RESEARCH REPORT

Adverse pregnancy outcomes around incinerators and crematoriums in Cumbria, north west England, 1956–93

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Study objective: To investigate the risk of stillbirth, neonatal death, and lethal congenital anomaly among babies of mothers living close to incinerators and crematoriums in Cumbria, north west England, 1956–93.

Design: Retrospective cohort study. Logistic regression was used to investigate the risk of each outcome in relation to proximity at birth to incinerators and crematoriums, adjusting for social class, year of birth, birth order, and multiple births. Continuous odds ratios for trend with proximity to sites were estimated.

Setting: All 3234 stillbirths, 2663 neonatal deaths, and 1569 lethal congenital anomalies among the 244 758 births to mothers living in Cumbria, 1956–1993.

Main results: After adjustment for social class, year of birth, birth order, and multiple births, there was an increased risk of lethal congenital anomaly, in particular spina bifida (odds ratio 1.17, 95% CI: 1.07 to 1.28) and heart defects (odds ratio 1.12, 95% CI: 1.03 to 1.22) around incinerators and an increased risk of stillbirth (odds ratio 1.04, 95% CI: 1.01 to 1.07) and anencephalus (odds ratio 1.05, 95% CI: 1.00 to 1.10) around crematoriums.

Conclusions: The authors cannot infer a causal effect from the statistical associations reported in this study. However, as there are few published studies with which to compare our results, the risk of spina bifida, heart defects, stillbirth, and anencephalus in relation to proximity to incinerators and crematoriums should be investigated further, in particular because of the increased use of incineration as a method of waste disposal.

The incineration of domestic and industrial waste releases dioxins and other chemicals into the environment.^{1–2} Crematoriums have been identified as sources of atmospheric mercury.^{3–4} Such pollutants, many of which act as endocrine disruptors, are hazardous to human health.^{5–8} However, very little is known about the public health impact of low dose, long term environmental exposure to these chemicals.^{6–9–10} Epidemiological studies have identified an increased risk of congenital anomaly and low birth weight in children born close to landfill sites, which are potential sources of this complex family of chemical pollutants.^{11–17} Higher levels of environmental pollutants—including dioxins, lead, and cadmium—have been found in the blood of children living near to waste incinerators in Belgium.⁷ Reduced testicular volume and delayed sexual maturity among children living in areas with high exposure were also reported,⁷ linking exposure to endocrine disruptors to components of the testicular dysgenesis syndrome.¹⁸ Despite concern over the health effects of emissions from incinerators⁶ and crematoriums,⁴ there is little information concerning pregnancy outcomes for mothers living in their vicinity. Identification of possible health effects of incinerators is important given the growth of incineration as a method of waste disposal¹⁹ and its widespread use for the disposal of animal carcasses during the 2001 outbreak of foot and mouth disease in the UK.²⁰

This study investigated the risk of stillbirth, neonatal death, and lethal congenital anomaly among the offspring of mothers living close to incinerators and crematoriums in Cumbria, north west England, between 1956 and 1993.

METHODS

The Cumbrian Births Database

The study area was the county currently defined as Cumbria.²¹ The Cumbrian Births Database has been described

in detail elsewhere.^{22–23} In summary, birth registration details of all 241 524 live births and 3234 stillbirths born to mothers usually resident in the study area, from the opening of the first crematorium in 1956 to 1993, were supplied from the Office for National Statistics and entered onto a computer database.^{22–23} During this period a stillbirth was defined as a fetal death occurring after 28 weeks gestation (from 1 October 1992 fetal deaths occurring after 24 weeks gestation were included, consistent with current legal definitions).^{24–25} Death registrations for the cohort, including those that occurred outside Cumbria, were supplied by the Office for National Statistics from the National Health Service Central Register (NHSCR), which was the primary source of ascertainment of deaths. NHSCR routinely records deaths of all residents of England and Wales who have ever registered with a general practitioner. However, hospital records within Cumbria and in regional referral centres outside Cumbria were searched to ascertain unregistered stillbirths and infant deaths.²⁶ All causes of stillbirth and death were coded to ICD-9. Causes of death and stillbirth were confirmed, where possible, through examination by a consultant neonatologist of details obtained from medical and/or postmortem records (the cause of about 50% of deaths were confirmed in this way). Thus, when post-mortem and/or clinical records were available, causes of stillbirth and death were validated from a number of sources and derived using a more robust method than relying on death or stillbirth certificates. Neonatal death was defined as death within the first four weeks of life.

