COMMITTEE ON TOXICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT

SECOND STATEMENT ON LANDFILL SITES

Background

1. In 1998, the COT was asked by the Department of Health (DH) to comment on the findings of an epidemiological study called the EUROHAZCON study [1]. This was a case-control study, which investigated the risk of congenital anomalies (birth defects) around 21 landfill sites in Europe, ten of which were in the UK. The combined results from the 21 sites suggested that women who lived within 3 kilometres (km) of a landfill site were more likely to have a fetus with a congenital anomaly than women living further away from the site. The Committee commented that the EUROHAZCON study was well conducted, but agreed with the authors that "there is a need for further investigation of whether the association of raised risk of congenital anomaly and residence near landfill sites is a causal one".

2. In 2001, the COT published a statement on a study from the Small Area Health Statistics Unit (SAHSU)[1] on health outcomes in populations living around landfill sites [2-4]. This study compared the frequency of birth outcomes and of certain cancers in populations living within 2 km of a known open or closed landfill site in Great Britain with that in the rest of the population. The COT noted that it is widely recognised that there are intrinsic limitations in ecological studies of this kind, and that there were also limitations in both the landfill data and health statistics data sets used in the study. Nevertheless the Committee considered the SAHSU study to have been well conducted. Slightly elevated relative risks were found for all congenital anomalies combined (1.01 and 1.07 around all waste sites and around hazardous waste sites, respectively), low birth weight (1.05 and 1.05, respectively) and very low birth weight (1.04 and 1.03, respectively). The Committee noted that the risk ratios for the adverse birth outcomes in this study were all close to unity, but commented that the finding of a risk ratio of 1.07 for congenital anomalies overall for populations living around hazardous waste landfill sites, whether or not it was related to the presence of the landfill sites, merited further investigation. The cancers studied - childhood and adult leukaemias, hepatobiliary cancers, and cancers of bladder and brain - were selected either to test hypotheses arising from previous studies of cancer risk around landfill sites or on the basis of the established human carcinogenicity of certain chemicals known to be present in them. The COT concluded that, taking the limitations of the study design into account, the finding of no excess risk for those living within 2 km of a landfill site for each of the cancer types studied provided a degree of reassurance.

[1] A full list of abbreviations and acronyms is given at the end of the statement.

3. The Committee was also informed that a programme of research and reviews was underway on congenital anomalies and landfill sites, and that this included a project to measure concentrations of chemicals, common air pollutants and biohazards at the boundaries of landfill sites, and to assess exposures of people living nearby. Further, SAHSU proposed that it would be possible to investigate whether there are individual sites (or a subset of sites) which significantly affect the health of the local population. This could be done by detailed mapping and statistical analysis of existing data to provide an indication of any systematic variation in rates and to analyse any resulting variations in relation to possible explanatory variables (e.g. landfill characteristics, geology, other exposure sources, deprivation). The Committee agreed that this could be a useful way forward but noted that the value of further analyses of existing datasets might be limited by the known problems of some of these datasets [3]. Both the exposure study and further SAHSU studies have now been completed. We were asked to comment on both the SAHSU studies and on the results of the exposure study in respect of the levels of chemicals detected[2]. We reviewed these during the period 2007 to 2009 and our views are given below.

Exposure Assessment

Preliminary work on exposure assessment

4. We were informed that, as a result of the findings of the EUROHAZCON study, a monitoring programme was initiated for substances potentially associated with congenital anomalies. Early in 2001, the Environment Agency (EA) began an intensive monitoring campaign at a single landfill site [5]. A mobile monitoring station was set up and used to monitor methane, oxides of nitrogen (NOx), particulates (PM_{10}) and hydrogen sulphide, together with the trace pollutants benzene and chloroethene (also known as vinyl chloride). Monitoring was carried out during two periods - an initial twenty days from January to February, followed by 88 days between July and October. The results showed that:

- methane, PM_{10}, NOx, benzene and hydrogen sulphide were all detectable at the downwind boundary of the landfill;
- NOx levels exceeded the annual average standard for the protection of vegetation and ecosystems from the National Air Quality Standard (NAQS) (2000);
- concentrations of chloroethene were all below the detection limit of 10 ppb.

However, as these substances are often present in air at similar levels to those measured and as monitoring only took place downwind, it was not possible to conclude how much the landfill site was contributing to the levels that were monitored.

5. The EA also commissioned a review of information on the trace components in landfill gas. The chemicals from these analyses were categorised and prioritised according to their toxicity and odour potential [6]. As a follow-on to this work, the EA

[2] The study also measured a number of common air pollutants (e.g. PM_{10}, NOx) and microbiological hazards (see Annex 1). It is outside the remit of the COT to advise on the health significance of these.

commissioned sampling and analysis of landfill gas trace components at six different types of landfill site [6].

6. A review was commissioned by the DH to assess the potential for developmental toxicity of chemicals known or expected to be released from landfill sites [7]. This classified a number of volatile organic compounds (VOCs) into four groups with regard to developmental toxicity: chemicals of possible interest, chemicals of less likely interest, chemicals of no/unlikely interest, and those with insufficient data for classification (Table 1). The information from these two reviews was used to inform further monitoring work.

Table 1: Classification of chemicals according to their developmental toxicity (after Sullivan et al, 2001) [7]

Classification	Substance
Chemicals of possible interest	benzene, 1,3-butadiene, carbon disulphide, chloroform, 1,2-dichloroethene, ethylbenzene, formaldehyde[3], chloromethane, tetrachloroethylene, trichloroethene, chloroethene
Chemicals of less likely interest	alpha-terpinene, dichlorobenzene, 2-ethyl-1-hexanol, hydrogen sulphide, 2-butanone, toluene, xylenes
Chemicals of no/unlikely interest	acetone, 2-butanol, carbon tetrachloride, dichloromethane, ethanol, limonene, 1-propanol, styrene, vinyl acetate
Chemicals with insufficient data for classification	1,1-dichloroethane, dichlorofluoromethane, ethanethiol, methanethiol, 2-methyl furan, nitromethane

Landfill sites

7. Landfill sites taking biodegradable waste are complex microbiological, chemical and biochemical reactors, wherein waste continues to degrade, often for more than 100 years. The degradation process is mainly anaerobic and produces a gas which consists mainly of methane and carbon dioxide. Rainfall which infiltrates the waste will dissolve substances to form leachate, a polluting liquid which collects at the base of the site and which contains high levels of ammonia and dissolved organic carbon [8].

8. Both leachate and landfill gas have to be controlled by the operator under permits issued by the EA. Leachate is typically collected and treated biologically before being discharged to sewer or, occasionally, to surface waters. Landfill gas is pumped from the waste mass and combusted in flares or in engines which power electricity generators.

[3] Note: IUPAC name for formaldehyde is “methanal”; however, as the term “formaldehyde” is in common usage, this was used in the report.

9. The main sources of emissions from landfill sites are as follows:

- the waste materials as they are brought onto site, normally in heavy goods vehicles;
- emissions from this transport and any heavy plant used on site;
- waste blown by the wind as it is tipped or deposited at the landfill site;
- dust generated from the surface of the landfill and when waste is tipped or unloaded;
- the waste materials which have previously been deposited in the landfill site;
- any gas generated as the waste breaks down, which is not collected and treated;
- any plant used to burn landfill gas, including gas flares or engines;
- any leachate produced as the waste breaks down;
- the discharges from any processes used to treat the leachate.

We were advised that, as areas of the site are filled, waste is deposited in new areas and, therefore, the location and nature of these sources vary throughout the lifetime of the site, which makes a representative survey of emissions extremely difficult.

