Study area*	75				67			
Crude rate		40.2	31.1	49.3		35.9	27.3	44.5
Cumulative rate (aged 0-74)		3.0	2.2	3.9		2.9	2.0	3.7
Age Standardised Rate		39.6	30.4	48.9		37.3	27.9	46.6
Control area*	54				61			
Crude rate		34.8	25.5	44.1		39.3	29.5	49.2
Cumulative rate (aged 0-74)		3.8	2.6	4.9		3.4	2.3	4.5
Age Standardised Rate		36.8	26.8	46.8		43.4	32.1	54.8

FEMALES

	1989-1993				1994-1998			
			95% CI				95% CI	
	n	RATE	LOWER	UPPER	n	RATE	LOWER	UPPER
Study area*	54				65			
Crude rate		26.8	19.6	33.9		32.2	24.4	40.0
Cumulative rate (aged 0-74)		1.4	0.9	2.0		1.9	1.3	2.5
Age Standardised Rate		19.3	13.9	24.6		24.3	18.1	30.6
Control area*	66				55			
Crude rate		40.0	30.3	49.6		33.3	24.5	42.1
Cumulative rate (aged 0-74)		1.9	1.2	2.6		2.2	1.5	2.9
Age Standardised Rate		29.6	22.1	37.0		28.1	20.4	35.8

* To calculate rates for the study and control area population data from the 1991 census have been used as the population estimates for 1989-1998

Table 8.16: Cancer of the Rectum, all ages, by area and quinnquennia

MALES

	1989-1993				1994-1998			
			95% CI				95% CI	
	n	RATE	LOWER	UPPER	n	RATE	LOWER	UPPER
Study area*	41				63			
Crude rate		22.0	15.2	28.7		33.8	25.4	42.1
Cumulative rate (aged 0-74)		2.0	1.3	2.7		2.8	2.0	3.6
Age Standardised Rate		22.5	15.4	29.6		34.9	26.1	43.8
Control area*	68				56			
Crude rate		43.8	33.4	54.3		36.1	26.6	45.6
Cumulative rate (aged 0-74)		4.5	3.3	5.6		2.6	1.7	3.5
Age Standardised Rate		47.7	36.2	59.2		39.0	28.3	49.6

FEMALES

	1989-1993				1994-1998			
			95% CI				95% CI	
	n	RATE	LOWER	UPPER	n	RATE	LOWER	UPPER
Study area*	44				39			
Crude rate		21.8	15.4	28.2		19.3	13.3	25.4
Cumulative rate (aged 0-74)		1.3	0.8	1.8		1.1	0.6	1.6
Age Standardised Rate		17.1	11.7	22.5		15.0	9.9	20.1
Control area*	36				23			
Crude rate		21.8	14.7	28.9		13.9	8.2	19.6
Cumulative rate (aged 0-74)		0.7	0.3	1.1		1.1	0.6	1.6
Age Standardised Rate		15.3	10.1	20.5		11.3	6.6	16.0

* To calculate rates for the study and control area population data from the 1991 census have been used as the population estimates for 1989-1998

Table 8.17 shows that although the proportion of smokers amongst cases diagnosed with lung cancer is higher in the study area than in the control area for both males and females for 1989-93, these results are not significant.

Table 8.17: Smoking proportions for lung cancer in the four comparison areas for 1989-1998

	No. OF SMOKERS WITH LUNG CANCER		No. OF LUNG CANCER CASES		PROPORTION OF LUNG CANCER CASES THAT ARE SMOKERS					
						95% CI			95% CI	
	MALES	FEMALES	MALES	FEMALES	MALES	LOWER	UPPER	FEMALES	LOWER	UPPER
Study area	212	128	279	185	76%	70%	82%	69%	61%	77%
Control area	205	78	297	126	69%	63%	75%	62%	51%	73%

Males and Females combined

	No. OF SMOKERS WITH LUNG CANCER	No. OF LUNG CANCER CASES	PROPORTION OF LUNG CANCER CASES THAT ARE SMOKERS	95% CI	
				LOWER	UPPER
Study area	340	464	73%	69%	78%
Control area	283	423	67%	61%	72%

SUMMARY OF FINDINGS

- The study and control areas have similar age, sex and socio-economic group profiles, and population sizes, and are located within Merseyside.
- **The study and control areas have a similar distribution of cancers.**
- **There were no significant differences between the study and the control areas for childhood cancers when considered altogether.**
- Overall, adult cancer rates were significantly lower in the study area compared with the control area for males and females.
- For all selected cancers, rates amongst females in the study area were similar to those of the control area.
- **For lung and rectal cancers in males, rates in the study area were significantly lower than rates in the control area. For all other selected sites, rates in males were similar in study and control areas.**

DISCUSSION

In selecting the control area account was taken of three major risk factors for cancer ie age, sex and socio-economic group. The control area was designed to be as similar as possible to the study area for these known cancer risk factors, and as different as possible for the main factor under consideration ie exposure to incinerator emissions, measured here as distance from an incinerator.

Problems associated with estimating the study area population were minimised by limiting the main analyses to the 5 years around the 1991 census year for which the population is known. The problems with the population data meant however that comparisons incorporating the control group could not be made over time.

The study area had significantly fewer cancers overall compared with the control area, raising the possibility that the control area was more deprived than the study area. Such an effect is likely to be small however, since similarity between the two areas was demonstrated for age,

sex and socio-economic status. Socio-economic status was however based upon an area rather than an individual measure, and thus the population selected may have included individuals who did not share the socio-economic characteristics of the area in which they resided. The profile used to select the control area was derived from the study area, and thus the same difficulties would be expected to arise in both cases. It is therefore unlikely that such bias would fall differentially upon the control area. The control area did however appear to contain a marginally greater preponderance of more deprived EDs (**Statistical Appendix**) which could account for the higher overall cancer rates.

A slightly more deprived control area would be more likely to give rise to differences between the control and the study areas for cancers which had a strong link to socio-economic group eg, lung (males and females), rectal and laryngeal (males only). In females, for all selected cancers, there were no significant differences between the study and control areas. For males, where significant differences were found for lung and rectal cancer, the excess was in the control area, consistent with the predicted effect of small differences in deprivation between the control and study areas. The numbers of rectal cancers within each 5 year period however fluctuated markedly and the control area appeared to have an unusually high number of cases in 1989-93. This suggests artefact as a possible alternative cause for this finding. Furthermore, with the exception of rectal cancers in males, the distribution of cancers of the colon and rectum, chosen for their lack of known association with incinerator emissions, was remarkably similar between control and study areas. Lung cancer is considered in more detail below.

For cancers having weaker links with deprivation, the expected finding would be no difference between the control and study areas. If other, for example environmental factors such as incinerator emissions were present and having an effect, this would tend to push study area rates higher than control area rates for these cancers. Small differences in deprivation between the control and study areas would be unlikely to have much effect on findings associated with these cancers. Most of the findings showed no difference between the control and the study areas and therefore provide no evidence for an effect of incinerator emissions. For several sites however, numbers were too small to enable full analysis and interpretation of the results.

DISCUSSION OF LUNG CANCER FINDINGS

Lung cancer merits more detailed discussion because cases are present in sufficient numbers to allow full analysis, and it is a cancer for which environmental, lifestyle and deprivation factors have all been causally implicated. The pattern of lung cancer in the study and the control areas varied for males and females, with rates in males relatively stable over the whole study period and significantly lower in the study area. In contrast, rates of lung cancer in females varied significantly over the same period in both study and control areas, with an almost doubling of the rates. The significant rise in study area lung cancer rates in females between 1979-83 compared with 1989-93, preceded the rise in the control area which occurred in 1994-98. Although initially lower than rates in the study area, by 1994-98, control area rates for females with lung cancer were higher than rates in the study area, although this just failed to achieve statistical significance. This change arose principally because of the significant rise in control area rates, with study area rates remaining relatively stable after 1993.

Lung cancer rates in males in the study area were higher than rates in Merseyside and Cheshire, similar to rates in LHA, and lower than rates in the control area, a pattern that was present throughout the time period studied. Higher control area lung cancer rates in males are not consistent with an environmental cause affecting the study area, suggesting the importance of other factors such as smoking and to a lesser extent deprivation. Lung cancer rates in females varied significantly over the study period. In 1989-93, the pattern was similar to that for males ie control area rates were not significantly different from study area rates and LHA rates, but all were significantly higher than rates for Merseyside and Cheshire overall. By 1994-98, control area rates for lung cancer in females had increased significantly, were significantly higher than rates in both LHA and Merseyside and Cheshire, and were higher than rates in the study area, although this just failed to reach statistical significance.

Higher rates of lung cancer in control area males are likely to reflect historical smoking patterns, since smoking is the major risk factor for this disease. Smoking data presented here for diagnosed cases of lung cancer however do not appear to explain higher rates in the control area, since the overall proportion of smokers is similar to that in the study area.

Differences in historical patterns of smoking between the control area and study area are likely to be the explanation, since current patterns of lung cancer reflect previous smoking patterns. Data availability however did not allow this to be confirmed with an analysis over time.

CONCLUSION

There is no evidence that rates of cancer in the study area are unusually high compared with LHA. The study area is similar to LHA in having significantly higher rates of some cancers when compared with Merseyside and Cheshire as a whole. No significant excess of cancers was found in the study area compared with the control area, despite study area residents being generally nearer to an incinerator. **There is no evidence from these analyses that cancer rates in the study area are unusually high compared with a control area having a similar socio-economic, age and sex profile. Deprivation and lifestyle factors such as smoking are more likely to explain these findings than environmental factors such as incinerator emissions**.

SPECIALIST GEOGRAPHICAL ANALYSIS

9.1. Data Used

Data on cancer cases and control population were provided by Dr Kate Ardern of Liverpool Health Authority and the Merseyside and Cheshire Cancer Registry. Ethical approval was obtained from the Chair of the Local Research and Ethics Committee for this aspect of the study.

This consisted of all known cases diagnosed between 1974 and 1998 within the area covered by post-code zones L9, L10 and L11.

Age and sex matched control locations were derived by random sampling from a data-base of GP registrations within the Liverpool Health Authority. This database listed the numbers of females and males in five-year age-bands resident at each unit post-code within the study area. The Townsend index (Townsend, Phillimore and Beattie 1988)[1] was obtained for each census enumeration district (ED) within the study area and used as an individual-level adjustment for social deprivation.

Residual risk of cancer after adjustment for age, sex and deprivation was initially modelled parametrically as a non-linear function of distance from the incinerator.[2,3] As an aid to interpretation of any possible variations in residual risk not attributable to proximity to the incinerator, a non-parametric generalised additive modelling approach (Kelsall and Diggle 1998)[4] was used as a secondary analysis for the three most common types of cancer (adult colorectal, lung and leukaemias and lymphomas).

CANCER CASES

Individuals aged 16 years or more at time of diagnosis were classified as adult cases and those people who were aged 15 years or less as childhood cases. A small number of cases (95 out of 10085) could not be included in the analysis because of a non-traceable postcode or a missing value for the Townsend index. Table 9.1 shows this information by cancer type.

Table 9.1: Numbers of Cases between 1974 and 1998 available for analysis

CANCER TYPE	NO. RECEIVED	NON TRACEBLE POSTCODE (CHILD AND ADULT CASES)	MISSING TOWNSEND VALUE (CHILD AND ADULT CASES)	NO. USED IN THE ANALYSIS
Colorectal	1166	0	4	1162
Lung	2369	9	15	2345
Liver	70	0	0	70
Laryngeal and nasopharynx	128	0	1	127
Leukaemia and lymphoma	392	0	2	390
Soft tissue sarcoma	51	0	3	48
Other cancer types	5909	8	53	5848
Total	10085	17	78	9990

Table 9.2 shows the numbers of cases available for analysis, further sub-divided into adult and childhood cases.