Several outcome groups were considered: stillbirth, neonatal death, stillbirth plus neonatal death, lethal congenital anomaly (overall and by cause category). Deaths from congenital anomaly (ICD740–759) were grouped by cause, using a standard classification of infant deaths,²⁷ into the following hierarchical and mutually exclusive categories: all neural tube defects (ICD740–742), congenital heart defects

Figure 1 Location of incinerators and crematoriums in Cumbria, 1956–93.

(ICD745–747), other congenital anomalies. Neural tube defects were subdivided into: anencephalus (ICD740), spina bifida (ICD741), other central nervous system anomalies (ICD742). All other lethal congenital anomalies were grouped because of the small numbers within each ICD-9 cause category.

The mother's address on the child's birth certificate was postcoded and hence grid referenced.²⁸ The father's occupation, as recorded on the birth certificate, was assigned a social class.²⁹ Algorithms based on parents' names were used to assign birth order and identify multiple births.²³

Geographical data (see table 1)

The grid references and dates of operation of incinerators in Cumbria were ascertained from Environment Agency records. No incinerators operated before 1977, and four operated between 1977–93. The locations of all crematoriums were ascertained from specialist digests and the dates of operation were obtained. During the period 1956–1993, three crematoriums operated. Details were captured in the geographical information system Arc/Info.³⁰ Mercury represents the main pollutant from crematoriums.^{3,4} By contrast, emissions from incinerators incorporate a more complex mixture of dioxins, furans, particulates (such as chloride and sodium), heavy

metals (including lead and chromium), and volatile organic compounds (such as chloroform).³¹ Because of the differences in emissions, incinerators and crematoriums were analysed separately. Three of the four incinerators in Cumbria all dealt with materials defined as difficult by the Environment Agency,³² the other (incinerator 1) processed only inert and biodegradable material.³² Because of the historical nature of this study no detailed emissions data were available. Details of the material dealt with at each incinerator are presented in table 1. The location of all incinerators and crematoriums in Cumbria, 1956–93 is shown in figure 1.

Analysis methods

A measure of exposure of each birth to incinerators and crematoriums was computed using the distance function $1/(D+0.1)^2$ where D was the distance in km from the site and the measure was summed over all sites that were in operation at the time of birth.

Stillbirth and neonatal death rates fell substantially over the study period.³³ The cause of stillbirth was recorded on the stillbirth registration only from 1961 onwards. Hence the analysis in relation to proximity to crematoriums was stratified by time period: 1956–60, 1961–71, 1972–82, 1983–93. As incinerators in Cumbria were in operation only between 1977 and 1993, this analysis was not stratified by time period.

Multivariate logistic regression³⁴ was used to model how the risk of each outcome varied in relation to proximity to incinerators and crematoriums, adjusting for the known demographic risk factors—year of birth, social class, birth order, and multiple births—using offsets from an analysis of the effects of demographic risk factors without the exposure function. Year of birth was modelled using both quadratic and linear terms. Social class, birth order, and multiple births were treated as categorical variables (social classes I, II, IIIn, IIIm, IV, V, armed forces, and unknown, father not recorded on the birth certificate; birth order 1, 2, 3, and ≥ 4 ; multiple births, yes/no). A sensitivity analysis was carried out repeating the logistic regression, but excluding births with the greatest influence, as measured by Pregibon's influence statistic.³⁴ For incinerators the analysis was repeated for the period before any incinerators were open, 1956–76. Because multiple births may not be considered independent events, robust estimates of variance were used and significance assessed from the corresponding p value.³⁵