2002-2005 landfill study

10. In 2008, we were asked to consider the toxicological aspects of a draft report of a project commissioned by the EA which monitored concentrations of chemicals, common air pollutants and biohazards over two years at the boundaries of two municipal waste landfill sites (see [9], [10] for copy of final report). We were informed that the two landfill sites (termed Sites A and B) were typical of the population of currently operated landfill sites in England and Wales and were selected on the basis of pre-defined criteria i.e. of a reasonable size, with landfill gas controls, near to population, with groundwater within 10 metres of the site base and surface water within 50 metres of the site boundary. All the potential pathways for exposure of the local population to emissions from landfill sites were identified and prioritised as part of a screening risk assessment. In the light of this, a detailed study of airborne levels of substances potentially emitted from the landfill sites was carried out.

11. The study provided the following:

- data on the concentrations of the principal chemicals expected to be emitted from the two landfill sites in air samples at the boundaries;
- health-based reference concentrations for these substances in air *(see paragraph 15)*;

- a comparison of the concentrations of the substances found against these reference concentrations and an assessment of how frequently they were found;
- an assessment of the risks to health posed by possible releases of waterborne pollutants;
- an estimate of exposure to polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans by both direct exposure from air and indirect exposure from modelled uptake into produce grown around the landfill sites.

12. All the known potential sources of pollution at the landfill site and their potential pathways to humans were considered. The risks associated with each possible source of pollution were screened and the sources which were most significant were subject to detailed monitoring and assessment. The pathways of greatest potential concern were identified as groundwater and air (both for landfill gas and for particulates). A groundwater risk assessment was carried out, but the authors considered that this showed no significant risk to the surrounding populations. Therefore, the main focus of the monitoring work was airborne emissions which were viewed as having the highest potential for exposure of the population.

13. The study used both continuous and discrete atmospheric sampling. The pollutants studied were based on the prioritisation from the three studies reported in paragraphs 4 to 6 above, which categorised trace components in landfill gas according to their potential for developmental toxicity and their concentrations. Both sites were monitored extensively for some 22 months over the study period (2002 and 2003). This included continuous monitoring for NOx, PM_{10} and total VOCs at the northeast boundary of both sites throughout the study. Continuous measurements were also carried out for these determinands for shorter periods at the southwest boundary of each site. These determinands were chosen as indicators of combustion and fugitive emissions. During the shorter survey periods, continuous measurements were also made of sulphur dioxide and hydrogen sulphide. In addition, over 1200 site boundary measurements were made of substances of specific concern to this Committee. The concentrations measured at the boundaries were highly variable, possibly depending on the time of day, meteorology and longer-term changes in the location and nature of operations at the sites.

14. Two problems were identified which affected the interpretation of the study results. Firstly, the prevailing wind direction at one site was different from that expected, which limited the amount of time the monitoring points were directly upwind and downwind from the site. To address this, the authors of the study estimated the significance of the potential health effects using the maximum concentrations measured at the site boundary over an appropriate sampling period. Secondly, the concentrations were very low, often at or below the limit of detection.

15. The study also sought to establish health-based reference concentrations for substances in air at which it was expected that there would be either no risk of health

effects over a lifetime or, in the case of non-threshold substances[4], a minimal risk to health. These were termed "Health Criteria Values (HCVs)". In this statement we have referred to them as "project-specific HCVs" to distinguish them from the HCVs published by the EA for use in the risk assessment of contaminants in soil [11]. The project-specific HCVs were applied in relation to the measured concentrations of these substances at the site boundaries.

Subsequent study

16. Following an initial review of the 2002-2005 study results, we requested further measurements of a number of the substances measured in the 2002-2005 study. The aim was to generate improved data for this subset of substances. These measurements were carried out during 2009 at two further typical landfill sites (termed Sites C and D) which were selected to be typical of landfill sites accepting household waste, and two background locations (general warehousing bordering a river and a non-industrial business park) [12]. Fewer measurements were taken at these locations, but the measurements were more closely targeted and, in some cases, involved greater analytical sensitivity. We have considered the results of this subsequent survey alongside the results of the 2002-2005 study in our statement.

Summary and discussion of results of both studies

Initial review of results

17. The main study considered over 60 chemicals or chemical groups (see Annex 1 to this statement). The maximum concentrations for each chemical at both sites were compared to the project-specific HCV. Twenty-three of the substances were not considered further because the maximum concentrations at both sites were below 1% of the HCV (see Annex 1).

18. The remaining substances were prioritised on the basis of the ratios of the concentration to the project-specific HCV. This is a conservative approach, because the concentrations to which members of the public are likely to be exposed are lower than those measured at the boundary of a landfill site. We considered in detail the following substances whose average measured boundary concentrations at one or both sites were at, or above, 75% of the project-specific HCV.

- Arsine
- Chromium
- 1,2-Dichloroethane
- Dimethyl disulphide
- Dimethyl sulphide
- Formaldehyde
- Methyl mercaptan (methane thiol)
- Polycyclic aromatic hydrocarbons

[4] Most toxicological effects are expected to exhibit a dose threshold i.e.there is a dose below which the adverse effect does not occur. However, for some chemicals, there is no identifiable dose threshold and it is assumed that a toxic effect may occur at any dose. These are referred to as "non-threshold" chemicals. Non-threshold toxicity most notably occurs in the case of chemicals which damage DNA i.e.genotoxic chemicals.

- Stibine
- Styrene
- Toluene

19. A limited review was carried out on the project-specific HCVs for the other chemicals by comparing them to the Health and Safety Executive Workplace Exposure Limit (WEL) for that chemical, adjusted to 24 hour, 7 days per week exposure, and divided by an additional uncertainty factor of 10 to allow for wider inter-individual variation in susceptibility in the general population than in the workforce [13]. Where the value derived from the WEL was similar to, or higher than, the project-specific HCV, no further action was taken, as this indicated that the HCV was likely to be conservative. In cases where the value derived from the WEL was lower than the project-specific HCV, the concentrations measured in the monitoring study were checked to see if they exceeded, or were close to, the value derived from the WEL and, if so, the derivation of the project-specific HCV was examined in more detail. This was the case only for nickel where the maximum concentration at Site B slightly exceeded the value derived from the WEL. A closer evaluation of the project-specific HCV for nickel, which was based on a recommendation by the EU Scientific Committee on Toxicity, Ecotoxicity and the Environment, indicated that it was sufficiently precautionary [14]. For 4 chemicals there was no project-specific HCV and no WEL. These were: 2-methyl furan, acenaphthylene, benzo(ghi)perylene and phenanthrene. These are discussed in paragraphs 94 to 95 below. Also, we noted that, although the median concentrations of chloroethene at both Sites A and B were well below the project-specific HCV of 1 ug m^{-3}, the maximum concentration found at Site B was 4.9 ug m^{-3}. We therefore included this compound in our more detailed review.

20. For all other chemicals, the concentrations measured at the boundaries of the sites were not considered to be a cause for concern.

21. Finally, we were asked to consider the health significance of estimated exposures to polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans, both via inhalation and via consumption following deposition on, or incorporation into, locally produced food.

Detailed review

Introduction

22. When reporting below the concentrations of chemicals measured at the boundaries of the landfill sites, we have presented the 50^{th} percentile values of the measurements made for each chemical at each site, together with the maximum concentration detected. Two values are given for the 50^{th} percentile: firstly, the value calculated by assuming that all concentrations below the detection limit were zero and, secondly, the value calculated by assuming that all concentrations below the detection limit were present at the detection limit. This is explained in more detail in Annex 2 to this statement.

23. Also, for all chemicals examined in detail, we considered whether the project-specific HCV established in the main study report (see paragraph 15) was

reasonable given currently available data on the toxicity of the chemical. In some cases, we concluded that this was not the case and derived our own health-based reference concentration. Where this was done, the derivation of the revised health-based reference concentration is described below.