Table 9.2: Number of cases used by cancer type and age- group between 1974 and 1998

CANCER TYPE	CASES USED
Colorectal (adult)	1162
Lung (adult)	2345
Liver (adult)	70
Leukaemia and lymphoma (adult)	365
Larynx and nasopharynx (adult)	126
Soft tissue sarcoma (adult)	45
Other adult cancers	5828
All selected adult cases	2951
All known adult cases	9941
Leukaemia and lymphoma (child)	25
Larynx and nasopharynx (child)	1
Soft tissue sarcoma (child)	3
Other childhood cancers	20
All selected child cases	29
All known child cases	49

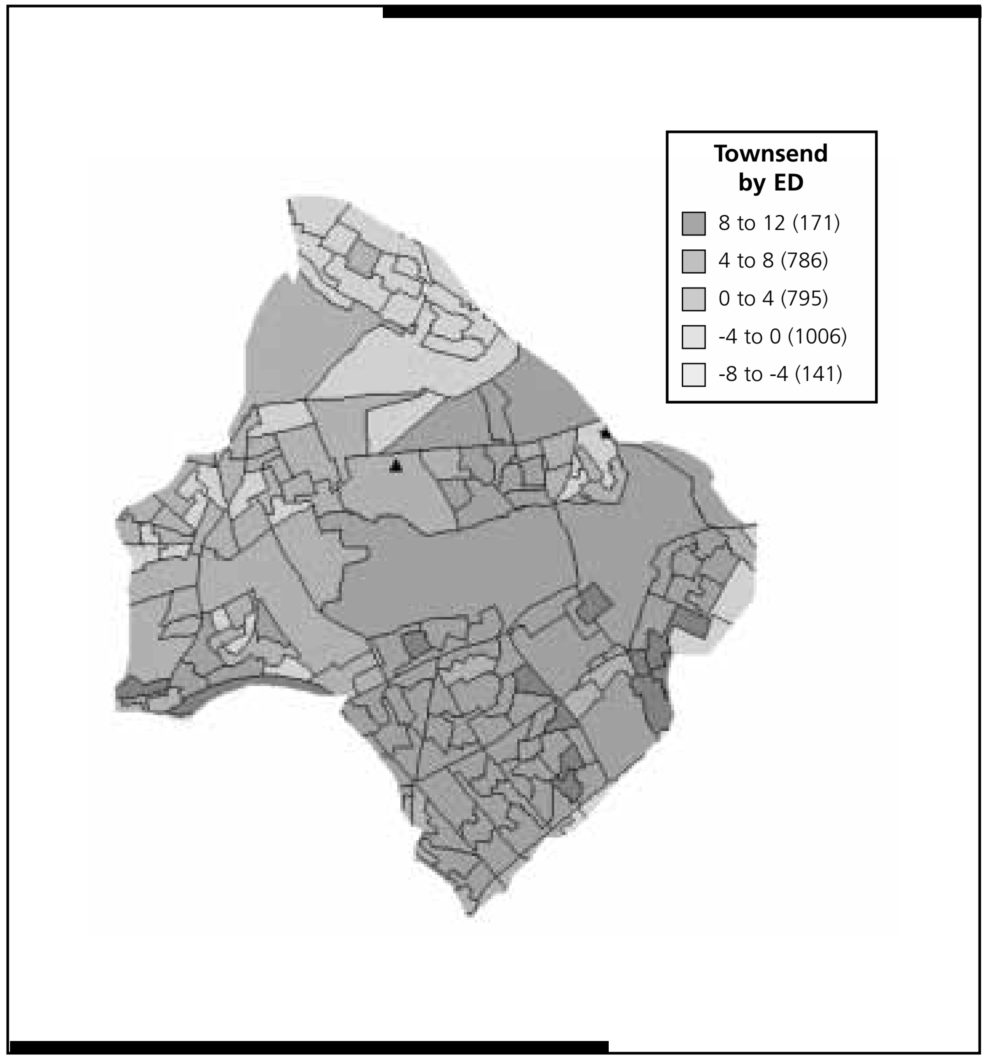
CONTROLS

Separate random samples of controls for adult and childhood cancers were selected using the database of GP registrations. Sex and age were recorded for each control (by random imputation within the relevant age interval) and the Townsend index for the enumeration district in which the individual's post-code was located. There were 10,000 controls for adults and 200 controls for children.

MAPS

Figure 9.1 is a map of the area surrounding the site of the former incinerator showing its former location, the enumeration district and ward boundaries. The study area (the postcode sectors L9,L10 and L11) corresponds very roughly to a diamond-shaped area extending approximately 6 kilometres from east to west and 7 kilometres from north to south, with the site of the former incinerator approximately 1 kilometre north of the centre of the diamond. Values of the Townsend index are also shown on Figure 1. Note that the Townsend index is positive for the majority of enumeration districts indicating that the study area as a whole is relatively deprived. Figure 9.1 also shows the grid reference (34000,39750) which, as is discussed in 9.4, lies with an area of significantly raised risk for adult colorectal and lung cancers after adjustment has been made for age, sex and social deprivation.

Figure 1: The area surrounding the Fazakerley hospital incinerator, showing ED boundaries, the location of the incinerator (triangle), the point at which the estimate of residual risk is maximised (star) and values of the Townsend index in each ED.



Figures 9.2 to 9.10 show the locations of cases for the six types of adult cancer, adult controls, childhood leukaemias/lymphomas and childhood controls, with the site of the now demolished incinerator shown as a black triangle. Qualitatively, these maps all show a similar distribution as would be expected, since they broadly reflect the spatial distribution of the population at risk. Note also that for the more common cancers (lung, colorectal) there is a high degree of multiplicity in the plotted locations.