RESULTS

Incinerators (see table 2)

The risk of stillbirth and neonatal death was not significantly increased closer to incinerators. However, the risk of lethal congenital anomaly was significantly higher ($p < 0.01$). This significantly increased risk was restricted to heart defects and neural tube defects, specifically spina bifida. Sensitivity analysis demonstrated that these results remained significant when the most influential births were excluded. Replication of the

Table 1 Incinerators and crematoriums in Cumbria in operation during the study period, 1956–93

Site	Location	Material*	Time period of operation
Incinerator 1	Barrow in Furness	Inert, biodegradable	1977–1992
Incinerator 2	Ulverston	Hazardous, flammable, chemicals	1978–1994
Incinerator 3	Dalston	Filter material, treatment sludges, biodegradable	1979–1991
Incinerator 4	Armathwaite	Biodegradable, putrescible	1991–present
Crematorium 1	Carlisle	—	1956–present
Crematorium 2	Barrow in Furness	—	1963–present
Crematorium 3	Distington	—	1974–present

*Information from the Sitefile Digest³² and Environment Agency records.

Table 2 Continuous odds ratios (OR)† for risk of stillbirth, neonatal death, and lethal congenital anomaly in relation to proximity to incinerators, 1956–76 (before incinerators opening)‡ and 1977–93, adjusted for social class, birth order, year of birth, and multiple births

Outcome	Number of cases	OR	95% CI	p
<i>1956–76 (before incinerators opening)</i>				
Stillbirth + neonatal death	4715	0.97	0.93 to 1.01	
Stillbirth	2622	1.00	0.96 to 1.03	
Neonatal death	2093	0.92	0.84 to 1.00	
Lethal congenital anomaly‡	1583	0.94	0.86 to 1.02	
All neural tube defects‡	602	0.95	0.85 to 1.06	
Anencephalus‡	262	0.96	0.82 to 1.13	
Spina bifida‡	244	0.86	0.67 to 1.10	
Other CNS anomaly‡	96	1.02	0.97 to 1.08	
Heart defects‡	247	1.01	0.91 to 1.12	
All other anomalies‡	303	0.94	0.81 to 1.09	
<i>1977–93</i>				
Stillbirth + neonatal death	1182	1.03	0.93 to 1.13	
Stillbirth	612	1.04	0.90 to 1.19	
Neonatal death	570	1.02	0.90 to 1.14	
Lethal congenital anomaly	417	1.10	1.03 to 1.19	**
All neural tube defects	132	1.13	1.04 to 1.23	**
Anencephalus	33	1.08	0.99 to 1.18	
Spina bifida	60	1.17	1.07 to 1.28	**
Other CNS anomaly	39	0.73	0.34 to 1.56	
Heart defects	104	1.12	1.03 to 1.22	**
All other anomalies	181	0.90	0.67 to 1.22	

* $p < 0.05$, ** $p < 0.01$. †These ORs are continuous, for example, the odds of lethal congenital anomaly at a distance, D, from an incinerator compared with the odds at 3 km from incinerators is $1.10^{1/(D+0.112) - 1/3.112}$. Hence the odds ratio comparing risk at a distance of 0.5 km compared with that at 3 km (or further) is about 1.3. ‡Before incinerators opening lethal congenital anomalies were analysed only for the time period 1961–76.

analysis, using the location of incinerators for the time period *before* they were open, showed no increased risk for any outcome (table 2).

Crematoriums (see table 3)

During 1956–93 there was a significantly increased risk ($p < 0.01$) of stillbirth closer to crematoriums, reflecting a consistently increased risk from 1961 onwards. The risk of anencephalus was also significantly increased during this period ($p < 0.05$), due to a significantly increased risk in 1961–71. Although most (92%) cases of anencephalus were stillborn, the significantly increased risk of stillbirth remained after exclusion of anencephalus cases from the analysis. From 1972 onwards there was an increased risk of all other congenital anomalies, excluding neural tube defects and heart defects, with increasing proximity to crematoriums, which was significant ($p < 0.01$) for the period 1983–1993. These findings remained significant after exclusion of the most influential births.