Arsine

24. The concentrations of arsine measured in the main study and the subsequent study are summarised in Table 2 below.

Table 2: Arsine concentrations in main and subsequent monitoring studies

	50th percentile (ng m^{-3})		**Maximum detected concentration (ng m^{-3})**	**Detection limit (ng m^{-3})**	**No. Detects/ No. Measure-ments**
	Assuming all non-detects were zero	Assuming all non-detects were present at detection limit			
Main study					
- Site A	0	440	N/A530	300-700	0/16
- Site B	0	630		<370-1100	2/8 [a]
2009 Study					
- Site C	0	10	N/A	10	0/6
- Site D	0	2	N/A	2	0/6

a: The second 'detected' level was 370 ng m^{-3}
N/A: not applicable. Not detected in any sample taken at this site

25. Arsine is a colourless, non-irritant gas with a mild, garlic-like odour. Exposure to arsine is most likely to occur in an occupational setting. The project-specific HCV for arsine was 7 ng m^{-3}. This had been based on the assumption that arsine is completely metabolised in humans to arsenic and, therefore, that it was appropriate to apply the World Health Organisation (WHO) Air Quality Guideline for Europe for arsenic of 6.6 ng m^{-3} to arsine. Long-term exposure to this concentration of arsenic had been estimated from occupational studies to carry a 1 in 10^5 lifetime risk of lung cancer [15]. It should be noted that the UK Expert Panel on Air Quality Standards (EPAQS) has recently recommended an air quality guideline for total inorganic arsenic for use in regulating emissions of arsenic from industrial plant. This is 3 ng m^{-3} in the PM_{10} size fraction, as an annual mean [16].

26. Limited information is available on the metabolism of arsine. There is some qualitative evidence of conversion to arsenic in experimental animals but there are no quantitative estimates of the extent of conversion [17]. The authors of the main study [18] assumed 100% conversion as a worst case, but we consider that this may be excessively conservative.

27. No genotoxicity studies have been reported for arsine; such studies would be difficult in view of its high volatility and low water solubility [19]. Arsine is excreted in the urine as dimethylarsinate, monomethylarsonate, trivalent arsenic, and, to a lesser extent, pentavalent arsenic, which are also metabolites of other arsenic

compounds. Thus it may well have carcinogenic potential [19]. There are no adequate reproductive or chronic toxicity studies of arsine in humans or animals. It is highly acutely toxic and health-based guideline values are usually recommended on the basis of its acute effects. A 2001 WHO Concise International Chemical Assessment Document (CICAD)[5] recommended a guideline value of 50 ng m^{-3}, which was based on the No Observed Adverse Effect Concentration (NOAEC) for haemolysis in short-term animal studies [17]. However, we do not consider that it would be appropriate to use this to assess the risks from chronic exposure to arsine.

28. We agree that concentrations of arsine at or below 50 ng m^{-3} are unlikely to give rise to acute toxic effects. However, given the lack of information on metabolism, genotoxicity or carcinogenicity of arsine, it is not possible for us to recommend a reference concentration of arsine in air which will protect against chronic health effects.

Discussion of results for arsine

29. In the main study, arsine was detected on two occasions at Site B at concentrations of 530 and 370 ng m^{-3}. These results are of some concern but the values were close to the detection limits of the analytical method used (500 to 1100 ng m^{-3}) and, therefore, it is not clear how reliable they are. No arsine was detected at Site A, but the detection limit was again high (400 to 700 ng m^{-3}). In the subsequent study, no arsine was detected above detection limits of 10 ng m^{-3} (Site C) or 2 ng m^{-3} (Site D), which is reassuring. It is difficult to provide an informative assessment from these limited data. We might be able to provide further advice if more sensitive sampling or analytical methods were to be developed and further monitoring data obtained from the boundaries of landfill sites. Also, there is a need for quantitative information on the conversion of arsine to arsenic in an appropriate experimental species.

Chloroethene (vinyl chloride)

30 . The concentrations of chloroethene measured in the main study and the subsequent study are summarised in Table 3 below.

Table 3: Chloroethene concentrations in main and subsequent monitoring studies

	50th percentile (µg m^{-3})		**Maximum detected concentration (µg m^{-3})**	**Detection limit (µg m^{-3})**	**No. Detects/ No. Measure-ments**
	Assuming all non-detects were zero	Assuming all non-detects were present at detection limit			
Main study					
- Site A	0	0.010	0.46	0.01 – 5	5/43
- Site B	0	0.015	4.9	0.01 – 7	8/48

[5] CICADS are written as part of the WHO/ILO/UNEP International Programme on Chemical Safety.

2009 study **- Site C** **- Site D**	 0 0	 0.04 0.06	 N/AN/A	 0.03 – 0.07 0.06	 0/6 0/6

N/A: not applicable. Not detected in any sample taken at this site

31. Chloroethene is a gas which is used in the plastics industry for the production of polyvinyl chloride (PVC). It has also been reported to be produced from the degradation of other chlorinated compounds in landfill sites [20]. Epidemiological studies have revealed a strong association between occupational exposure to chloroethene by inhalation and angiosarcoma of the liver, and it is classified by the International Agency for Research on Cancer (IARC) as carcinogenic to humans [21].

32. The project-specific HCV for chloroethene was 1 µg m^{-3}, which was the concentration estimated from occupational studies to present a 1 in 10^6 cancer risk, cited in the WHO Air Quality Guidelines for Europe [15]. We agree that this is an appropriate reference concentration against which to assess the risk to public health from airborne concentrations of chloroethene.

33. We note that chloroethene was classified by Sullivan *et al* (2001) as a "chemical of possible interest" with regard to its developmental toxicity (see Table 1) [7]. Sullivan *et al* (2001) concluded that chloroethene was not teratogenic in laboratory animals by inhalation and is embryotoxic only at maternally toxic doses. The evidence concerning human occupational or environmental exposure to chloroethene and malformations or spontaneous abortions was considered to be inconclusive. An inhalation NOAEC for embryotoxicity of 130 mg m^{-3}, administered for 7 hours per day, was identified in mice. This is well above the concentrations detected in the monitoring studies.

34. Background concentrations of chloroethene in air are reported to be usually less than 3 µg m^{-3} although higher concentrations have been observed near chloroethene production sites and waste disposal sites (up to 100 µg m^{-3} and 1000 µg m^{-3}, respectively) [22]. The background level in the 2009 monitoring survey was below the detection limit of 0.06 µg m^{-3}.

Discussion of results for chloroethene

35. The concentrations measured in the main monitoring study do not give rise to concern in relation to either angiosarcoma of the liver or developmental effects. Occasional concentrations above the HCV, while undesirable, are likely to be associated with a negligible risk of carcinogenicity. Chloroethene was not detected in the further monitoring exercise.

<u>Chromium</u>

36. The concentrations of chromium measured in the main study and the subsequent study are summarised in Table 4 below. In the main study, total chromium was measured but, in the subsequent study, concentrations of hexavalent chromium (see below) alone were measured.

37. Chromium is a metal which commonly exists either in the trivalent form [chromium (III)] or the hexavalent form [chromium (VI)]. The toxicity of chromium varies depending on its valency state: hexavalent chromium is more toxic than trivalent chromium, which is an essential trace element. Hexavalent chromium and its compounds are oxidizing agents capable of directly inducing tissue damage. Epidemiological studies have found an association between exposure to hexavalent chromium and lung cancer and IARC has classified chromium (VI) as carcinogenic to humans [23].