Figure 9.2: Locations of Colorectal Cancers

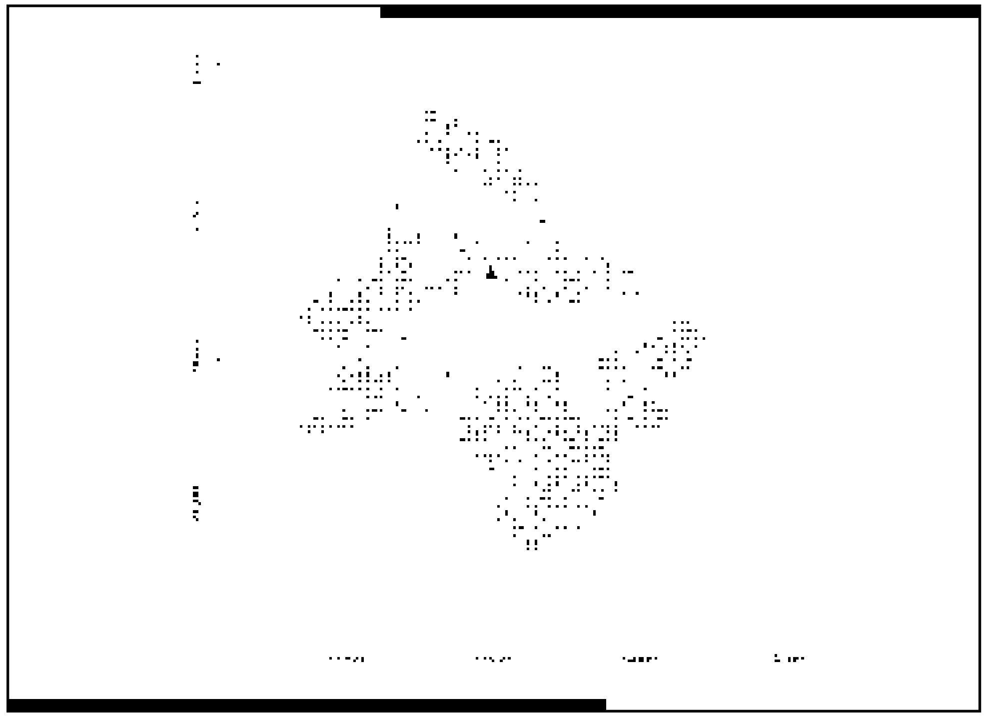


Figure 9.3: Locations of Lung Cancers

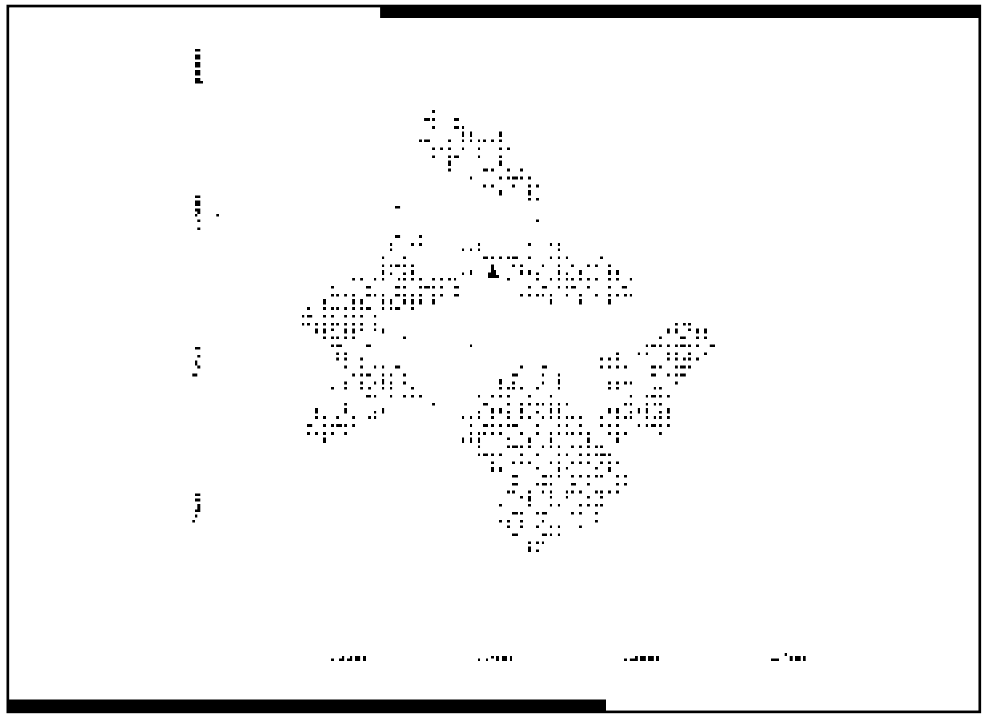


Figure 9.4: Locations of Liver Cancers

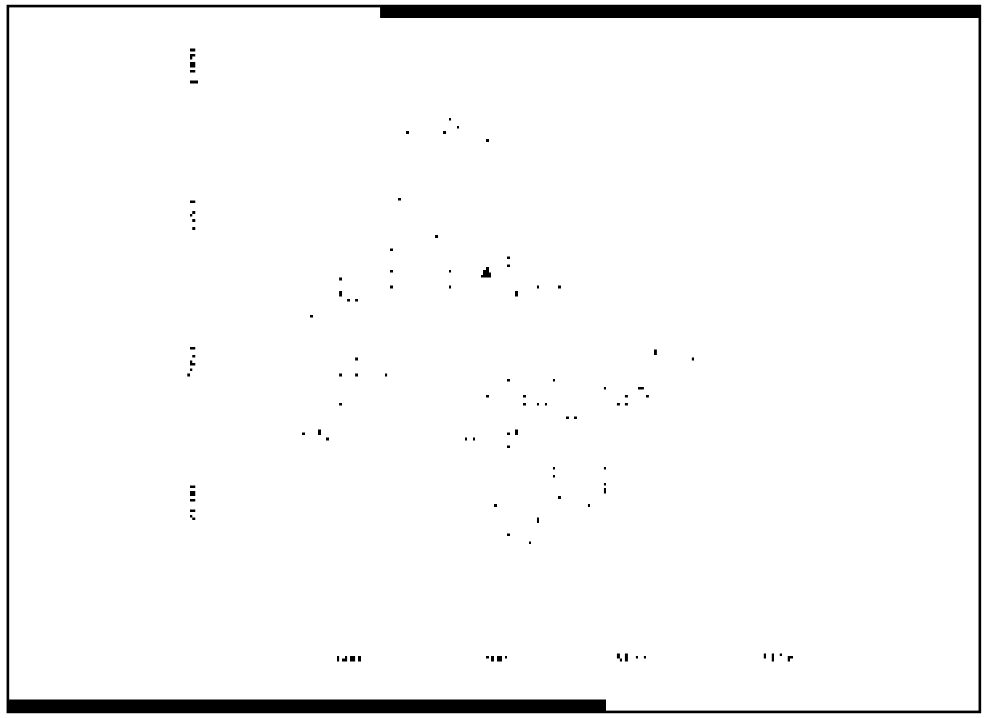


Figure 9.5: Locations of Larynx/Nasopharynx Cancers

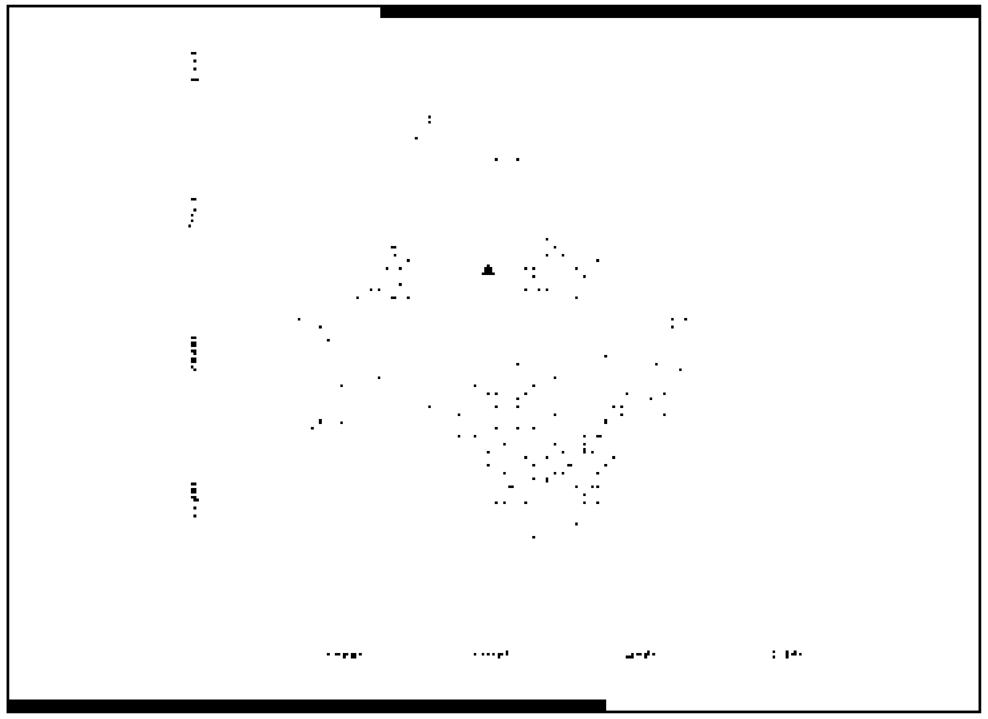


Figure 9.6: Locations of Adult Leukaemias/Lymphomas

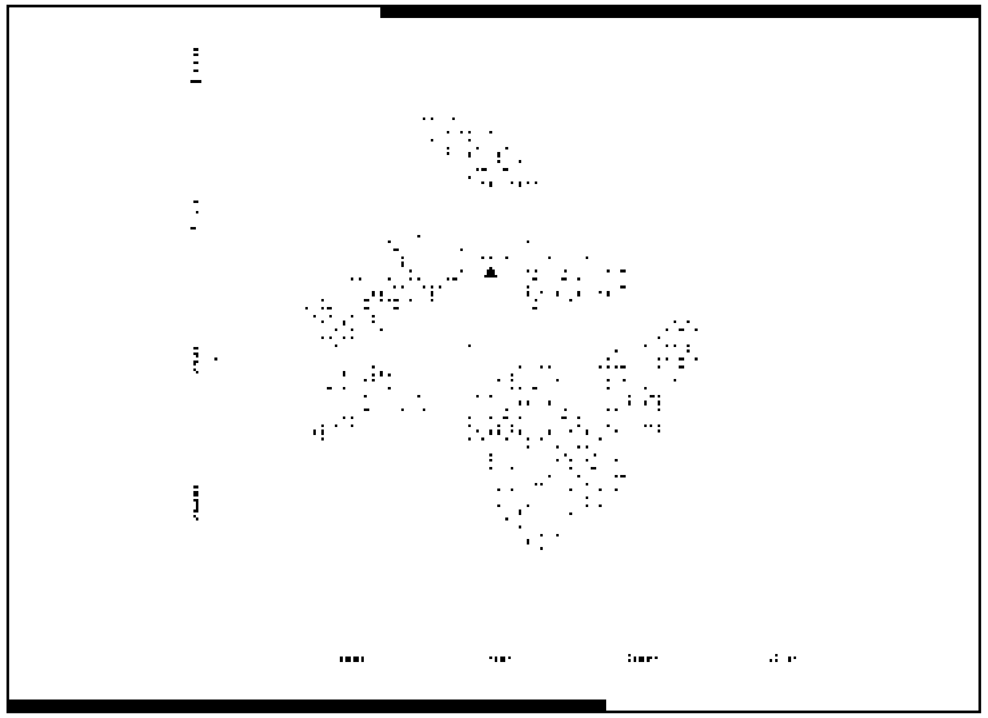


Figure 9.7: Locations of Soft Tissue Sarcomas

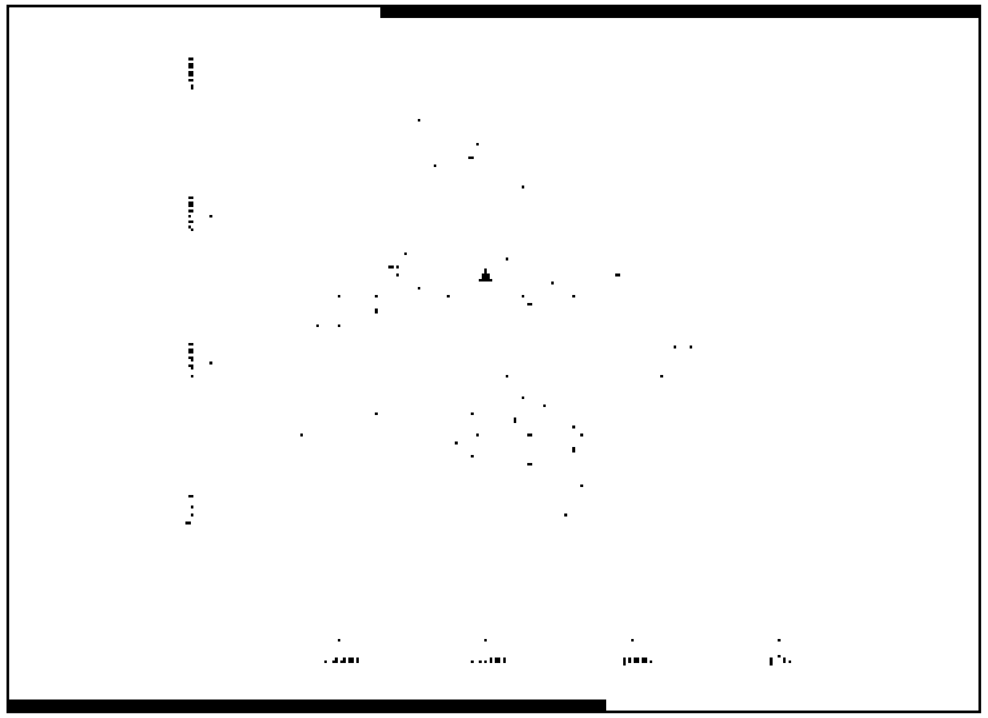


Figure 9.8: Locations of Adult Controls

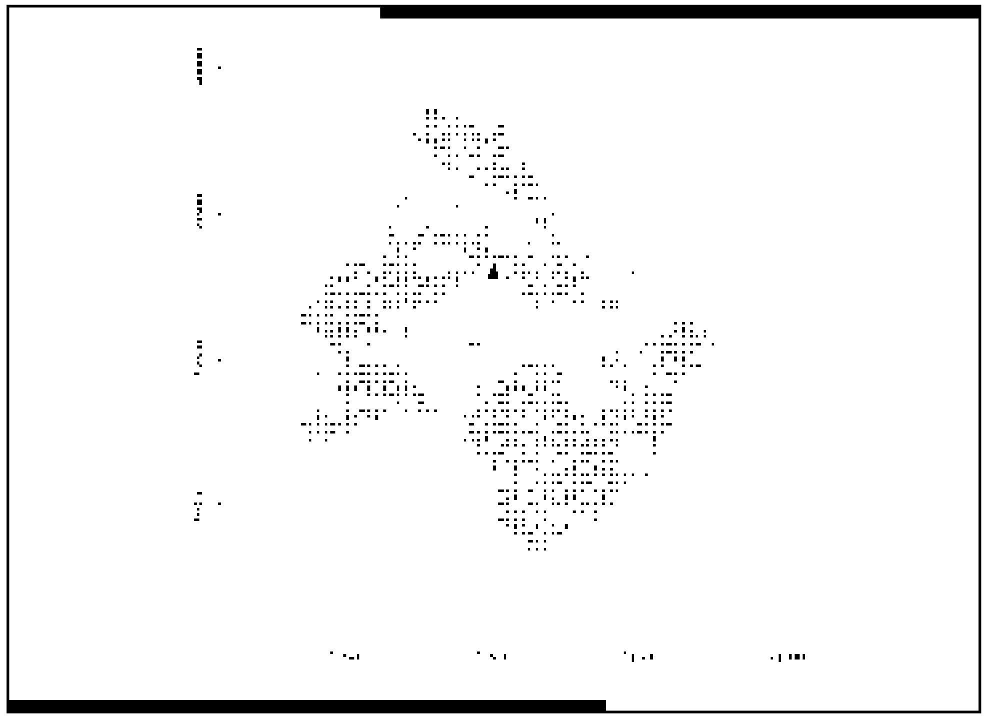


Figure 9.9: Locations of Childhood Leukaemias/Lymphomas

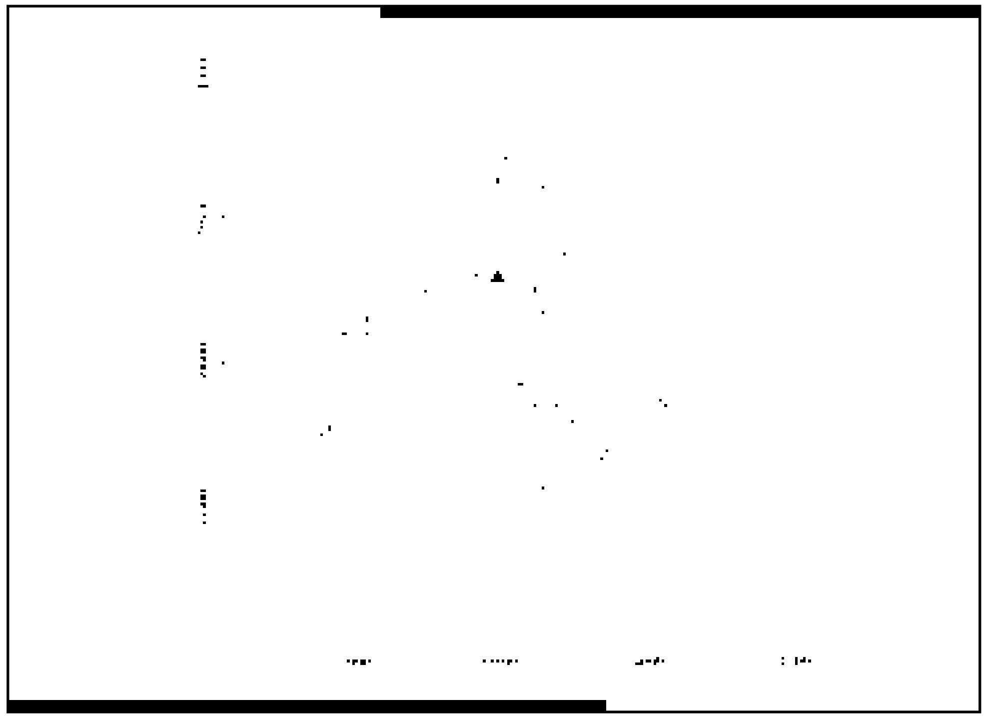
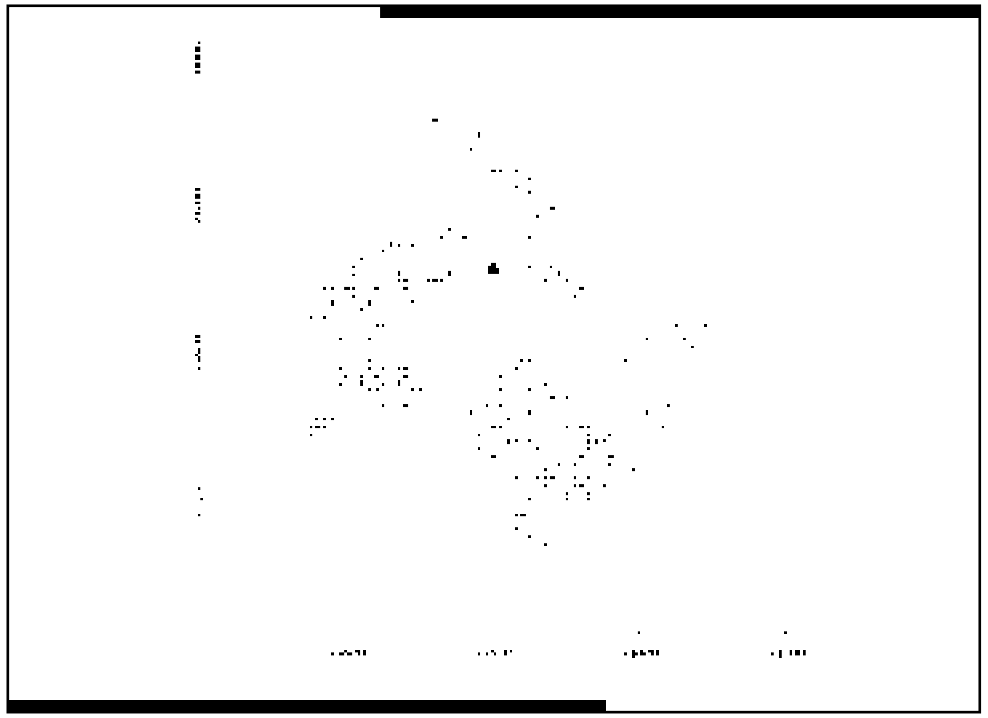


Figure 9.10: Locations of Childhood Controls



9.2. Method of Analysis

The primary analysis, as agreed beforehand, consisted of fitting the model of Diggle and Rowlingson[2,3] in order to test for significant elevation in risk of cancer close to the incinerator. This model assumes that the spatial distribution of cases has an intensity (mean number of cases per unit area) which varies spatially according to the equation

λ(lambda) (x) = λ_0(x) ρ {1+ α(alpha) exp (-d^2 /β(beta)^2) } exp { (z(x)^1 θ(theta)) } **(1)**

In the equation **(1)** *the symbols used have the following meanings:*

- x denotes location.
- λ_0(x) is the intensity of the population at risk, is related to the overall incidence of the disease in question.
- α represents the increase (or decrease if $\alpha<0$) in risk at the point source (incinerator).
- d is distance from the point source.
- β measures the rate at which risk declines towards its neutral value with increasing distance from the point source.
- Z(x) is a vector of the explanetory variables (age, sex, Townsend value) associated with the individual at location x.
- θ is the corresponding set of log-linear regression parameters.