DISCUSSION

Summary

We found a significantly increased risk of lethal congenital anomaly (specifically spina bifida and heart defects) in relation to proximity to incinerators, but not of stillbirth or neonatal death. In contrast with Elliott *et al.*,¹² who found an increased risk for certain congenital anomalies in areas where landfill sites were later opened, we found no increased risk for any outcome in areas where incinerators were subsequently opened. Hence, there was no evidence that these increased risks might be attributable to features of the environment where incinerators were located.

Around crematoriums, there was a consistently increased risk of stillbirth from 1961 onwards. There was also a significantly increased risk of anencephalus during 1961–1971, when case ascertainment was highest because this time period largely pre-dated antenatal screening for this outcome.

In the later two time periods there were very few cases of anencephalus in term pregnancies and hence statistical power to detect an effect was greatly reduced. There was a significantly increased risk of all other lethal congenital anomalies around crematoriums from 1983 onwards. This increased risk was not observed in earlier time periods despite a greater number of cases, suggesting either that a small association was obscured in earlier time periods by cases due to causes that were eliminated or reduced during 1983–93, or that the significant association in 1983–93 was a chance finding.

The significant statistical associations are different for incinerators and crematoriums. While we cannot infer a causal effect from these statistical associations, the inconsistency may be attributable to the different pollutants emitted by crematoriums and incinerators,^{14–31} or it may reflect confounding with other unmeasured risk factors, or it may be a chance finding. In addition, the time periods of operation of incinerators and crematoriums were different (1977–93 and 1956–93 respectively). Hence, while we observed a significantly increased risk of anencephalus with proximity to crematoriums during 1961–1971, we did not in the later time periods for either crematoriums or incinerators. It is unlikely that any association between proximity to incinerators or crematoriums and the risk of anencephalus would be detectable in later time periods when the number of cases was low because of prenatal screening and therapeutic termination.

Strengths and weaknesses of the study

Our study covered 38 years, allowing us to investigate a potential environmental hazard with a large cohort of 244 758 births. Changes in medical practices over time may have affected the results. Medical advances, such as improved antenatal care, which allows more fetuses to be carried to at least 28 weeks, and improved gestational dating, may have increased the number of deaths classified as stillbirths. However, other advances, such as better fetal monitoring and

Table 3 Continuous odds ratios (OR)‡ for risk of stillbirth, neonatal death, and lethal congenital anomaly in relation to proximity to crematoriums, 1956–93, by time period, adjusted for social class, birth order, year of birth, and multiple births