Table 4: Chromium concentrations in main and subsequent monitoring studies

	50th percentile (ng m^{-3})		**Maximum detected concentration (ng m^{-3})**	**Detection limit (ng m^{-3})**	**No. Detects/ No. Measure-ments**
	Assuming all non-detects were zero	Assuming all non-detects were present at detection limit			
Main study					
- Site A	0.4 (total Cr)	1.0 (total Cr)	3.9 (total Cr)	0.1 – 1.0 (total Cr)	19/32
- Site B	1.0 (total Cr)	1.0 (total Cr)	28 (total Cr)	0.1 – 1.0 (total Cr)	28/32
2009 Study					
- Site C[a]	0 (CrVI)	1000 (CrVI)	N/A	1000 (CrVI)	0/6
- Site D	0 (Cr VI)	6 (Cr VI)	N/A	6 (CrVI)	0/3

a: Results for Site D should be viewed with caution because the methodology has not been validated
N/A: not applicable. Not detected in any sample taken at this site

38. The project-specific HCV was 2.5 ng m^{-3} total chromium. This was equivalent to the guideline value for inhalation of chromium from soil recommended by the UK Government in 2002 [24]. This in turn was the concentration of Cr(VI) estimated from occupational studies to present a 1 in 10^4 cancer risk, cited in the WHO Air Quality Guidelines for Europe [15]. The choice of a risk estimate of 1 in 10^4 rather than 1 in 10^5 for chromium in soil is not explained but probably reflects the fact that the estimate is for chromium (VI), not total chromium, and therefore the risk from total chromium in soil will be lower than for the same quantity of chromium (VI). We were informed that there is little readily available information on the speciation of chromium at landfill sites, but the reducing environment, together with the presence of readily oxidisable organic compounds, would be expected to be more conducive to its presence as chromium (III) than chromium (VI). We agree that 2.5 ng m^{-3} is an appropriate reference concentration for total chromium. We note that EPAQS has recently published a new guideline for chromium in ambient air of 0.2 ng m^{-3} chromium as chromium (VI) for use in regulating emissions from industrial plant. This was derived from the Lowest Observed Adverse Effect Concentration (LOAEC) for mortality from lung cancer in occupational studies [16].

39. There are no studies in the literature on developmental effects in humans or animals after inhalation exposure to chromium or its compounds [25]. There is some evidence of developmental effects in animal studies in which chromium (VI) was

administered by the oral route but the effects were observed at relatively high doses (≥35 mg kg bw^{-1} day^{-1}) and were usually associated with maternal toxicity [25].

40. Background concentrations of particulate total chromium in air in the UK have been reported to be 0.2 to 0.7 ng m^{-3} in rural areas and 4.1 to 17.2 ng m^{-3} in urban areas [26]. During the 2009 survey, the background concentration at an urban location was reported to be less than 6 ng m^{-3}.

Discussion of results for chromium

41. In the main study, the maximum concentrations of total chromium at both sites A and B exceeded the HCV of 2.5 ng m^{-3} by up to 11-fold but the 50th percentile concentrations were well below the HCV and below current urban background concentrations of chromium. It is the long-term average concentration of chromium which is of most concern when considering the risk of lung cancer. It is not possible to draw reliable conclusions about potential health risks because of lack of information on speciation and inadequacies of available measurement techniques. However, it is relevant to note that EPAQS has commented that "at current upper UK urban levels of chromium of around 15 ng m^{-3} containing an estimated 4 ng m^{-3} of Cr (VI), the increased risk of lung cancer would amount to a little under 1 in 10,000 which is comparable to the rate of death from lung cancer in non-smokers derived from a 20 year follow-up of male British doctors" [16]. We note that the detection limits for chromium (VI) at sites C and D were 1000 ng m^{-3} and 6 ng m^{-3}, respectively, which are higher than the new EPAQS guideline of 0.2 ng chromium (VI) m^{-3}. Therefore, it is not known whether the concentrations at the boundaries of these sites exceeded the guideline or not. We recommend that, should techniques be developed to measure chromium (VI) in air at lower detection limits, it would be appropriate to use these in future monitoring.

1,2-dichloroethane

42. The concentrations of 1,2-dichloroethane measured in the main study and the subsequent study are summarised in Table 5 below.

Table 5: Concentrations of 1,2-dichloroethane in main and subsequent monitoring studies

	50th percentile (µg m^{-3})		**Maximum detected concentration (µg m^{-3})**	**Detection limit (µg m^{-3})**	**No. Detects/ No. Measure-ments**
	Assuming all non-detects were zero	Assuming all non-detects were present at detection limit			
Main study					
- Site A	0	0.077	2.4	0.01 – 1	17/55
- Site B	0.045	0.050	1.5	0.01 – 0.6	29/36
2009 study					
- Site C	0	0.02	0.13	0.02 – 0.03	1/6
- Site D	0	0.06	0.2	0.06	1/6

43. 1,2-dichloroethane is a volatile, synthetic chemical with no known natural sources [27]. It has demonstrated genotoxic potential both *in vitro* and *in vivo* [28]. The project-specific HCV for 1,2-dichloroethane was 0.36 µg m^{-3}. This was derived from a 1998 assessment by an international expert group of a 1978 oral carcinogenicity study in rats and mice which, although of poor quality, indicated dose-related increases in tumours at multiple sites in both species [27, 29].

44. We note that a good quality inhalation carcinogenicity study on 1,2-dichloroethane was published recently by Nagano et al [30]. In this study, groups of F344 rats and BDF1 mice were exposed to 1,2-dichloroethane vapour or 'clean air' (controls) for 6 hours per day, 5 days per week for 104 weeks. These exposures are equivalent to 7.1, 28.6 and 114.3 mg m^{-3} in rats and 7.1, 21.4 and 64.3 mg m^{-3} in mice when averaged over 24 hours per day, 7 days per week (referred to as Time Weighted Average (TWA) exposures). Treatment-related increases in cancer incidence were seen at several sites in both species. We carried out Benchmark Concentration (BMC) calculations for those neoplastic endpoints which showed a statistically significant dose-related trend, for a Benchmark Response of 10%, using the US EPA BMDS 2.0 software. The lowest $BMCL_{10}$ [6] values were 24.05 mg m^{-3} for subcutaneous fibroma in male rats and 12.95 mg m^{-3} for combined mammary adenoma, fibroadenoma and adenocarcinoma in female rats (all values are TWA doses). We then divided the lowest $BMCL_{10}$ value by the concentrations of 1,2-dichloroethane measured at the landfill sites to give the Margin of Exposure (MOE). The results are given in Table 6 below.

Table 6: Margins of Exposure for concentrations of 1,2-dichloroethane

	For 50th percentile concentration assuming all non- detects at DL	**For maximum concentrations**
Main study		
- Site A	170,000	5,400
- Site B	260,000	8,600
2009 study		
- Site C	650,000	100,000
- Site D	220,000	65,000

45. 1,2-dichloroethane was not reviewed by Sullivan *et al* (2001). However, the weight-of-evidence from studies in animals indicates that no adverse reproductive or developmental effects would be expected at the exposures reported in the monitoring studies [28].

46. Few data are available on background concentrations of 1,2-dichloroethane. The background levels measured during the further monitoring exercise were close to or below the detection limits of 0.03 and 0.06 ug m^{-3}.

[6] $BMCL_{10}$: lower 95% confidence limit of the benchmark concentration for a 10% response.

Discussion of results for 1,2-dichloroethane

47. To assess the significance of the MOEs in Table 5, we used the banding system recommended by the Committee on Carcinogenicity of Chemicals in Food, Consumer Products and the Environment (COC) to assist with the risk management and risk communication of the health significance of genotoxic contaminants in food and the environment [31]. This is given in Table 7 below. Using this system, the 50th percentile concentrations measured in both the main and subsequent studies are unlikely to be a concern. The maximum concentrations measured in the main study may be a concern if present on a continuous basis, but the risk of carcinogenic effects following exposure to occasional concentrations of this magnitude is highly unlikely to be a concern.