By using case-control data to fit the model, we eliminate the nuisance function λ0(x) and obtain estimates of the parameters of interest, which are α, β, and, to a lesser extent in the present application, θ (theta). Our particular objective is to test the hypothesis of no relationship between risk and distance from the incinerator; in the model (equation 1) this corresponds to α=0 with β, indeterminate. The model accounts for possible risk effects of age, sex and deprivation by the log-linear regression term exp { (z(x) θ (theta) }. Supplementary analyses, intended to pick up qualitative departures for the assumptions implicit in the model (equation 1) used the generalised additive modelling methodology in Kelsall and Diggle 1998. This gives a non-parametric description of residual spatial variation in cancer risk after adjustment for the log-linear effects of sex, age and deprivation, which can be presented as a continuously varying grey-scale map. The surface is constructed using a kernal smoothing method essentially a spatial moving average. This methods requires the bandwidth (h) to be specified. This determines the spatial extent of the moving average. In this study the bandwidth (h) = 0.5 kilometres which is roughly equivalent to averaging over circular discs of radius 0.5 kilometres. In addition to an estimated surface of residual risk, this method provides an overall test of significance for departure from constant residual risk and, in the event that the overall result is statistically significant, an identification of any neighbourhoods where the estimated local risk is significantly (at the 5% level) higher or lower than the average for the whole area.

The advantage of this generalised additive modelling method is that it is less reliant than the Diggle and Rowlingson (1994) model on assumptions which may not hold for the data in question. Its disadvantage is that it leads to a statistical test with lower power against specific alternatives to the null hypothesis of constant risk, and is, therefore, effective only when applied to large data-sets. This is because the method attempts to estimate a whole surface of unspecified form, rather than a simple function of distance. In this particular study, therefore, the supplementary method of analysis was only applied to the cancer types that had large enough numbers of cases namely the three most common types of adult cancer : colorectal, lung and leukaemias and lymphomas.

9.3. Results

Table 9.3 shows the results of applying the Diggle and Rowlingson (1994) method to eleven data-sets consisting of:

- Six selected adult cancers
- All selected adult cancers
- All adult cancers
- Childhood leukaemias and lymphomas
- All selected childhood cancers
- All childhood cancers

The term "selected" means the cancer types listed in Table 9.2. It is important to note that the numbers of childhood cancers other than leukaemias/lymphomas were too small to permit any kind of analysis.

It is also important to note that the most common types of cancer are adult colorectal, lung and leukaemias/lymphomas. If emissions from the former incinerator had been a contributory factor to an increased risk of cancer, this would more likely show itself in the distribution of lung cancer (for which there is biological plausibility see Section 5), than in colorectal or leukamias/lymphomas for which there is no such plausibility.

In Table 9.3, the test statistic has a null sampling distribution which is approximately chi-squared on 2 degrees of freedom and this approximation has been used to compute the quoted p-values (Diggle and Rowlingson - 1994). **In all eleven analyses, the test for a distance-related component of risk does not achieve significance at the conventional 5% or even the less stringent 10% level. In other words, there is no association of higher risk of cancer, the nearer people live to the former incinerator site**. No adjustment has been made for multiple testing as each result should be interpreted separately in relation to any prior scientific hypothesis. However this adjustment is academic in this study as it would tend to render any nominally significant results non-significant rather than the other way round. Given that the results are non-significant, there is no justification for interpreting the point estimates of a and b, although they are included in table 9.3 for completeness. In particular, when the likelihood surface is almost flat (as indicated by a test statistic close to zero), the parameters are very poorly identified.

Table 9.3: Summary of results from fitting the Diggle and Rowlingson (1994) model

CANCER TYPE	α(ALPHA) VALUE	β(BETA) VALUE	TEST STATISTIC	ρ VALUE
Colorectal (adult)	0.65	0.44	2.27	0.32
Lung (adult)	12.88	0.0025	0.00	1.00
Adult larynx and nasopharynx	0.06	0.12	0.02	0.99
Adult leukaemia/lymphoas	0.38	0.28	1.33	0.51
Adult soft tissue sarcoma	$1.0*10^6$	0.34	2.85	0.24
Liver (adult)	1.20	0.043	0.56	0.75
Selected adult cancers	4.28	393.6	0.00	1.00
All adult cancers	0.82	31.06	0.00	1.00
Childhood leukaemia/lymphomas	4.32	0.13	4.17	0.12
Selected childhood cancers	0.37	0.18	1.12	0.57
All childhood cancers	4.71	0.07	4.49	0.11

Table 9.4 shows for completeness the results of testing the significance of age, sex and deprivation as risk factors for the different types of cancer under investigation.

AGE

Firstly, and as would be expected, the effect of age is highly significant and positive for all adult cancer types considered- the risk of developing adult cancer increases with your age - this applies universally to cancers not just to cancers in this particular population. Age was not a significant risk factor for childhood cancers.

SEX

Sex is highly significant for adult colorectal, lung and cancer of the larynx and nasopharynx with the risk higher for men than women. It is less significant for adult liver and leukaemias/lymphomas and not significant for adult sort tissue sarcomas. This may to some extent simply reflect the generally smaller sample sizes available for less common types of cancer. When the different types of cancer are pooled, the effect of sex is highly significant which is unsurprising because the estimated overall effect is dominated by the contributions of the more common types of cancer.

DEPRIVATION

Deprivation, as measured by the Townsend Index, is highly significant for adult colorectal, lung and laryngeal and naso-pharyngeal cancers. It is less significant for leukaemias and lymphomas and not significant for liver cancer and soft tissue sarcoma. As with the effect of sex, the lack of significance may be due to small numbers of cases rather than absence of a genuine effect - ie deprivation may well be a risk factor for these cancers but the numbers are too small to show this. Similarly, the effect of deprivation on the risk of childhood cancer is not significant but the the sample size is very small with only 49 childhood cancer cases between 1974 and 1988 available for analysis.

Table 9.4: Parameter estimates of Regression effects qassociated with age, sex and deprivation and the associated likelihood ratio tests of significance.

CANCER	^0 AGE	^0 SEX	^0 DEPRIVE P-VALUE	D AGE P-VALUE	D SEX P-VALUE	D DEPRIVE
Colorectal	0.076	-0.31	0.033	1651< 0.001	19.8<0.001	9.7<0.001
Lung	0.077	-0.97	0.086	2725< 0.001	322<0.001	105.6< 0.001
Liver	0.062	-0.56	0.71	88.5<0.001	5.2 0.02	3.5 0.06
Larynx/ nasopharynx	0.059	-1.34	0.125	140.7< 0.001	47.7<0.001	18.3<0.001
Leukaemia/ lymphoma	0.048	-0.30	0.043	288.7< 0.001	7.8 0.005	6.5 0.01
Soft tissue sarcoma	0.045	-0.32	-0.048	32.3<0.001	1.1 0.29	1.1 0.28
Selected adult	0.073	-0.85	0.078	2999< 0.001	299<0.001	103<0.001
All adult cancers	0.075	-0.14	0.036	6788< 0.001	16.0<0.001	47.4<0.001
Children's leukaemias/ lymphomas	0.041	-1.04	0.048	0.7 0.40	5.1 0.02	0.5 0.47
Selected childhood cancers	-0.006	0.18	-0.010	2.22 0.14	1.63 0.20	0.21 0.65
All childhood cancers	0.047	-0.44	-0.032	1.67 0.20	1.82 0.18	0.45 0.50

VARIATIONS IN THE PATTERNS OF ESTIMATED RISK FOR THE MOST COMMON TYPES OF ADULT CANCER (COLORECTAL, LUNG AND LEUKAEMIA AND LYMPHOMA)

The generalised additive modelling procedure (as outlined in 9.2) was used to examine more closely the pattern of variation in estimated risk for the most common types of adult cancer (colorectal, lung and leukaemia and lymphoma). As noted above, the research evidence (discussed in Section 5) whilst currently failing to establish conclusively that emissions from incinerators are a cause of cancers, does indicate that the links to some types of cancer are more plausible than others. This is due to what is already known about the causes of some types of cancer and ways in which cancer-causing substances might enter the body - e.g via the nose and mouth. In this study, therefore, it is more reasonable to suppose that any possible association with the incinerator is more likely to be shown in the results for lung cancer as opposed to colorectal or leukaemia and lymphoma.

Figures 9.11 to 9.13 show the estimated surfaces of residual spatial variation in risk for colorectal, lung cancers and leukaemia and lymphoma. The grey-scale on these maps is logarithmic to base 2, meaning that every unit increase on the grey-scale corresponds to a doubling of residual risk. The solid and dashed contour lines identify locations within the study area where the estimated local risk is significantly (at the 5% level) higher or lower than the estimated average risk for the whole study area. These significance contours are constructed by conducting separate significance tests at every location on a fine grid covering the study area and are therefore subject to the mis-interpretation due to the effects of multiple testing, unless the overall test of departure from the constant risk is also significant.

Figure 9.11: Kernal estimate of residual spatial variation in risk for adult colorectal cancers

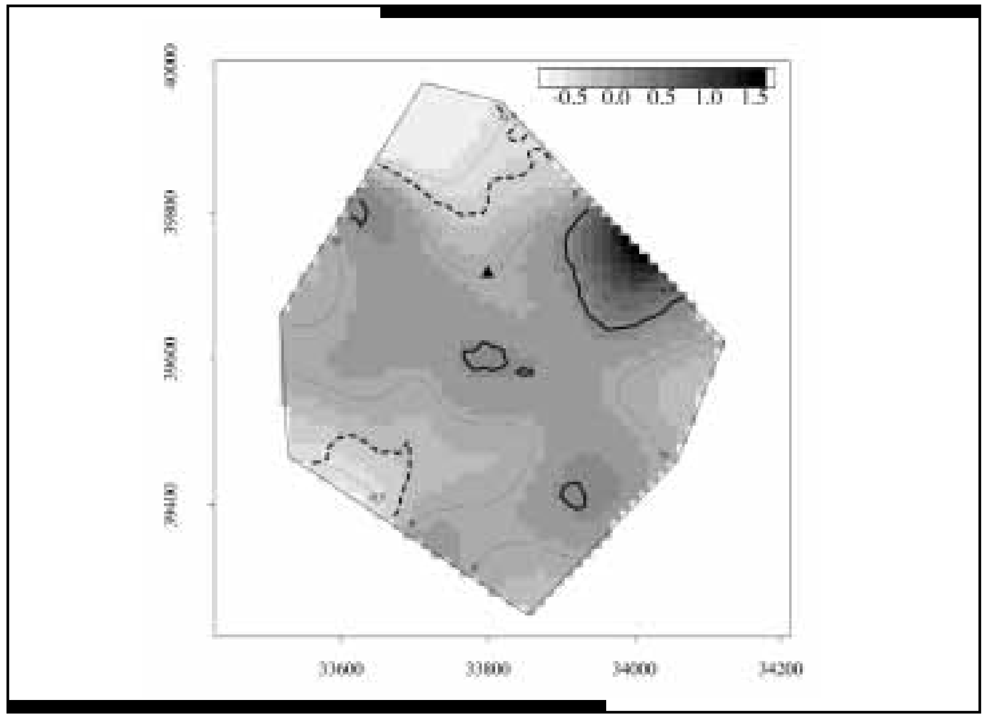


Figure 9.12: Kernal estimate of residual spatial variation in risk for adult lung cancers