Outcome	Number of cases	OR	95% CI	p
1956–60				
Stillbirth + neonatal death	1508	0.95	0.74 to 1.22	
Stillbirth	887	0.85	0.60 to 1.20	
Neonatal death	621	1.08	0.77 to 1.52	
1961–71				
Stillbirth + neonatal death	2559	1.10	1.01 to 1.20	*†
Stillbirth	1413	1.19	1.09 to 1.31	**
Neonatal death	1146	0.93	0.75 to 1.15	
Congenital anomaly	906	1.10	0.95 to 1.27	
All neural tube defects	493	1.12	0.94 to 1.33	
Anencephalus	219	1.23	1.01 to 1.50	*
Spina bifida	1968	1.06	0.79 to 1.42	
Other central nervous system	76	0.65	0.33 to 1.26	
Heart defects	177	1.21	0.91 to 1.62	
All other anomalies	236	0.95	0.66 to 1.38	
1972–82				
Stillbirth + neonatal death	1212	0.98	0.87 to 1.09	
Stillbirth	602	1.04	0.93 to 1.16	
Neonatal death	610	0.89	0.72 to 1.11	
Congenital anomaly	462	0.80	0.59 to 1.09	
All neural tube defects	200	0.68	0.39 to 1.16	
Anencephalus	69	0.35	0.11 to 1.18	
Spina bifida	88	0.71	0.28 to 1.78	
Other central nervous system	43	0.97	0.64 to 1.47	
Heart defects	125	0.58	0.26 to 1.27	
All other anomalies	137	1.04	0.84 to 1.29	
1983–93				
Stillbirth + neonatal death	618	0.99	0.87 to 1.23	
Stillbirth	332	1.01	0.97 to 1.05	
Neonatal death	286	0.84	0.60 to 1.17	
Congenital anomaly	201	1.02	0.99 to 1.05	
All neural tube defects	41	0.76	0.37 to 1.58	
Anencephalus	7	0.65	0.13 to 3.19	
Spina bifida	18	1.02	0.97 to 1.08	
Other central nervous system	16	0.08	0.00 to 5.62	
Heart defects	49	0.50	0.15 to 1.62	
All other anomalies	111	1.03	1.01 to 1.06	**
1956–93				
Stillbirth + neonatal death	5897	1.02	0.99 to 1.05	
Stillbirth	3234	1.04	1.01 to 1.07	**
Neonatal death	2663	0.91	0.78 to 1.04	
Congenital anomaly (1961–93)	1569	1.02	0.96 to 1.08	
All neural tube defects	734	1.00	0.87 to 1.16	
Anencephalus	295	1.05	1.00 to 1.10	*
Spina bifida	304	0.99	0.77 to 1.27	
Other central nervous system	135	0.70	0.43 to 1.14	
Heart defects	351	1.00	0.77 to 1.31	
All other anomalies	484	1.04	1.00 to 1.07	*

* $p < 0.05$, ** $p < 0.01$. †Became non-significant when most influential births were excluded. ‡These ORs are continuous, for example the odds of anencephalus at a distance, D , from crematoriums compared with the odds at 3 km from crematoriums in 1961–71 is $1.23^{1/(D+0.1)^2 - 1/(3.1)^2}$. Hence the odds ratio comparing risk at a distance of 0.5 km compared with that at 3 km in 1961–71 is about 1.77.

improved resuscitation, may have decreased the number of stillbirths either by shifting potential stillbirths into the category of neonatal deaths or by preventing infant death. The introduction of antenatal screening and elective termination reduced the number of stillbirths and deaths attributable to congenital anomalies in recent years.³⁶ Thus the clinical characteristics of the cases in the 1950s and 1960s may be intrinsically different from those in the 1990s. However, all analyses were adjusted for year of birth, such that the risk of stillbirth, lethal congenital anomaly or neonatal death to mothers living close to incinerators or crematoriums was, in effect, compared with that of other mothers giving birth around the same time. Hence, the objectives of our study were not affected by changes in the nature of cases over time.

Because the Cumbrian Births Database recorded all birth registrations in Cumbria during the study period by date of birth and postcode of mother's residence, we had precise data on the population at risk and the location of each birth. Consequently we were able to estimate exposure and risk within a continuous model unconstrained by the availability of population statistics from other sources and we did not have to restrict our analysis to traditional geographical areal units. However, a limitation of our study was the unavailability of data on pregnancies less than 28 weeks gestation (24 weeks since 1 October 1992), which will affect the population at risk because some serious congenital anomalies might not continue to this stage of maturity, either through spontaneous abortion or termination. The inability to include such cases in

Key points

- Incinerators and crematoriums are sources of harmful chemicals (including dioxins), although little is known about the effects of long term low dose exposure.
- We investigated the effects of proximity to incinerators and crematoriums on stillbirth, neonatal death, and lethal congenital anomaly.
- We used precise details of the population at risk and the distance of each birth from all sites.
- We found an increased risk of spina bifida and heart defects in relation to proximity to incinerators and an increased risk of stillbirth, anencephalus, and other congenital anomalies in relation to proximity to crematoriums.