Table 7: MOE banding approach agreed by COC

MOE Band	Interpretation
<10,000	May be a concern
10,000-1,000,000	Unlikely to be a concern
>1,000,000	Highly unlikely to be a concern

Dimethyl sulphide and dimethyl disulphide

48. The concentrations of dimethyl sulphide (DMS) and dimethyl disulphide (DMDS) measured in the main study and in the subsequent study are summarised in Table 8 below.

49. DMS and DMDS are similar substances both chemically and toxicologically and have, therefore, been considered together. They are foul-smelling compounds, with odour thresholds of 6.5 and 15.2 µg m^{-3}, respectively. There are limited toxicity data on these compounds. The project-specific HCV for both compounds was 5 µg m^{-3}. We note that this was derived from a subchronic inhalation study on DMS in rats quoted in Opydke (1979) [32]. A good quality subchronic inhalation study has recently been published in which rats were exposed to DMDS vapour by whole-body exposure at TWA concentrations of 3.5, 17.2 or 86.0 mg m^{-3} [33]. The TWA NOAEC was 3.5 mg m^{-3}. Changes in biochemical parameters and organ weights were seen above this level. An uncertainty factor (UF) of 125 (2.5 for interspecies toxicodynamics[7], 10 for intraspecies differences and 5 for the limited database) applied to the TWA NOAEC gave a reference concentration in air of 28 µg m^{-3}. We used this to assess the risk of the combined concentrations of both substances.

[7] The usual uncertainty factor of 4 for interspecies toxicokinetics was not applied. Rats have a higher respiratory rate than humans and are exposed to 4 times more of a compound for a given air concentration (European Chemicals Agency. Guidance on information requirements and chemical safety assessment: Chapter R.8. May 2008).

Table 8: Concentrations of DMS and DMDS in main and subsequent monitoring studies

	50th percentile (µg m^{-3})		**Maximum detected concentration (µg m^{-3})**	**Detection limit (µg m^{-3})**	**No. Detects/ No. Measure-ments**
	Assuming all non-detects were zero	Assuming all non-detects were present at detection limit			
DMS Main study					
- Site A	0	2	375	0.07 – 5	21/44
- Site B	0	2.7	59	0.07 – 7	21/48
DMS 2009 study					
- Site C	0	0.21	N/AN/A	0.2 – 0.3	0/6
- Site D	0	0.06		0.06	0/6
DMDS Main study					
- Site A	0.04	0.75	56	0.07 – 5	22/44
- Site B	0	1.7	16	0.07 – 7	21/48
DMDS 2009 Study					
- Site C	0	0.02	N/AN/A	0.02 – 0.03	0/6
- Site D	0	0.06		0.06	0/6

N/A: not applicable. Not detected in any sample taken at these sites

50. We could find no reproductive or developmental toxicity data on DMS or DMDS nor any reliable data on background concentrations of these substances in the UK, although they are both likely to be present as a result of industrial and biological processes.

Discussion of results for dimethyl sulphide and dimethyl disulphide

51. In the main study, the 50th percentile concentrations were well below the reference concentration of 28 ug m^{-3}. Therefore, on the basis of the limited data available, we would anticipate no adverse health effects from chronic exposure to DMS or DMDS. The combined maximum measured concentrations of DMS and DMDS at both sites A and B exceeded the reference concentration but we consider it unlikely that these concentrations would pose an acute risk to those on or near the site in view of the minor effects seen above the NOAEC in the recent subchronic inhalation study. Moreover, the maximum concentrations exceeded the odour thresholds, which is likely to deter individuals from staying in the vicinity. In the subsequent monitoring exercise, which achieved detection limits of 0.02 to 0.3 ug m^{-3}, no DMS or DMDS was detected.

Formaldehyde (methanal)

52. The concentrations of formaldehyde measured in the main study and the subsequent monitoring exercise are summarised in Table 9 below.

53. Formaldehyde is widely present in most living systems and in the environment. Exposure also arises from vehicle emissions, from building and household materials, from tobacco smoke and in some occupational activities. Most formaldehyde released to the environment is rapidly degraded.

Table 9: Concentrations of formaldehyde found in main and subsequent monitoring studies

	50th percentile (µg m^{-3})		**Maximum detected concentration (µg m^{-3})**	**Detection limit (µg m^{-3})**	**No. Detects/ No. Measure-ments**
	Assuming all non-detects were zero	Assuming all non-detects were present at detection limit			
Main Study - Site A - Site B	 0 64	 38 64	 213 487	 5 – 47 10 – 21	 3/8 6/8
2009 Study - Site C - Site D	 1.6 38	 1.6 38	 4.6 46	 1.2 4	 5/6 3/3

54. Formaldehyde is an irritant and can cause nose and throat irritation and is a weak allergen [34]. Epidemiological studies have found an association between exposure to formaldehyde and cancer of the nasopharynx. IARC has classified it as carcinogenic to humans [35]. The Committee on Mutagenicity of Chemicals in Food, Consumer Products and the Environment (COM) advised in 2007 that there was no convincing evidence from *in vivo* mutagenicity studies in experimental animals, nor from biomonitoring studies of genotoxicity in workers exposed to formaldehyde, for a direct *in vivo* systemic mutagenic effect of inhaled formaldehyde [36].

55. The project-specific HCV of 10 µg m^{-3} was derived from a US Agency for Toxic Substances and Disease Registry (ASTDR) chronic duration inhalation exposure Minimal Risk Level for mild damage to the nasal epithelium following occupational exposure to formaldehyde [34]. However, according to the WHO Air Quality Guidelines for Europe, damage to the nasal mucosa in workers exposed to formaldehyde may have been caused by concomitant exposures to other substances [15]. Therefore, we consider that it would be more appropriate to use the WHO Air Quality Guideline for formaldehyde of 100 µg m^{-3} (as a 30-minute average) to assess the significance of the concentrations found in the monitoring studies. This guideline is set on the basis that, provided the respiratory tract tissue in humans is not repeatedly damaged, exposure to low, noncytoxic concentrations of formaldehyde will be associated with a negligible cancer risk. This is considered consistent with

epidemiological findings. Therefore, the guideline is based on the lowest reported threshold for nose and throat irritation in the general population i.e. 100 µg m^{-3}.

56. Formaldehyde was classified by Sullivan *et al* (2001) as a "chemical of possible interest" with regard to its developmental toxicity (see Table 1) [7]. The authors concluded that "while animal studies suggest absence of teratogenic potential even at very high exposures, there is evidence of effects on fetal weight at high exposures" (24 mg m^{-3} and above). They further concluded "in humans, there is consistent evidence of an increase in spontaneous abortion rates of up to 2-fold and limited evidence of reduced fertility at airborne exposure levels at or just above those currently permitted in the workplace (1.2 mg m^{-3})".

57. Background concentrations of formaldehyde have been reported to be from 1 to 20 µg m^{-3} in ambient air, 30 to 60 µg m^{-3} in a conventional home [15] and 4 to 800 µg m^{-3} in mobile homes [37]. The background levels measured as part of the 2009 monitoring exercise were 1.2 to 4 µg m^{-3}.

Discussion of results for formaldehyde

58. In the main study, the 50th percentile concentrations were below the WHO Air Quality Guideline for Europe although maximum concentrations exceeded it. We do not consider that these results raise any concerns about chronic health effects or adverse developmental effects, but some individuals close to landfill sites might experience irritant effects at the maximum concentrations reported. In the subsequent study, both 50th percentile and maximum concentrations were below the guideline, which is reassuring.