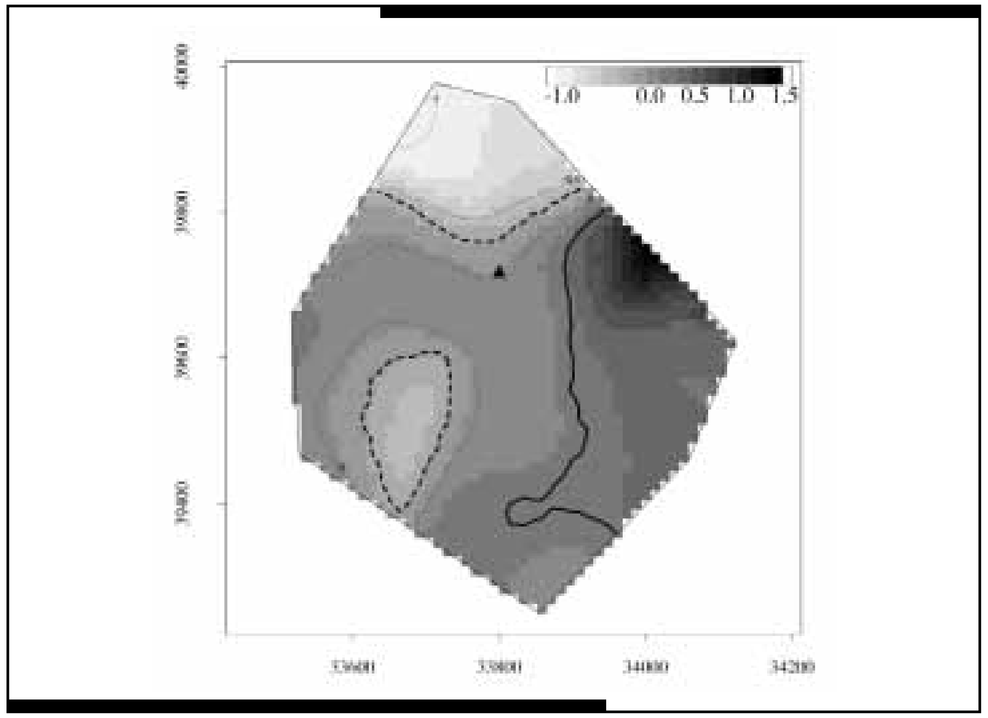
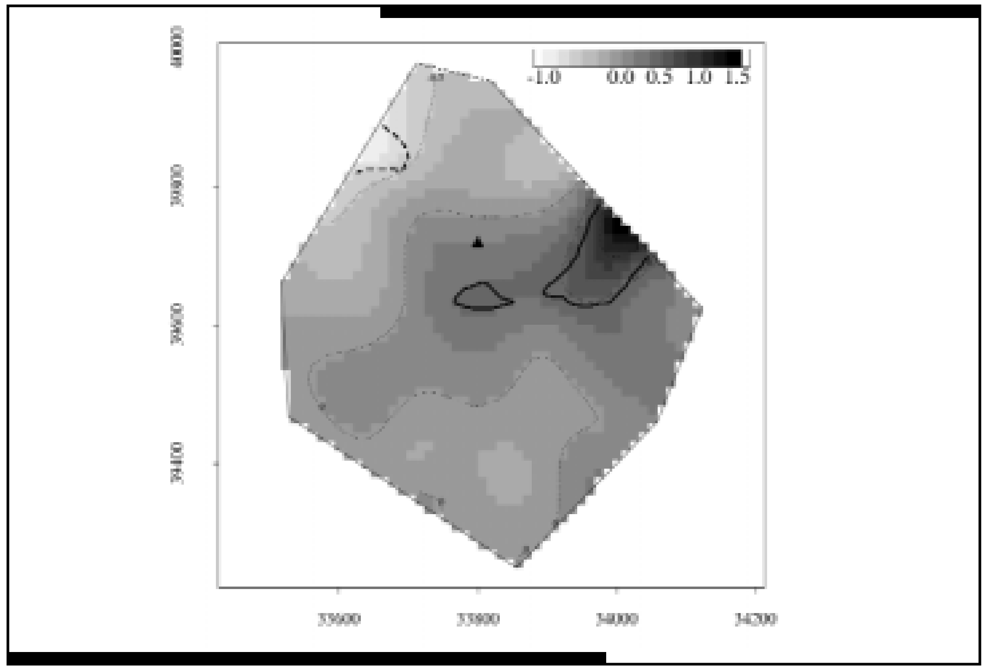


Figure 9.13: Kernal estimate of residual spatial variation in risk for adult leukaemias and lymphomas



All three maps show a common pattern of elevated risk 3 kilometres to the east of the site of the former incinerator at the eastern edge of the study area. The p-values for the overall test of departure from constant risk are:

- 0.05 for colorectal cancer (total number of cases in the whole study area 1974-1998= 1162)
- 0.01 for lung cancer (total number of cases in the whole study area 1974-1998=2345)
- 0.67 for adult leukaemia and lymphoma (total number of cases in the whole study area 1974-1998=390)

Bearing in mind the respective sample sizes for each type of adult cancer, the non-significant result for leukaemia and lymphoma may reflect insufficient numbers of cases to demonstrate an effect rather than absence of an effect. The statistically significant results for colorectal and lung cancers and the identification of elevated risk in all three types of cancer in approximately the same location would suggest that there is a genuine elevation in risk to the east of the former incinerator site across a range of cancer types. These include cancers like colo-rectal cancers which are strongly associated with lifestyle risk factors such as type of diet, obesity, lack of exercise and smoking and have no association (or biologically plausible link to) emissions from incinerators. The peak in this estimated risk is located 3km to the east of the former incinerator site at grid reference 34000,39750 which is just inside the Cherryfield ward in Knowsley.

9.4. Discussion

The primary analysis of the data reported above followed a protocol which had been agreed beforehand as a means of testing the null hypothesis of no spatial variation in risk in the immediate vicinity of the site of the former incinerator. The result was that the null hypothesis was accepted at the conventional 5% level of significance, for all the types of cancer considered (childhood and adult) after adjustment for age, sex and deprivation as measured by the Townsend index at enumeration district level. **The conclusion is that there is no increased risk of developing cancer the closer a person lives to the site of the former incinerator.**

The additional analysis of any variation in the residual risk of cancer for the most common types of adult cancer in the study area - colorectal, lung and leukaemia and lymphoma showed a consistent excess of cancer incidence centred on the location at grid reference (34000,39750). This at the eastern edge of the study area and is approximately 3 kilometres to the east of the site of the former incinerator. This excess was significant in two of the most common types of adult cancer - lung and colorectal. In the case of the former, as discussed in Section 8, the high rates of lung cancer in Liverpool and Knowsley have been known for some time and reported in several publications produced by MCCR. Smoking is known to be the major cause of lung cancer and is thought to account for 90% of cases . In the case of colo-rectal cancers, there is no biologically plausible link to incinerator emissions.

The overall conclusion is that there is a statistically significant spatial variation on the risk of the more common types of adult cancer in the study area but that the pattern of variation does not show a simple distance-based relationship to the site of the former incinerator. Possible explanations for this observed pattern of variation include the following:

STATISTICAL ARTEFACT

There may be some systematic discrepancy between the true spatial distribution of the population at risk and the population implied by the use of unit postcodes as controls; the use of unit postcodes as controls in this kind of analysis has been rightly criticised in the past; see for example Knox[5] and the subsequent discussion by Bithell and Draper[6]. However, this study the usual criticisms do not apply because the postcodes were obtained from a data-base of GP registrations showing, for each unit postcode within the study area, the number of registered patients sub-divided by sex and five-year age-bands. Hence, although the controls are not identifiable as individuals, they do represent a random sample from the population defined by GP registrations.

AN EFFECT OF THE INCINERATOR WHICH IS NOT DISTANCE-RELATED

There may be some local features and/or characteristics of the pattern of dispersal of emissions from the incinerator that leads to the area 3km east of the incinerator being more greatly affected than areas closer to the incinerator. Whether or not this is a plausible explanation would depend on information and expertise which we do not have available to us. Although this is unlikely given the results of measurements carried out by Environmental Health department at Liverpool City Council in the early 1990s (Section 6).

AN EFFECT DUE TO SPATIAL VARIATION IN AN UNMEASURED RISK FACTOR

Although our analysis accounts for age, sex and social deprivation, there are other risk factors for lung cancer. Most obviously smoking history as the primary casual risk factor for lung cancer and diet and levels of exercise as risk factors strongly associated with colo-rectal cancers which are not allowed for explicitly in this analysis, and any discrepancy in the spatial distribution of such risk factors between the cases and control samples could induce the unexplained spatial variation in risk which our analysis has found.

INCOMPLETENESS OF THE GP REGISTRATION DATA-BASE

If residents in the location where the residual elevated risk has been identified are less likely to register with a GP, their consequent under-representation amongst the controls would lead to a spuriously elevated estimate of risk in this location.

CONCLUSION

In the opinion of the research team from Lancaster University who undertook this geographical analysis, the last two of these four explanations are the most likely to be the reasons for the residual elevated risk, and should be investigated further. Note, in this connection that the study area embraces a diversity of land-use types, including residential, industrial and open space. Ideally, if any further investigation is undertaken, it should include a prospective case-control study to obtain data on individual-level lifestyle risk factors such as smoking, diet and exercise levels. Failing this, consideration should be given to obtaining information on additional risk factors at Enumeration Districts level.

CONCLUSIONS & RECOMMENDATIONS

10.1. General Conclusions

Currently in the UK, one third of all adults will die from some form of cancer. Thus cancers as a group of diseases are common. The investigation into the Fazakerley and Gillmoss concerns has taken account of the fact that Liverpool as a whole has high death rates from cancer. Over the period 1996-98 the average Standardised Mortality Ratio (SMR) for Liverpool for a common cancer such as lung cancer was in excess of 200 (persons) with the worst SMRs in the most socio-economically deprived wards. Vauxhall had the highest SMR for that period of 381. The high death rates for lung cancer were matched by high death rates for other diseases such as heart disease, chronic obstructive pulmonary disease and stroke associated with the 2 major risk factors - poverty and cigarette smoking.

In conducting this investigation, the Incident Team has been very aware of the complexity of investigating public concerns about the possible health consequences of incinerators and other industrial processes and environmental hazards. It was always going to be difficult to investigate a potential cancer cluster retrospectively when the main cause of concern no longer existed as a physical structure and therefore could not be monitored or tested. We needed to establish whether there was an increase in cancer cases that could not be explained by any other factor operating in the local population. Additional difficulties included establishing whether there any other potential environmental, economic, social or lifestyle risk factors and their relative contributions to cancer incidence in Fazakerley and also whether the incinerator contravened the air quality standards in place at the time of its operation. This proved to be extremely difficult due to paucity of primary information in some cases and its reliability in others. The following sub-sections set out the conclusions of the investigation and also important recommendations for further action. This investigation has led to the Incident Team to call for and initiate the development of a formal surveillance system for environmental public health. Lessons from this investigation are also being used to prepare national protocols and standards to assess health impacts of industrial processes.

10.2. Epidemiological Conclusions on Reviewing the Current Research on Incinerators and Cancer Risk

- Relatively few studies have been published on actual measured health effects in people associated with living near or working at incinerators.
- There has been no consistent pattern of an excess in specific cancers observed.
- The epidemiological evidence that chronic low-dose environmental or occupational exposure to incinerator emissions may cause cancer is weak.
- The possibility of adverse effects on health from living near or working at incinerators cannot be excluded because in general, studies tended to be too small in size to have the power to detect small increases in risk. Other major limitations are difficulties in controlling for other factors which might influence cancer rates, difficulty in measuring exposures accurately and the possibility of long latency between exposure and development of cancer.
- Future epidemiological studies need to address these limitations.
- Better exposure data are required.

10.3. CONCLUSIONS OF REVIEW OF CANCER CASES IN FAZAKERLEY 1974-1998

Currently in the UK, one third of all adults will die from some form of cancer. Thus cancers as a group of diseases are common. The investigation into the Fazakerley and Gillmoss concerns has taken account of the fact that Liverpool as a whole has high death rates from cancer. Over the period 1996-1998 the average Standardised Mortality Ratio (SMR) for Liverpool for a common cancer such as lung cancer was in excess of 200 (persons) with the worst SMRs in the most socio-economically deprived wards. Vauxhall had the highest SMR for that period of 381. The high death rates for lung cancer were matched by high death rates for other diseases such as heart disease, chronic obstructive pulmonary disease and stroke associated with the 2 major risk factors - poverty and cigarette smoking.