Recommendations

- Further work is needed to establish whether this statistical association is causal or not.

our study is likely to have resulted in a conservative estimate of the effects of proximity to sources of pollution. A further limitation of our study was the exclusion of non-lethal congenital anomalies, although we were rigorous in our ascertainment of deaths, stillbirths, and lethal congenital anomalies.²⁶ Because no data were available for non-lethal congenital anomalies these cases could not be excluded from the live birth control group. However, as the control group comprised all live births that survived over 28 days non-lethal congenital anomalies would have comprised a very low proportion of the comparison group.

We were able to incorporate exposures of each birth to putative pollution from several sites. In addition, we had demographic information for each birth and hence were able to take account of individual risk factors, such as social class, which we have shown previously to be a better predictor of stillbirth rates than community based deprivation measures such as the Townsend score.²³ Grid references for incinerators and crematoriums were supplied to an accuracy of 100 metres.

We assumed that the mother's residence during pregnancy was the same as that recorded on the birth registration. Hence migration of mothers during pregnancy may have resulted in misclassification of exposure, which would have tended to obscure any association between risk of adverse pregnancy outcome and proximity to crematoriums or incinerators.

A further limitation was that, as actual pollution levels around each site were unknown and would be impossible to ascertain retrospectively over such a long time period, we relied on a function of distance as a surrogate for potential exposure. The form of the exposure function, $1/(D+0.1)^2$, assumed that exposure increased rapidly with proximity to the sites. A potential mechanism for absorption of toxic pollutants from incinerators or crematoriums by pregnant women might involve direct inhalation of pollutants or contact through food, soil, or water contamination. We assume higher pollution levels closer to the point source and thus the distance function is a reasonable surrogate indicator that has been used in many similar studies investigating health risks around pollution sources.^{11–13 15} Although we could not consider any changes in pollution levels over time all analyses were adjusted for year of birth, so the risk of adverse pregnancy outcomes for mothers living close to crematoriums/incinerators was compared with that of mothers giving birth in the same year.

The facilities in Barrow in Furness and Dalton in Furness are located near to industrial sites defined as hazardous by the Environment Agency. Hence, there is some potential for confounding between proximity to incinerators/crematoriums and proximity to hazardous industrial sites.

There is potential for confounding between distance from incinerators and crematoriums and unmeasured risk factors,

such as diet, lifestyle, or occupational exposures. However, we adjusted for individual social class, which is likely to be related to such lifestyle factors. Hence, this study can only identify a potential statistical association between exposure to incinerators or crematoriums (modelled by a function of distance) and adverse pregnancy outcomes. We cannot establish the biological plausibility of these findings given the lack of detailed emissions data. Further studies are now required using actual pollution levels around crematoriums and incinerators to investigate the biological plausibility of our findings.

We undertook a large number of comparisons and hence it is possible that some of the significant results may be chance findings, arising through multiple significance testing. However, our results of raised risk of stillbirth, congenital heart defects, and neural tube defects were generally consistent between time periods and sensitivity analysis showed they were robust, which lessens the probability of them being chance findings. As with all geographically based studies, there was potential for confounding with lifestyle and sociodemographic risk factors that were not included in the analysis, although we were able to adjust for individual level socioeconomic status, which has not been possible in many other studies.

Comparison with other studies

Although several studies have considered pregnancy outcomes for mothers living close to hazardous waste and municipal landfill sites,^{11–15 17 37 38} there is a paucity of epidemiological data concerning pregnancy outcomes around incinerators and crematoriums with which to compare our study. Our finding of an increased risk of lethal congenital anomalies, in particular neural tube defects and congenital heart defects, in babies born close to incinerators is consistent with the results of some studies of congenital anomalies around hazardous waste and municipal landfill sites^{11–15} but not with others.^{37 38} Nevertheless, our findings need to be interpreted cautiously, as both the pollutants and exposure pathways associated with these sources differ. While incinerators are sources of a range of chemicals, including some also emitted by hazardous waste and municipal landfill sites, they also emit dioxins, heavy metals, and particulates.³¹ Furthermore, the exposure pathways from incinerators and landfill sites are different^{12 13 31}; exposure of humans to landfill pollution results from water supply contamination, groundwater run off, and atmospheric contamination from landfill gases,^{12 13} whereas pollutants from incinerators are primarily dispersed atmospherically.