Methyl mercaptan

59. The concentrations of methyl mercaptan measured in the main study and the subsequent monitoring exercise are summarised in Table 10 below.

Table 10: Concentrations of methyl mercaptan found in main and subsequent monitoring studies

	50th percentile (µg m^{-3})		**Maximum detected concentration (µg m^{-3})**	**Detection limit (µg m^{-3})**	**No. Detects/ No. Measure-ments**
	Assuming all non-detects were zero	Assuming all non-detects were present at detection limit			
Main study					
- Site A	0.33	2.0	22	0.07 – 5	27/43
- Site B	0.23	2.3	7.5	0.1 – 7	27/48
2009 Study[a]					
- Site C	0	0.2	N/A	0.2 – 0.3	0/6

a: No measurements were made at Site D

N/A: not applicable. Not detected in any sample taken at this site

60. Methyl mercaptan is a colourless gas with the smell of rotten cabbage. The odour threshold is 3.1 µg m^{-3}. There are limited toxicity data on methyl mercaptan. The project-specific HCV was 4 µg m^{-3} and was derived from a non-standard study in which rats were exposed at 3 dose levels by inhalation 7 hours per day, 5 days per week for 3 months [38]. To derive the HCV, the lowest dose of 4 mg m^{-3} was designated a "minimum LOAEC" and an UF of 1000 applied. The authors of the monitoring report did not adjust for continuous exposure – this would have given a HCV of 0.83 µg m^{-3}.

61. We reviewed the relevant study and noted that, although occasional statistically significant changes in clinical chemistry parameters were seen in all dose groups when compared with the control group, the only dose-related trend was a decrease in albumin concentrations, which was not statistically significant. We consider that the NOAEC was the mid dose of 33 mg m^{-3} which was equivalent to a TWA concentration of 6.9 mg m^{-3}. A statistically significant reduction in terminal body weight was seen at the high dose (TWA concentration 23.2 mg m^{-3}). Applying an UF of 125 (see paragraph 49 for rationale) to the NOAEC gives a reference concentration of 55 µg m^{-3}.

62. With regard to its developmental toxicity, methyl mercaptan (methane thiol) was classified by Sullivan *et al* (2001) as a "chemical with insufficient data for classification".

63. Few data were found on background concentrations of methyl mercaptan. The annual mean concentrations around paper mills in South Karelia, Finland, were estimated to be 2 to 5 µg m^{-3} and the highest daily average concentration to be 50 µg m^{-3} [39]. During the 2009 survey, background levels of methyl mercaptan at a non-industrial business park were found to be less than 0.2 µg m^{-3}. Methyl mercaptan is also a product of biological processes and a component of bad breath [40].

Discussion of results on methyl mercaptan

64. The monitoring data indicate that all measured concentrations of methyl mercaptan were below our recommended health-based reference concentration of 55.2 ug m^{-3}. Thus, although the available data are limited, they do not indicate any health concerns from the concentrations measured in this study. Moreover, methyl mercaptan has a low odour threshold, which is likely to deter the public from tolerating prolonged exposure to the chemical.

<u>Polycyclic aromatic hydrocarbons (PAHs)</u>

65. Polycyclic aromatic hydrocarbons (PAHs) are a large group of structurally similar chemicals which are ubiquitous in the environment, where they are found both as gases and associated with particulates. Environmental exposure of humans is always to complex mixtures of different PAH constituents. Epidemiological studies have found an association between exposure to mixtures of certain PAHs and tumours of the lung, skin and possibly bladder and other sites. In addition, several PAHs have been shown to be carcinogenic in experimental animals when tested individually [41]. However, extensive toxicity data are available for only one PAH,

benzo(a)pyrene (B(a)P), and thus the evaluation of health risks from mixtures of PAHs is difficult.

66. In the main 2002-2005 study, the concentrations were measured of six carcinogenic PAHs commonly found as air pollutants. The results are summarised in Table 11. In the subsequent monitoring exercise, another carcinogenic PAH, dibenzo(a,l)pyrene, was also measured. The results from the subsequent study are given in Table 12.

Table 11: Concentrations of PAHs found in main monitoring study

	Concentrations found in study (ng m^{-3})			**No. detects[a]/ no. measure-ments**
	50th percentile assuming all non-detects were zero	50th percentile assuming all non-detects were present at detection limit	Maximum detected concentration	
Benzo(a)pyrene - Site A - Site B	 0 0	 0.12 0.10	 1.32 0.58	 14/32 6/32
Benzo(a)anthracene - Site A - Site B	 0 0	 0.22 0.11	 3.83 1.00	 15/32 14/32
Benzo(b,k) fluoranthene - Site A - Site B	 0.11 0.1	 0.14 0.16	 0.92 1.23	 21/32 18/32
Chrysene - Site A - Site B	 0.11 0.10	 0.15 0.12	 4.09 1.51	 20/32 19/32
Dibenz(a,h)anthracene - Site A - Site B	 0 0	 0.11 0.10	 0.29 0.81	 4/32 7/32
Indeno(123-cd)pyrene - Site A - Site B	 0 0	 0.11 0.10	 0.40 0.81	 11/32 6/32

a: we are informed that the range of detection limits was 0.01 to 0.6 ng m^{-3}

Table 12: Concentrations of PAHs in subsequent monitoring study

	Concentrations found in study (ng m^{-3})[a]			**No. detects[b]/ no. measure-ments**
	50th percentile assuming all non-detects at zero	50th percentile assuming all non-detects were present at detection limit	Maximum detected concentration	
Benzo(a)pyrene				
- Site C	0.11	0.11	0.11	2/2
- Site D (vapour phase)	0	0.06	0.06	1/3
- Site D (solid phase)	0	0.06	N/A	0/3
Benzo(a)anthracene				
- Site C	0.16	0.16	0.22	2/2
- Site D (vapour phase)	0	0.11	N/A	0/3
- Site D (solid phase)	0	0.11	N/A	0/3
Benzo(b,k) fluoranthene				
- Site C	0.22	0.22	0.22	2/2
Benzo(b)fluoranthene[c]				
- Site D (vapour phase)	0	0.06	0.07	1/3
- Site D (solid phase)	0	0.07	0.09	1/3
Benzo(k)fluoranthene[b]				
- Site D (vapour phase)	0	0.06	N/A	0/3
- Site D (solid phase)	0	0.06	N/A	0/3
Chrysene				
- Site C	0.56	0.56	1.01	2/2
- Site D (vapour phase)	0	0.14	N/A	0/3
- Site D (solid phase)	0	0.14	N/A	0/3
Dibenz(a,h)anthracene				
- Site C	0.06	0.11	0.11	1/2
- Site D (vapour phase)	0	0.06	0.07	1/3
- Site D (solid phase)	0	0.06	N/A	0/3
Indeno(123-cd)pyrene				
- Site C	0.11	0.11	0.11	2/2
- Site D	0	0.08	0.08	1/3
Dibenzo(a,l)pyrene[d]				
- Site D (vapour phase)	0	0.07	N/A	0/3
- Site D (solid phase)	0	0.07	N/A	0/3

a: At Site D, the filter placed in front of the absorption tube was analysed separately from the absorbent tube.. At Site C, a single solvent extraction was used for the filter and absorbent tube.
b: We are informed that the range of detection limits was 0.015 to 0.75 ng m^{-3} at Site C and 0.06 to 0.14 ng m^{-3} at Site D
c: At Site D, benzo(b)fluoranthene and benzo(k)fluoranthene were separately analysed.
d: Not measured at Site C
N/A: not applicable. Not detected in any sample taken at this site

67. The project-specific HCV for PAHs was 0.25 ng m^{-3} B[a]P as an annual average, which was recommended as a UK air quality standard by EPAQS in 1999 [41]. This standard was derived using B[a]P as a marker for the carcinogenic risk from all 7 PAHs commonly found in air and was based on the incidence of lung cancer in workers at an aluminium smelter. Carcinogenic potencies of the other 7 PAHs relative to B[a]P were derived from limited animal studies. The potencies are given in Table 13 below. Using this approach, the estimated contribution of BaP to the total carcinogenicity of the 7 PAH compounds was found by EPAQS to be similar in ambient air at two UK municipal sites, where it was calculated to be 44.6% and 37.5%, and the aluminium smelter, where it was calculated to be 49.3%. The studies at the smelter were therefore considered to form a suitable basis for recommending an air quality standard.