- In general the overall pattern of cancer in the study area was similar to that in LHA and Merseyside and Cheshire as a whole, in relation to the distribution by cancer site, gender and over time. The exception is lung cancer, for which the proportions were higher in the study area and LHA compared with Merseyside and Cheshire for males and females.
- Overall cancer rates were similar in the study area and LHA for males and females, however both were higher compared with Merseyside and Cheshire. This finding was significant in all cases except for females in 1989-93 when the study area had similar rates compared with Merseyside and Cheshire as a whole.
- There were no significant differences between the incidence rates in the study area, LHA and Merseyside and Cheshire for: leukaemias, non-Hodgkin's lymphoma, and cancers of the colon and rectum in males and females, and cancer of the larynx in males only.
- There were no significant differences in the incidence rates of childhood cancer in the study area, LHA and Merseyside and Cheshire as a whole.
- There were no significant differences between the study area and LHA as a whole for the rarer cancers considered. Some statistically significant differences were found for liver and laryngeal cancer in males and females respectively, between LHA and Merseyside and Cheshire as a whole, although these are difficult to interpret.

LUNG CANCER

The analysis undertaken by MCCR gave risk to the following conclusions in relation to lung cancer for the period 1974-1998:

- Lung cancer rates in the study area were not significantly different from rates in LHA for males and females, and over time.
- Lung cancer rates in the study area and LHA were significantly higher than rates for Merseyside and Cheshire as a whole for males in both 1979-83 and 1989-93, although rates are decreasing over time.
- Lung cancer rates in females in the study area and LHA were significantly higher than rates for Merseyside and Cheshire as a whole, in 1979-83 and 1989-93 and rates are increasing over time.
- The rate of lung cancer in females in the study area almost doubled between 1979-83 and 1989-93.
- The socio-economic profile of the study area is similar to that of LHA and both areas are more deprived overall compared with Merseyside and Cheshire.
- Within socio-economic group lung cancer rates in the study area were similar to those in LHA and Merseyside and Cheshire as a whole for males and females
- Lung cancer rates in females were higher in the study area for most socio-economic groups, although this was not statistically significant.

It could be suggested that since the study area constitutes a part of LHA (16%) it is the rates in the study area that could be causing the high rates in LHA, compared with Merseyside and Cheshire as a whole. If this were true, then we would except to see significantly higher rates in the study area, whereas in practice study area rates have been found to be similar to those of LHA. Thus we may conclude that, at least, some other areas in Liverpool must have rates similar to those of the study area. Colon and rectal cancers, chosen for their lack of known association with incinerator emissions, have shown similar patterns of distribution in the comparison areas over time, and by gender, suggesting that the study area does not generally have unusual background levels of cancer. The significantly higher overall lung cancer rates in the study area and LHA compared with Merseyside and Cheshire generally lose their statistical significance when rates are compared within socio-economic group. In this analysis however, numbers are small especially in the study area and LHA compared with Merseyside and Cheshire and this is further supported by their respective socio-economic profiles.

10.4. CONCLUSIONS OF GEOGRAPHICAL ANALYSIS

- After adjustment for the variations in risk factors attributable to age, sex and social deprivation, there is no significant evidence of a relationship between risk of developing cancer and proximity of residence to the site of the former incinerator.
- There were only 49 childhood cancer cases between 1974 and 1988 in the study area of L9,10 and 11 compared to 9941 cases of adult cancer.
- The effect of age is highly significant and positive for all adult cancer types considered- the risk of developing adult cancer increases with your age - this applies universally to cancers not just to cancers in this particular population. Age was not a significant risk factor for childhood cancers.
- Sex is highly significant for adult colorectal, lung and cancer of the larynx and nasopharynx with the risk higher for men than women. It is less significant for adult liver and leukaemias/lymphomas and not significant for adult sort tissue sarcomas.
- Deprivation, as measured by the Townsend Index, is highly significant for adult colorectal, lung and laryngeal and naso-pharyngeal cancers. It is less significant for leaukaemias and lymphomas and not significant for liver cancer and soft tissue sarcoma.
- The additional analysis of any variation in the residual risk of cancer for the most common types of adult cancer in the study area - colorectal, lung and leukaemia and lymphoma showed a consistent excess of cancer incidence centred on the location 3 kilometres to the east of the site of the former incinerator at grid reference (34000,39750) just inside the Cherryfield ward in Knowsley.
- There is a significant result of elevated residual risk for colorectal cancer which is strongly associated with lifestyle risk factors such as type of diet, obesity, lack of exercise and smoking and has no association (or biologically plausible link to) emissions from incinerators.
- The overall conclusion is that there is a statistically significant spatial variation on the risk of the more common types of adult cancer in the study area but that the pattern of variation does not show a simple distance-based relationship to the site of the former incinerator.
- The most likely explanations for this is that it is either due to an unmeasured individual-level risk factor such as lifestyle risk factors: smoking, diet, exercise levels etc or to under-representation of the residents of this particular location in the GP registration data -base.
- Ideally, any further investigation should a prospective case-control study to obtain data on individual-level lifestyle risk factors such as smoking, diet and exercise levels. Failing this, consideration should be given to obtaining information on additional risk factors at Enumeration District level.

10.5. Environmental and Toxicological Conclusions

EMISSIONS

- The incinerator under consideration no longer existed as it had been demolished following ceasing operation in September 1995. This meant that it was not possible to carry out test firings and record dispersion plumes.
- When the Fazakerley incinerator closed down in 1995, it met or was better than the standards, which were in place at the time.
- Liverpool City Council's Environmental Health Department's report in April 1994 concluded that the weekly monitoring programme since November late 1993 had failed to provide any evidence which would justify the City Council requesting additional or more frequent monitoring of emissions from either the hospital incinerator or boilers.
- The worst case scenario produced by the MERMAID model showed that the highest concentration of sulphur dioxide (SO_2) from the incinerator at ground level was **0.012-0.024 parts per million**. These concentrations were found to exist at greater distance from the source than the properties on Lower Lane and complied with all the then (1994) air quality standards.
- The review of the environmental data and reports conducted by Manchester Metropolitan University concluded that even with the "worst case" scenario produced by the MERMAID model the maximum pollutant concentration levels were indicative of the incinerator operating within regulatory limits.
- The lack of regulatory legislation and monitoring requirements for older incinerators prior to 1990 means that little data exists on the chemical emissions or their dispersion paths.
- Studies associating incinerators with specific disease entities are frequently based on proximity to the incinerators; associations cannot be accurately attributed to specific emissions or assessed for the effects of different concentrations of a chemical.
- Provisions in the legislation and the existence of Crown immunity prior to 1990 did not require emissions from hospital incinerators to be regulated or monitored.
- Current UK legislation will in future strictly regulate permitted levels of emissions from incinerators, and the Environment Agency will be required to co-consult with Health Authorities. The latter are expected to anticipate and assess the health impact of emissions on their local populations.
- The Government has called for multi-agency consultation and public participation assessing health impacts of new major developments and review of existing industries by either the Environment Agency or the Local Authority.
- It should be emphasised that the health impacts from waste incineration are still not well understood and the Environment Agency is working with the Department of Health to prepare national protocols and standards to assess health impacts. Members of the Incident Team are closely involved in this work.

10.6. Recommendations

ENVIRONMENTAL PUBLIC HEALTH SURVEILLANCE SYSTEM

Addressing the public health issues related to potential environmental hazard is a fundamental and long-standing role of public health practitioners and environmental health officers. Prior to 1974, Medical Officers of Health from Dr. WH Duncan (Liverpool and the world's first MOH) onwards had responsibility for the investigation and monitoring of these issues (see interview with Professor Semple Section 4.3.). This role was diluted with the move of Public Health from Local Authorities into the National Health Service in 1974 and the organisational separation from Environmental Health colleagues. Consultants in Communicable Disease Control who took on the former MOH responsibilities for communicable disease prevention and surveillance and environmental public health have tended (as implied by their title) to concentrate on the former rather than the latter. This is understandable as the 1980s and 1990s saw the emergence of HIV and other new important infectious diseases such as variant Creutzfeld-Jakob disease as well the re-emergence of tuberculosis, gonorrhoea and hospital-acquired infections.

Public Health has recognised the increasing importance of environmental public health as knowledge and awareness about the acute and chronic health implications of environmental pollution has improved. The establishment of the Environmental Epidemiology and Small Area Health Statistics Units at Imperial College London are good examples of this as are the Chemical Incident Response providers. The Chemical Incident Response Service, Guys and St Thomas' Hospital, London, (CIRS) is one of the services which provides and interprets toxicological information. In this investigation CIRS has provided summaries of chemical information, environmental toxicity and human toxicology and have offered assistance in investigating these clusters through scientific collaboration with experts in a number of agencies. New environmental legislation increasingly recognising the need for Public Health advice - for example, the UK Government enacted the new European Integrated Pollution Prevention Control Directive (IPPC) in July 2000. **This for the first time in UK law, required the Environment Agency to consult with Health Authorities on a statutory basis on the health implications of licensing new and existing major industrial processes.**

This means that investigations such as the subject of this report will no longer be isolated incidents but will increase in their frequency and complexity. This will put a major demand on the resources of Public Health which will need to increase its workforce and skills to be able to respond. Liverpool and Sefton Health Authorities were amongst the first Health Authorities in England to recognise the need for a dedicated environmental public health resource and in July 1998 appointed a Consultant in Public Health Medicine (KA) to take lead responsibility for environmental public health, and health impact assessment. In addition, in 2001 the Merseyside Health Authorities have appointed two Zonal Health Emergency Planning Officers to develop the public health response to major environmental incidents.

During the course of this investigation, it has become increasingly apparent that there is a need for an active environmental surveillance system, similar to the formal national and regional surveillance systems for communicable disease, in order to provide the necessary information intelligence to interpret and monitor public health concerns adequately. Currently there is considerable information stored in the two cancer registries in the North West as well as information held by the Congenital Malformations Surveillance System (CESDI) at Liverpool University, Local Authority Planning and Environmental Health Departments, the Environment Agency, the Health and Safety Executive and other partner agencies.

The Incident Team is recommending that a proposal to bring together information from these different sources into a regional surveillance system which would actively monitor the health of populations following chemical incidents and also monitor prospectively the health of populations around existing or new major industrial or environmental hazard sites - such as those identified through IPPC consultation or the new Contaminated Land registers.

A feasibility study lead by Drs Kate Ardern and Sarah Woodhouse has already commenced. Preliminary discussions are being held with key agencies and the recommendation has the support of the Regional Epidemiologists, the Directors of the North West and Merseyside and Cheshire Cancer Registries and Dr Chris Harrison, the Cancer Intelligence Network lead for the North West (NHS) Regional Office.

COMMUNICATING RISK

- Public concern about the hazards of emissions existed even in the 1950s but there was little acknowledgement of this. There has been a sea change in attitudes to the public concerns about environmental impact on health.
- There is uncertainty about the impact of incinerator emissions on human health. It is the role of the government and of Public Health to communicate this uncertainty in an honest but understandable manner to the public.
- Issues about waste reduction, curtailing waste and the implications of incineration as a disposal option should be openly debated, so that the scientific community and the public are empowered to influence future projects and to minimise the consequent adverse effects on human health and wellbeing.

The Incident Team recommends that the lessons learnt from this study are used by Governmental Advisory Committees to develop better public information on the potential risks to health from industrial processes and to consider how to develop public participation in reviewing research evidence. For example, the Cochrane Collaboration has a model of patient and service user involvement that could assist in this.

The Incident team recommends that lessons from this study on communicating risk are incorporated into the work currently being undertaken by the Chemical Hazards Management and Research Centre at Birmingham University and the Chemical Incident response Service at Guys and St Thomas' Hospital London in developing the Health Authorities' response, in their new role as statutory consultees, to Integrated Pollution Prevention Control applications .

The Merseyside and Cheshire Cancer Registry has already undertaken to revise and update its public information on cancer as a result of participating in this study

DISSEMINATION OF THIS REPORT

This report will be formally presented to both Liverpool Health Authority and Liverpool City Council. It will then be made publicly available by a formal launch with copies supplied to all key stakeholders and a formal media launch to be held in Autumn 2001. Following its launch it will be published on the **NW Regional Public Health Observatory's website: www.nwpho.org.uk** and the **Liverpool Health Authority website: www.liverpool-ha.org.uk**.

Copies of this report are also being sent to the following Government Advisory Committees:

- The Committee for the Medical Effects on Air Pollution.
- The Committees on Toxicity, Mutagenicity, Carcinogenicity of chemicals in Food, Consumer Products and the Environment.