Although incinerators and crematoriums in Cumbria were located in urban areas, there were so few in operation that only 10% of the Cumbrian birth cohort were born within 2 km of an incinerator or crematorium, in contrast with the finding by Elliott *et al*¹² that 80% of the population in England and Wales live within 2 km of a landfill site.

Conclusions

We found an increased risk of lethal congenital anomaly (specifically spina bifida and heart defects) in relation to proximity to incinerators and an increased risk of stillbirth and anencephalus in relation to proximity to crematoriums. In view of the scarcity of published data and our use of a distance function to represent potential exposure it is difficult to assess whether these statistical associations reflect a causal effect. Further investigations using actual pollution levels and high quality data, including lethal and non-lethal outcomes in term pregnancies and elective terminations, are required. Sufficient investment must be made in national registration systems to ensure these issues can be investigated adequately. The UK system for registration of congenital anomalies is known to be incomplete and this severely restricts its credibility.³⁹

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REFERENCES

- Schuhmacher M, Domingo JL, Xifro A, *et al*. Presence of dioxins and furans in vegetation samples collected in the neighbourhood of a municipal solid waste incinerator. *J Environ Sci Health A* 1998;**33**:195–212.
- Sakai SI, Hayakawa K, Takatsuki H, *et al*. Dioxin-like PCBs released from waste incineration and their deposition flux. *Environ Sci Technol* 2001;**35**:3601–7.
- Mills A. Mercury and crematoria chimneys. *Nature* 1990; **346**: 615.
- Maloney SR, Phillips CA, Mills A. Mercury in the hair of crematoria workers. *Lancet* 1998;**352**:1602.
- Mocarelli P, Gerthox PM, Ferrari E, *et al*. Paternal concentrations of dioxin and the sex ratio of offspring. *Lancet* 2000;**355**:1858–63.
- Mukarjee D. Health impacts of polychlorinated dibenzo-p-dioxins: a critical review. *J Air Waste Manag Assoc* 1998;**48**:157–65.
- Staessen JA, Mawrot T, Den Hond E, *et al*. Renal function, cytogenetic measurements, and sexual development in adolescents in relation to environmental pollutants: a feasibility study of biomarkers. *Lancet* 2001;**357**:1660–9.
- Crews D, Willingham E, Skipper JK. Endocrine disrupters: present issues, future directions. *Q R Biol* 2000;**75**:243–60.
- Larkin M. Public-health message about dioxins remains unclear. *Lancet* 1999;**353**: 1681.
- Watanabe S, Kitamura K, Nagahashi M. Effects of dioxins on human health: a review. *J Epidemiol* 1999;**9**:1–13.
- Vrijheid M, Dolk H, Armstrong B, *et al*. Chromosomal congenital anomalies and residence near hazardous landfill sites. *Lancet* 2002;**359**:320–2.
- Elliott P, Briggs D, Morris S, de Hoogh C, *et al*. Risk of adverse birth outcomes in populations living near landfill sites. *BMJ* 2001;**323**:363–8.
- Dolk H, Vrijheid M, Armstrong B, *et al*. Risk of congenital anomalies near hazardous-waste landfill sites in Europe: the EUROHAZCON study. *Lancet* 1998;**352**:423–7.
- Goldman LR, Paigen B, Magnant MM, *et al*. Low birthweight, prematurity and birth defects in children living near the hazardous waste site, Love Canal. *Haz Waste Haz Mat* 1985;**2**:209–23.
- Geschwind SA, Stolwijk JAJ, Bracken M, *et al*. Risk of congenital malformations associated with proximity to hazardous waste sites. *Am J Epidemiol* 1992;**135**:1197–207.