Table 13: Relative potencies for PAHs found in the monitoring studies, after EPAQS [41]

PAH	Relative Potency
Benzo(a)pyrene	1
Benzo(a)anthracene	0.1
Benzo(b,k)fluoranthene	0.11
Chrysene	0.03
Dibenz(a,h)anthracene	1.91
Indeno(123-cd)pyrene	0.08

68. Using the relative potencies estimated by EPAQS and the values for average measured concentrations of PAHs at landfill sites A and B in the main study, the contribution of BaP to the carcinogenicity of PAHs at Site A was calculated to be 45.1% which is similar to that at the aluminium smelter. Therefore, we consider that it is appropriate to use the EPAQS air quality standard to assess the risk from PAH concentrations at Site A. However, the contribution of BaP to the carcinogenicity of PAHs at Site B was only 15.7%, approximately one-third of that at the smelter. It is therefore appropriate to compare the concentration of BaP at this site with 15.7/45.1 of the air quality standard i.e. 0.08 ng m^{-3} B[a]P as an annual average.

69. A similar exercise was not carried out for the measurements recorded in the subsequent study at Sites C and D, in view of the small number of samples for each PAH. However, we note that the concentrations measured at these sites were generally lower than those measured in the main study.

70. Other air pollution monitoring in the UK has detected the presence of the PAH dibenzo(a,l)pyrene (DB(a,l)P) in a number of samples. DB(a,l)P is rarely measured in environmental studies because of the lack of a suitable analytical method and was monitored at only Site D, where it was not detected (limit of detection: 0.07 ng m^{-3}). The COC has advised that DB(a,l)P should be considered as a highly potent genotoxic carcinogen in experimental animals and that it is 10 to 1000 times more potent than B(a)P on the basis of results in short-term studies by non-inhalation routes [42]. There were insufficient data to draw any conclusions about its relative potency by the inhalation route.

71. No inhalation studies were found on the reproductive or developmental toxicity of PAHs and only limited oral studies are available. Lowest Observed Adverse Effect Levels (LOAELs) in these studies ranged from 10 to 133.3 mg kg bw^{-1} day^{-1} BaP [43].

72. Background concentrations of PAHs in air, measured at an urban and a semi rural site, are given in Table 14 below.

Table 14: Background concentrations of PAHs in air (ng m^{-3}), 2007 data [44]

PAH	London Brent (urban background)	Hazelrigg (semi rural)
Benzo[a]pyrene	0.085	0.084
Benz[a]anthracene	0.086	0.13
Dibenz[ah]anthracene + Dibenz[ac]anthracene	0.040	0.019
Benzo[b]fluoranthene + Benzo[j]fluoranthene	0.26	0.30
Benzo[k]fluoranthene	0.079	0.073
Indeno[123cd]pyrene	0.20	0.13
Chrysene	0.19	0.33

Discussion of results for PAHs

73. In the main study, the 50^{th} percentile exposure to B(a)P at Site A was below the EPAQS standard but the maximum exposure exceeded it. At Site B, the levels of B(a)P should be compared with a concentration of 0.08 ng m^{-3} B[a]P (see above). This concentration is slightly exceeded by the 50^{th} percentile concentration when calculated using the assumption that all the non-detects were at the detection limit and is exceeded 7-fold by the maximum concentration detected. Also, we note that measurements at Site B might have underestimated the concentrations emitted as the monitoring point was not always downwind of the site. Since the air quality guideline for B(a)P should be applied as an annual average, the concentrations measured at these sites are not a major cause for concern. Nevertheless, it would be desirable to ensure that the average concentrations are below the reference concentration wherever possible.

<u>Stibine (antimony trihydride)</u>

74. Concentrations of stibine were analysed only in the main study because analytical standards were no longer available when the subsequent study was carried out. The findings are summarised in Table 15 below.

75. Stibine is a colourless gas with an odour like rotten eggs. There are few toxicity data for stibine. It is haemolytic, but lethal concentrations are in the order of several hundred mg m^{-3} (see [18], Volume 2). The project-specific HCV was 5 µg m^{-3} and was based on a former occupational exposure standard of 0.52 mg m^{-3}. However, there is currently no occupational standard for stibine in the UK.

Table 15: Stibine concentrations in main study

	50th percentile (µg m-3)		Maximum detected concentration (µg m-3)	Detection limit (µg m-3)	No. Detects/ No. Measure-ments
	Assuming all non-detects were zero	Assuming all non-detects were present at detection limit			
Main study					
- Site A	82	82	222	14 – 35	10/16
- Site B	0	10	N/A	7 – 18	0/8

N/A: not applicable. Not detected in any sample taken at this site

Discussion of results for stibine

76. We consider that there are insufficient data to set a health-based reference concentration. We note that stibine concentrations at Site B were below the detection limit of 20 µg m^{-3}, but that it was present at up to 10 times this level at Site A. We are unable to assess the significance of this level until further information becomes available on the toxicity of stibine.

Styrene

77. Concentrations of styrene analysed in the main study are summarised in Table 16 below.

Table 16: Styrene concentrations in main study

	50th percentile (µg m-3)		Maximum detected concentration (µg m-3)	Detection limit (µg m-3)	No. Detects/ No. Measure-ments
	Assuming all non-detects were zero	Assuming all non-detects were present at detection limit			
Main study					
- Site A	0.1	1.0	109	0.01 – 1.0	32/55
- Site B	0.27	0.3	4.6	0.01 – 0.4	32/36

78. Styrene is a volatile, oily liquid with a sweet smell. Findings from occupational studies indicate that it is neurotoxic and can impair colour discrimination. The project-specific HCV for styrene was 70 µg m^{-3}. The WHO Air Quality Guidelines for Europe recommended in 2000 that the air quality guideline for styrene could be either 0.26 mg m^{-3} as a weekly average, based on LOAECs for subtle neurotoxic effects in occupationally exposed populations, or 70 µg m^{-3} as a 30-minute average, based on the odour detection threshold [15]. Subsequently, the neurobehavioural studies on styrene were reviewed comprehensively by the Health and Safety Executive (HSE) in a European Union Risk Assessment Report (RAR) on styrene [45]. This review criticised the available neurotoxicity studies and concluded that the crucial issue in relation to the impact of styrene on the nervous system is the need to avoid acute CNS depressant effects and associated symptomatology. The NOAEC

for such effects was considered to be 428 mg m^{-3} for 7 hours exposure, with minor impairment seen at 856 mg m^{-3} for 1 hour. We agree with this assessment.

79. Carcinogenicity studies have been carried out on styrene in the mouse by the inhalation and oral routes, and in the rat by the oral route. In both mouse studies, styrene caused lung neoplasms [45]. However, we agree with HSE that this finding is not relevant to humans because of differences in the metabolism of styrene in mouse and man. No treatment-related tumours were seen at any other site. There is no evidence from extensive epidemiological investigations that long-term exposure to styrene has produced lung damage or lung cancer in humans and styrene has shown no evidence of carcinogenicity in several bioassays in the rat when administered by the oral or inhalation route. Colleagues on the COC and COM have advised that it is unlikely that styrene poses a mutagenic risk leading to adverse health consequences in humans [46-47].