Section 3

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Section 4

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Section 5

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Table A1: Characteristics of included studies of cancer in population living near incinerators

METHODS	PARTICIPANTS	EXPOSURE	OUTCOMES	NOTES
RETROSPECTIVE COHORT **Confounding:** **Adjusted for socio-economic confounding.**	Study population: Population within 10 km of 10 incinerators 390 cases recorded 5 years after each site was registered or began operation; (410 cases excluded due to lag-time) 70 cases recorded 10 years after each site was registered or began operation. (further 320 cases excluded due to lag-time) Comparison group: National population cancer incidence rate (regionally adjusted) Study duration:1974-84 (England and Wales) and 1975-87 (Scotland)	Distance from 10 incinerators of waste solvents and oil in Great Britain. 1) within 3km (2) within 3-10km Lag-periods of 5 and 10 years.	Cases of cancer of larynx and lung for which a full postcode was available, within 10 km of exposure. No statistically significant excess when compared with expected nor trend of decreasing risk with distance.	Limitations noted by authors include incompleteness of cancer-registration; migration not taken into account; limited lag-time; limitations in exposure data.
OBSERVATIONAL RETROSPECTIVE COHORT **2 stage study** **(1) random sample 20 incinerators stratified by population size;** **(2) significant findings tested in the remaining 52 incinerators.** **Confounders:** **Deprivation was considered by:** **a) deprivation-index,** **b) liver cancer incidence compared with other cancers.**	Study-population: People living near all municipal solid-waste incinerators in UK, burning household, commercial and/or industrial waste, for which publicly available information was available. >14 million males and females living near 72 incinerators identified. Comparison-with national cancer incidence rates (regionally adjusted); stratified by deprivation; liver cancer incidence subsequently compared with all cancers except liver and also with stomach and lung. Study-duration: up to 13 years; 1974-86 England 1974-84 Wales 1975-87 Scotland 2 different lag periods used – 5 and 10 years.	1) Living within 7.5km of incinerator. 2) Living within 3 km of incinerator. Incinerators built after 1976 were excluded. Model of declining exposure with distance assumed.	Cancer-incidence measured(cancer registry). With 10 year lag, at 1 km, observed/expected ratios: All cancers: 1.06,stomach cancer: 1.14, colorectal cancer: 1.04, liver cancer: 1.37, lung cancer: 1.14. Liver cancer excess reduced from 37% to 21% when incidence compared with other cancers. Significant decline in risk with distance for all cancers combined, stomach, colorectal, liver and lung cancers; $p<0.05$. No decline for non-Hodgkins lymphoma and soft-tissue sarcomas.	Authors note possibility of residual socio-economic confounding

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METHODS	PARTICIPANTS	EXPOSURE	OUTCOMES	NOTES
OBSERVATIONAL; **Revision of Elliott 1996**	Study population: 235 cases of liver cancer identified in Elliott 1996. All 87 cases at <1km from municipal waste incinerator and random samples of 74 from each of 1-7.5km and >7.5km. 119 cases of liver cancer selected using criteria of availability of histopathological material and/or medical records;	Distance from 52 municipal solid waste incinerators in Great Britain.	55% of registered primary liver cancer cases were confirmed as primary liver cancer; 18% confirmed as definite secondaries; giving revised estimates of between 0.53 and 0.78 excess cases per 10^5 per year within 1km.	
PROSPECTIVE COHORT **followed from 1975 to 1990**	Cohort of population of area defined as polluted by incinerator, comprising 4131 individuals. Comparison groups: Control cohort was population of a non-polluted area of similar socio-economic background, comprising 5946 individuals;	Computerised simulation model used to define area polluted by a waste incinerator.	Cancer incidence recorded in regional cancer registry. No difference found in cancer incidence.	Only abstract available.
RETROSPECTIVE CASE CONTROL **Confounders:** **Adjusted for age, smoking (next of kin questionnaire), likelihood of exposure to occupational carcinogens (expert evaluation) and levels of air particulate.**	Study population: 938 cases identified from cancer registry of histologically confirmed lung cancer among males resident in the province of Trieste who died from 1979 to 1981 or from 1985 to 1986; age not specified. 755 cases included; 182 excluded because failed to find next of kin; 1 excluded because lived outside province. Comparison group: random selection of 755 male controls; resident in province of Trieste who died within the same 6 month period at the same age +/– 2 years; deaths due to chronic lung disease/cancer of the upper aerodigestive-tract /urinarytract /pancreas/liver/GIT were excluded.	Distance from 4 sources of environmental pollution (shipyard, iron foundry, incinerator and city-centre); risk gradient and directional effects estimated separately for each. Operation-timespan of incinerator unknown; length of residence not individually assessed.	Relative risk of death from lung cancer at distances from point sources of pollution. Excess relative risk found at city-centre and at location of incinerator. Highest excess risk at city-centre, with a slow decline in risk moving away from city-centre. The excess risk at the incinerator after adjusting for individual risk-factors and city-centre was 6.7; p=0.0098 with a very rapid decay in risk, moving away from incinerator.	Environmental exposure was adjusted for total particulate deposition and residence location. Authors note that residual confounding due to other unmeasured exposure cannot be excluded; selection bias cannot be excluded; misclassification bias due to a change in residence cannot be excluded
ECOLOGICAL **Confounding:** **Allowed for socio-economic status, age and sex.**	Study-population: Population within 10 km radius of area; 341,389 males and females. Number of observed deaths from different types of cancer. Comparison: Expected mortality rate; (not specified how calculated, e.g. whether regionally adjusted). Setting: a suburb of Rome Study duration: 1987-1993	Distance from a large waste disposal site, a waste incinerator plant and an oil refinery plant, operational since the 1960s. Incinerator closed in 1985. No differentiation between sources of exposure. Divided into bands:0-3km, 3-8km, 8-10km.	Gender-specific mortality from cancer of the liver, larynx, lung, kidney, lymphatic and haema-topoietic systems as defined by death-certificates. Measured by: (1) excess risk, (2) decline with distance. Decline with distance in laryngeal cancer mortality in men (p=0.06) but no significant excess risk. Standardised mortality ratio of 198 for mortality from cancer of the kidney in women in the 3-8km band (95%CI=111-325). Otherwise no significant excesses found.	Reported significant decline with distance in mortality from laryngeal cancer (p=0.03) but not significant when adjusted for socioeconomic index. Authors note possibility of finding significance results by chance because so many tests were carried out.

METHODS	PARTICIPANTS	EXPOSURE	OUTCOMES	NOTES
ECOLOGICAL **Cluster analysis.** **Confounding:** **controlled for age, sex and patterns of medical referral.**	Incident cases of soft-tissue sarcoma (STS) and non-Hodgkins lymphoma (NHL) in the department of Doubs (485,000 inhabitants);110 cases STS observed and 803 cases NHL observed. Comparison: expected number of cases for each canton: incidence rates for the whole department adjusted for person-years for each canton and adjusted for gender and age. For the cluster analysis, cases of Hodgkins disease were used as the control group (noted by authors as not consistently associated with dioxin exposure); 176 cases observed. Setting: 26 cantons of the departement of Doubs, France Study duration: 16 years from 1980 – 1995	Clustering around a municipal solid waste incinerator; high dioxin emissions* operating since 1971; urban setting 4km west of city centre.	An excess of 14 cases of STS was found; standardised incidence ratio of 1.44. An excess of 61 cases of NHL was found; standardised incidence ratio of 1.27. Significant clusters of STS (p=0.004) and NHL (p=0.0003) were found around the incinerator; there were no clusters anywhere else; no cluster was found for Hodgkins.	Authors note that confounding by socioeconomic status, urbanisation or patterns of medical referral seem unlikely to explain the clusters. Hodgkins disease follows same referral patterns as NHL. Data aggregated to canton level; Note: "area around incinerator" is 2 cantons in size.
GEOGRAPHICAL **Migration studies of childhood cancer**	Study-population: 9224 children who had died from cancer before their 16th birthday in Great Britain and who had moved at least 0.1 km between birth and death. 4385 or 47.5% whose addresses at birth and death were known were included. No control group. Study duration: 1953-1980	Distance from nearest source at birth and death: 70 municipal waste incinerators, 307 hospital incinerators, 460 toxic waste landfill sites.	Ratio between numbers of children migrating away from compared with towards their nearest hazard. Significant relative risk of outward migration ranging from 1.25 to 2.26, depending on exclusion of incinerators whose timespan did not enclose the lifespan of the child, proximity to incinerator and age of incinerator.	Could not separate out effects of other industrial sources close to incinerators. No confidence intervals for the ratios are reported. Analysis examined directional biases; no evidence of significant wind-direction effects reported.

***Dioxin concentration in emissions in December 1997 was 16.3 ng international toxic equivalency factor (I-TEQ)/m^3. In 1994, the European Union limited dioxin emissions from municipal solid waste incinerators to 0.1ng I-TEQ/m^3.**

Dioxin concentrations in cows milk in nearby farms were 0.58 (2km), 0.59(1.5km) and 1.03 (0.9km) ngI-TEQ/kg of fat. The common guideline for cow's milk is 6 ngI-TEQ/kg of fat.

Table A2: Characteristics of included studies of cancer in workers at incinerators

METHODS	PARTICIPANTS	EXPOSURE	OUTCOMES	NOTES

RETROSPECTIVE COHORT
 	
Confounding: smoking data of cohort obtained by surrogate interview, validity concerns.
	Exposed group: All workers at an incineration plant who had been working at the plant for at least 1 year between 1920-1985: 183 males / 4412 person-years.
	
	Comparison group: National and local population mortality rates; gender not specified. Standardised by 5 year age and 5 year calendar-time.
	
	Setting: plant outside Stockholm
	
	Study-period: 1951-1985
	
	Those who died before 1951 (4), had emigrated (2) or were lost to follow-up (1) were excluded.
		Employment at the plant for at least 1 year between 1920 and 1985. Household & industrial wastes incinerated until 1955; only household waste since then.
		
		No subdivision by job-type. Reported that almost all had started as general process workers and that tasks were intermixed.
			Standardised mortality ratios:
			
			LOCAL RATES
			
			all causes SMR=99;
			
			95% CI=79-122.
			
			all cancers SMR=107;
			
			95% CI=67-162.
			
			Lung cancer SMR=197;
			
			95% CI=90-374.
			
			NATIONAL RATES
			
			All causes SMR=113;
			
			95% CI=90-140.
			
			all cancers SMR=135
			
			95% CI=85-205.
			
			Lung cancer SMR=355;
			
			95% CI=162-675.
				Local comparison group likely to be more appropriate.
				
				22 cancer deaths and 9 lung-cancer deaths observed.
				
				Small numbers / power; wide confidence intervals.
				
				Only mortality measured. Different lengths of exposure from as little as 1 year; outcome measured in 1985 so inadequate latency period.

RETROSPECTIVE COHORT
 	
Confounding: inadequate smoking data.
	Exposed group: All workers ever employed at 2 garbage plants between 1962-1992: 532 males / 8385 person-years of observation.
	
	Comparison group: Regional population mortality rates; gender not specified. Standardised by 5 year age and 5 year calendar-time.
	
	Setting: suburbs of Rome
	
	Study-period: 1962-1992
	
	Loss of follow-up of 8 subjects.
		Ever employment at 2 municipal plants for garbage recycling and incinerating. 13 incinerators operating between 1962 and 1985 and then 12 shut. Exposure data incomplete, lack of details of work activities.	Standardised mortality ratios:
			
			all cause SMR =0.71; 90% CI=0.51-0.95.
			
			all cancers SMR=0.95; 90% CI=0.58-1.46.
			
			lung cancer SMR=0.55;
			
			90% CI=0.15-1.42.
			
			gastric cancer SMR=2.79;
			
			90%CI=0.94-6.35.
				15 cancer deaths and 4 lung cancer deaths observed. Low power/Small no.; Wide confidence intervals.
				
				Only mortality measured. Outcome measured in 1992 so inadequate latency period.