- Berry M, Bove F. Birthweight reduction associated with residence near a hazardous waste landfill. *Environ Health Perspect* 1997;**105**:856–61.
- Croen LA, Shaw GA, Sanbonmatsu L, *et al*. Maternal residential proximity to hazardous waste sites and risk for selected congenital malformations. *Epidemiology* 1997;**8**:347–54.
- Skakkebaek NE, Rajpert-De Meyts E, Main KM. Testicular dysgenesis syndrome: an increasingly common developmental disorder with environmental aspects. *Hum Reprod* 2001;**16**:972–8.
- Hu SW, Shy SM. Health effects of waste incineration: a review of epidemiologic studies. *J Air Waste Manag Assoc* 2001;**51**:1100–9.
- Hunter M. Public health concerns grow over foot and mouth outbreak. *BMJ* 2001;**322**:881.
- Local Government Act 1972, Schedule 1 page 236. London: HMSO, 1972.
- Parker L, Smith J, Dickinson HO, *et al*. The creation of a database of workers at a nuclear facility - an exercise in record linkage. *Appl Occup Environ Hyg* 1997;**12**:40–5.
- Dummer TJB, Dickinson HO, Pearce MS, *et al*. Stillbirth risk with social class and deprivation: no evidence for increasing inequality. *J Clin Epidemiol* 2000;**53**:147–55.
- Births and Deaths Registration Act 1953. London: HMSO, 1953.
- Stillbirth (Definition) Act 1992. London: HMSO, 1992.
- Dickinson HO, Parker L, Harris D, *et al*. Audit of ascertainment of deaths to children born in Cumbria, UK, 1950–89 through the NHS Central Register. *J Epidemiol Community Health* 1997;**51**:438–42.
- Alberman E, Botting B, Blatchly N, *et al*. A new hierarchical classification of causes of infant deaths in England and Wales. *Arch Dis Child* 1994;**70**:403–9.
- Royal Mail Postcode Services. *Postzon*. Portsmouth: National Postcode Centre, 1992.
- Office of Population Censuses and Surveys. *Standard Occupational Classification Volumes 1, 2, 3*. London: HMSO, 1990.
- Environmental Systems Research Institute. *Arc/Info version 7.0.3*. California: Environmental Systems Research Institute, 1995.
- Hinshaw GD, Trenholm AR. Hazardous waste incineration emissions in perspective. *Waste Management* 2001;**21**:471–5.
- Aspinwall and Company. *The Sitefile Digest: a digest of authorised waste treatment and disposal sites in Great Britain*. Shrewsbury: Aspinwall and Company, 1991.
- Botting B, ed. *The health of our children*. OPCS Decennial supplement. London: HMSO, 1995.
- Hosmer DW, Lemeshow S. *Applied logistic regression*. New York: Wiley, 1989.
- Royall RM. Model robust confidence intervals using maximum likelihood estimators. *International Statistical Review* 1986;**54**:221–6.
- Northern Region Health Authority. *Regional fetal abnormality and perinatal mortality surveys. Collaborative survey of perinatal, late neonatal and infant death in the Northern Region*. Newcastle upon Tyne: Northern Region Health Authority, 1989.
- Shaw GM, Chulman PH, Frisch JD, *et al*. Congenital malformations and birthweight reduction in areas with potential environmental contamination. *Arch Environ Health* 1992;**47**:147–53.
- Marshall EG, Gensburg UJ, Deres DA, *et al*. Maternal residential exposure to hazardous wastes and risk of central nervous system and musculoskeletal birth defects. *Arch Environ Health* 1994;**49**:416–25.
- Working Group of the Registrar General's Medical Advisory Committee. *The OPCS monitoring system for congenital malformations*. London: Office of Population Censuses and Surveys, 1995. (Occasional paper 43)