Table A3: Summary information on pollutant types by health effects and guidelines for exposure limits

POLLUTANT	TOXIC EFFECTS AND RECOMMENDED EXPOSURE LEVELS
PARTICULATES including smoke, soot dust, PM_{10} and total particulates	Pollutants associated with particulates may include poly aromatic hydrocarbons, nitrosamine, transitional metals and other chemicals **Short- and long-term health effects** *Non-carcinogenic effects:* ▪ Acute exposure associated with increased cardiovascular and respiratory morbidity and mortality, more frequent asthma attacks. ▪ Long term exposure effects difficult to assess but a decline in lung function may be accelerated. **Exposure limits:** No threshold established below which no adverse events including concerns about ultrafine particles. UK EPAQS: set 50_g/m³ (equivalent to 1mg in 24 hours) Committee on Medical Effects of Air Pollution have four bands:[iv] PM_{10} concentration <50µg/m³ = low concentration PM_{10} concentration 50-74µg/m³ = moderate concentration PM_{10} concentration 75-99µg/m³ = high concentration PM_{10} concentration >100µg/m³ = very high concentration
Metals: examples: arsenic, cadmium, chromium, mercury and nickel	
ARSENIC	Waste incineration products may exceed LAQS[1] **Chronic clinical effects** *Non-carcinogenic effects:* ▪ Chronic arsenic ingestion is characterised by weakness, anorexia, gastrointestinal disturbance, impairment of cognitive function, peripheral neuritis and neuropathy, hepatomegaly, jaundice, irritation of nose and throat, perforation of nasal septum, and skin disorders including ulceration, hyperkeratosis of palms and soles, hyperpigmentation, eczemoid and allergic dermatitis. ▪ Conjunctivitis, keratoconjunctivitis, corneal necrosis and ulceration have been reported. Pigment spots on the corneal and conjunctival epithelia accompany the pigmentation of the skin. Brittle nails with Mee's lines (white transverse discoloration) are common. ▪ Myocarditis has been reported following chronic poisoning. Pancytopenia, aplastic anaemia or leukaemia has been reported following chronic exposure. *Carcinogenic effects:* ▪ IARC2 Group 1(Carcinogenic to humans) ▪ Arsenical dermatoses, epidermal carcinoma and lung, liver, bladder, larynx, lymphoid system, viscera and kidney cancers are associated risks of prolonged or repeated exposure to arsenic compounds. *WHO 1987 air quality guidelines:* "because arsenic is carcinogenic and there is no known safe threshold, no safe level…can be recommended" **Exposure limits** OSHA: Arsenic 10_g/m³ HSE: Arsenic (excluding arsine) 0.1_g/m³ (MEL;8-hr TWA RP) Arsine 0.2_g/m³ (OES 8-hr TWA RP)

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POLLUTANT	TOXIC EFFECTS AND RECOMMENDED EXPOSURE LEVELS
CADMIUM (Cd)	Waste incineration products may exceed SAQS[3] and LAQS Human contact primarily through food High Cd levels in cigarettes (10% absorbed) **Chronic clinical effects:** *Non-carcinogenic effects at occupational levels:* ▪ respiratory system: emphysema genito-urinary: proteinuria, kidney damage, stones. osteomalacia, yellow staining of the teeth, anaemia, anosmia also reported ▪ Itai-Itai (ouch-ouch) disease bone disease with kidney dysfunction ▪ Carcinogenic effects at occupational levels-increased risk of lung cancer, prostate cancer **Exposure limits:** OSHA*: Cd dust - 200_g/m3 Cd fumes 100_g/m3 HSE**: Cd and Cd compounds 25_g/m3 (MEL;8-hr TWA RP) Cd oxide fume 25_g/m3 (OES; 8-hr-TWA RP) 50_g/m3 (OES; 15-min RP) Cd sulphide and sulphide pigments 40_g/m3 (OES; 8-hr TWA RP)
CHROMIUM	Possible maximum ground level concentration could be significantly higher than LAQS. *Non-carcinogenic effects:* ▪ Damage to nasal septum, dermatitis *Carcinogenic effects:* ▪ Chromium (VI) IARC Group 1 (carcinogenic to humans). Associated with lung cancer in occupational life-time exposure studies. **Exposure limits:** OSHA: Metallic chromium and insoluble salts 1000:g/m3 (TLV++; 8-hr TWA RP) Cr (II) and (III) soluble salts 500_g/m3 (TLV; 8-hr TWA RP) Cr (VI) and chromic acid 100_g/m3 (TLV; 8-hr TWA RP) HSE: Cr (III) 500_g/m3 (OES; 8-hr TWA RP) Cr (VI) 50_g/m3 (MEL; 8-hr TWA RP)

POLLUTANT	TOXIC EFFECTS AND RECOMMENDED EXPOSURE LEVELS
MERCURY	Bioaccumulative **Chronic clinical effects:** *Non-carcinogenic effects:* ▪ CNS disturbances: headaches, personality changes, delirium, irritability, ataxia, paraesthesiae, excessive perspiration, blushing, bilateral fine tremors, ▪ Nausea, vomiting, conjunticvitis, pyrexia ▪ Gingivitis, stomatitis are commonly described; dark spots or blue lines along the gums may occur ▪ Renal dysfunction and acute renal failure ▪ Lens and corneal disease with impaired vision may occur ▪ Acrodynia – severe leg cramps, irritability, maculopapular rash and peeling skin on fingers and feet. *Carcinogenic effects:* ▪ Inconclusive. IARC Group 2B (possibly carcinogenic in humans.) **Exposure limits:** OSHA: Mercury vapour 0.05_g/m3 HSE: Mercury and mercury compounds 0.025_g/m3 (OES; 8-hr TWA RP) except alkyls Mercury alkyls 0.01_g/m3 (OES; 8-hr TWA RP) 0.03_g/m3 (OES; 15-min RP)
NICKEL	Possible maximum ground level concentration could be significantly higher than LAQS. WHO (1987) unable to recommend safe air levels. **Chronic clinical effects** *Non-carcinogenic effects:* ▪ Chronic exposure to nickel dust may cause eczematous dermatitis, asthma and Loeffler's syndrome (pulmonary eosinophilia).[2] Nasal irritation, damage of nasal mucosa, perforation of the nasal septum and loss of smell have occasionally been reported following chronic exposure to nickel aerosols and other contaminants.[7] ▪ Pneumoconiosis has been reported following occupational exposure to nickel dust, although exposure to other known fibrogenic substances could not be excluded. ▪ Chronic exposure to nickel carbonyl may lead to decreased serum monoaminoxidase and EEG abnormalities. *Carcinogenic effects:* ▪ IARC classifies nickel compounds as human carcinogens and metallic nickel as a possible carcinogen. IARC Group 1 (carcinogenic to humans) Lung and nasal cancers. ▪ Chronic exposure may lead to nasal carcinomas, nasal epithelial dysplasia, lung cancer and potentially cancer of the larynx.

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POLLUTANT	TOXIC EFFECTS AND RECOMMENDED EXPOSURE LEVELS
NICKEL continued	**Exposure limits:** OSHA: Nickel compounds that dissolve easily in water 100_g/m^3 (8-hr day, 40-hr week) Nickel compounds that do not dissolve easily in water 1mg/m^3 HSE: Metallic nickel 0.5mg/m^3 (MEL; 8-hr TWA RP) Soluble nickel compounds 0.1mg/m^3 (MEL; 8-hr TWA RP) Insoluble nickel compounds 0.5mg/m^3 (MEL; 8-hr TWA RP) Organic nickel compounds 1mg/m^3 (OES; 8-hr TWA RP) 3mg/m3 (OES; 15-min RP)
Acid and combustion gases: example: sulphur dioxide	
SULPHUR DIOXIDE (SO_x)	*Non-carcinogenic effects:* ▪ increased cardiovascular and respiratory morbidity and mortality *Carcinogenic effects not proven.* **Exposure limits:** EPAQS: 100 ppb(267_g/m^3; 15-min averaging period) HSE: 5 mg/m3 (8-hr TWA RP)13 mg/m^3 (15-min RP)
Organic compounds: examples: dioxins and furans, poly aromatic hydrocarbons, polychlorinated biphenyls	
DIOXINS AND FURANS	Much public concern May be produced in waste incineration in quantities, which exceed SAQS and LAQS. Bio accumulative Environmental information: Air quality and incinerator generated particulate mass were measured to determine if incinerators caused adverse respiratory effects. Authors did not detect differences in concentrations of particulate matter among the three pairs of study communities in USv **Chronic Clinical Effects** *Non-carcinogenic effects:* ▪ Chloracne associated with high levels of exposure ▪ Links between chronic exposure to TCDD and cardiovascular disorders, such as atherosclerosis and myocardial infarction, have been investigated; these studies have not shown these disorders to be conclusively related to TCDD exposure. Enzyme induction is prominent in chronic exposure. Workers exposed to dioxins and who consume alcohol seem to have a greater risk of elevated serum GGT concentration; the risk increases with dioxin concentrations[3,9] ▪ Self-limiting haemorrhagic cystitis has been reported following chronic exposure to TCDD-containing soil[10] *Carcinogenic effects:* Dioxin is a probable human carcinogen, linked with soft tissue sarcomas. **Exposure limits:** COT‡ 2,3,7,8-TCDD 10pg/kg bw/day (TDI)[4]

POLLUTANT	TOXIC EFFECTS AND RECOMMENDED EXPOSURE LEVELS
POLY AROMATIC HYDROCARBONS (PAHs)	**Chronic Clinical Effects** *Carcinogenic effects:* ▪ IARC Group 2 (probably carcinogenic to humans). Probably carcinogenic as a chemical mixture as people are not exposed to single PAHs. ▪ Cancer is the most significant effect of chronic PAH exposure. Epidemiological studies have shown increased mortality due to lung cancer in humans exposed to coke oven emissions, roofing-tar emissions, and cigarette smoke. Occupational exposures have been attributed to increased incidences of urinary bladder cancer, asthma-like symptoms, chronic bronchitis and cataracts. *Non-carcinogenic effects:* ▪ Non-carcinogenic effects reported include photosensitivity and eye irritation, leukoplakia, haematuria and immunosuppression. Corneal ulceration, chorioretinitis, scotoma, decreased visual acuities and lenticular opacities have also been reported. Benign and reversible verrucae were reported in a chronic dermal study.[1] Naphthalene exposure from bedding and nappies stored with mothballs has induced haemolytic anaemia in babies. **Exposure limits:** OSHA: BaP 0.2 mg/m^3
POLYCHLORINATED BIPHENYLS (PCBs)	*Non-carcinogenic effects:* ▪ Few adverse effects definitely associated with low level long-term exposure. *Carcinogenic effects:* ▪ Inadequate evidence for carcinogenicity **Exposure limits:** OSHA: 42% chlorinated biphenyls 1mg/m^3 (PEL[5]; 8-hr TWA RP) 54% chlorinated biphenyls 0.5mg/m^3 (PEL ; 8-hr TWA RP) HSE: 42% chlorinated biphenyls 1mg/m^3 (OES ; 8-hr TWA RP) 54% chlorinated biphenyls 0.5mg/m^3 (OES ; 8-hr TWA RP)

[a] The Royal Commission on Environmental Pollution was appointed in 1970 "to advise on matters, both national and international, concerning the pollution of the environment; on the adequacy of research in this field; and the future possibilities of danger to the environment".

[1] LAQS: long term air quality standards

[2] IARC: International Agency for Research on Cancer

[3] SAQS: short term air quality standards

* OSHA: Occupational Health and Safety Administration

** HSE: Health and Safety Executive

\+ TLV: Threshold limit value

‡ COT: UK Committee on Toxicity of Chemicals in Food

[4] TDI: Tolerable daily intake

[5] Permissible Exposure Level

Statistical Methods Appendix

CRUDE RATES
These rates were calculated by dividing the total number of cases by the population and are expressed as a rate per 100,000. This rate allows a comparison of areas but does not account for any differences in the age structure of the population.

CUMULATIVE RATE (CR)
The cumulative rate provides a method of direct age standardisation. The advantage of the CR is that it avoids the problem of using a reference value. This measure is the sum of the age specific rates for each age band from birth to age 74.

The CR approximates to the Cumulative Risk and therefore has greater intuitive appeal. It can be interpreted as the risk of developing cancer by the age of 75. For CR values of less than 10% the difference between the CR and Cumulative Risk is small but for larger values of the CR the cumulative risk is overestimated without correcting the rate using the following formula:

Cum. Risk = 100 x [1-exp)-Cum. Rate/100)]

AGE STANDARDISED RATE (ASR)
The ASR is the theoretical rate that would occur if the observed age specific incidence rates applied in a standard population. The standard population used in this report is the European standard.

ASR = Age Specific Incidence Rate x Standard Population 100,000 for each age group

The results give, for each age band, the number of cases that would occur in the standard population if the local rates were applied. Summing these numbers produces the ASR.

Table A4. European Standard populations[1]

AGE GROUP	EUROPEAN
0-4	8000
5-9	7000
10-14	7000
15-19	7000
20-24	7000
25-29	7000
30-34	7000
35-39	7000
40-44	7000
45-49	7000
50-54	7000
55-59	6000
60-64	5000
65-69	4000
70-74	3000
75-79	2000
80-84	1000
85+	1000
Total	100,000

[1]*Source: 1991 World Health Annual of Statistics – based on J. Waterhouse et al (eds) Cancer Incidence in five continents, Lyon, IARC (Vol. 3, page 456).*

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