80. We reviewed the NOAECs and NOAELs for other toxicological endpoints as given in the HSE review. These were corrected for continuous exposure and an appropriate uncertainty factor was applied to give a possible health-based reference concentration based on that endpoint. The results are given in Table 17.

Table 17: Toxicological endpoints for styrene, and corresponding possible reference concentrations

Health endpoint (species)	**NOAEC or NOAEL**	**Proposed reference concentration (mg m^{-3}) (correction factors and uncertainty factor (UF))**
Skin and respiratory tract irritation (humans)	433 mg m^{-3} for 7 h	43 (not adjusted to 24 h exposure, UF=10)
Ototoxicity (active rat)	1300 mg m^{-3} for 12 h day^{-1}, 5 d $week^{-1}$ for 4 weeks	19 (adjusted to 24 h exposure; UF=25) [a]
(sedentary rat)	2165 mg m^{-3} for 6 h day^{-1}, 5 d $week^{-1}$ for 4 weeks	16 (adjusted to 24 h exposure; UF=25) [a]
Systemic effects Rat Mouse	 1000 mg kg^{-1} day^{-1} 150 mg kg^{-1} day^{-1}	 35 (UF= 25) [b] 7.5 (UF = 10) [c]
Developmental effects (rat)	650 mg m^{-3} for 6 h day^{-1}, 7 d $week^{-1}$ [d]	1.6 (adjusted to 24 h exposure; UF = 100)

a: It may not be appropriate to convert this to 24 hour exposure
b: Assumptions: 100% absorption by both routes and rat 24 hour respiratory volume of 1.15 m^3 kg^{-1} bw. For UF, see paragraph 49.
c: Assumptions: 100% absorption by both routes and mouse 24 hour respiratory volume of 1.995 m^3 kg^{-1} bw. Endpoint based on liver toxicity: the RAR states that "in extrapolation to humans careful consideration has to be taken of the specifics of mouse metabolism and the high sensitivity of this species for liver toxicity as compared to eg the rat" [45]. Therefore, no intraspecies UF has been used.
d: Postnatal developmental delays and decreased body weight. No structural anomalies seen.

81. Styrene was classified by Sullivan et al (2001) as a "chemical of no/unlikely interest" with regard to reproductive effects. The authors concluded that there was little evidence that styrene exerts any reproductive or developmental toxicity. However, Table 17 includes the results of a recent, good quality study which demonstrated postnatal developmental delays and effects on body weight at a TWA concentration of 541 mg m^{-3}. The TWA NOAEC was 163 mg m^{-3} [48] .

Discussion of results for styrene

82. Both the 50th percentile and maximum concentrations of styrene recorded in the main study were below the lowest proposed reference concentrations in Table 17 and we consider that there are unlikely to be any health concerns associated with these concentrations.

Toluene

83. Concentrations of toluene analysed in the main study are summarised in Table 18 below.

Table 18: Toluene concentrations in main study

	50th percentile (µg m^{-3})		**Maximum detected concentration (µg m^{-3})**	**Detection limit (µg m^{-3})**	**No. Detects/ No. Measure-ments**
	Assuming all non-detects were zero	Assuming all non-detects were present at detection limit			
Main study					
- Site A	7.3	7.3	923	0.01 – 1	53/55
- Site B	1.1	1.1	41	0.01 – 1	36/36

84. Toluene is a clear liquid with a distinctive smell. It is an aromatic hydrocarbon which has a number of industrial uses. There are substantial data on the effects of toluene in occupationally exposed humans. The weight-of-evidence from these studies indicates neurologic effects as the most sensitive endpoint. The project-specific guideline for toluene was 0.26 mg m^{-3} and was equivalent to the WHO Air Quality Guideline for Europe [15], which was set on the basis of neurobehavioural effects in a small occupational exposure study [49-50].

85. In 2005, the US Environmental Protection Agency (USEPA) updated its more extensive review of toluene and identified a number of new epidemiological studies which clarify the dose-response relationship for neurotoxicity in humans [51]. It identified a mean NOAEC from 4 occupational studies of 128 mg m^{-3} (TWA exposure). This was converted from occupational to continuous exposure[8], giving an adjusted concentration of 46 mg m^{-3}. An uncertainty factor of 10 was applied for

[8] The conversion was made using the assumption that a worker would inhale 10 m^3 of air over an 8-hour shift and works five days a week, and an adult member of the public inhales 20 m^3 over a 24 hour period.

interindividual variation, giving a RfC[9] of 5000 µg m^{-3}. We consider that this RfC should be used as the health based guideline value in assessing the public health significance of airborne concentrations of toluene.

86. Toluene was classified by Sullivan *et al* (2001) as a "chemical of less likely interest." This was based on observations of fetotoxicity, but not teratogenicity, in mice and rats; behavioural deficits and disturbances in brain development in mice and rats; limited studies suggesting an increase in menstrual disorders and, possibly, reduced fecundity and an increase in spontaneous abortions, in women exposed occupationally to toluene; and substantial evidence of a characteristic toluene embryopathy in babies born to women abusing toluene by recreational sniffing, where short term exposures can be extremely high, in the range 18750 to 45000 mg m^{-3} [7]. The US EPA concluded that reproductive effects occurred at higher exposures than those which caused other effects [51].

Discussion of results for toluene

87. Both the 50th percentile and maximum concentrations of toluene recorded in the main study were below concentrations which might give cause for concern.

Polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans

88. Polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans are collectively known as "dioxins". They are persistent and widely dispersed environmental contaminants. Dioxins are produced in a number of thermal reactions and as trace contaminants during the synthesis of many organochlorine compounds and during some industrial processes. Concentrations of dioxins were only analysed in the main study. The results are given in Table 19 below. The concentrations are expressed as "WHO-Toxic Equivalents" (TEQ) [52].

Table 19: Dioxin concentrations in main study

	50th percentile (fg WHO-TEQ m^{-3})		**Maximum detected concentration (fg WHO-TEQ m^{-3})**	**Detection limit (typical) (fg m^{-3})**	**No. Detects/ No. Measure-ments**
	Assuming all non-detects at zero	Assuming all non-detects were present at detection limit			
Main study					
- Site A	15	15	77	<4 – 6	32/32
- Site B	15	15	1839		27/32

[9] RfC: inhalation reference concentration. A US term for an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.

89. In addition to the airborne measurements made above, the potential exposure to dioxins of individuals living near the landfill sites was assessed using the Environment Agency's "Dioxin Risk and Exposure Assessment Model" (DREAM) version 1.0x (see [18], section 5.4.1). This model provides estimated exposures to dioxins, based on a given airborne concentration profile, applying a set of detailed assumptions about the consumption of different types of food, body weights and other variables. Using this information, the model provides estimated exposures for individuals aged 1.5-2.5 years, 2.5-3.5 years and 3.5-4.5 years, school children and adults. The estimated exposures by all routes (direct inhalation together with indirect pathways) are set out in Table 20 below. The results for Site B were dominated by a single high value in the summer survey. According to the study authors, no reason was found for this but it was considered unlikely to be typical of concentrations at Site B.

Table 20: Estimated exposures of local residents to dioxins from local produce

Population group	Site A	Site B	Site B (excluding single high value)
	Dioxin exposures (pg WHO-TEQ per kg body weight)		
Children aged 1.5 -2.5	1.80	7.74	1.63
Children aged 2.5 -3.5	1.52	6.52	1.37
Children aged 3.5 -4.5	1.35	5.77	1.22
School children	0.56	2.47	0.52
Adults	0.30	1.29	0.27

90. No airborne project-specific HCV was set for dioxins. In general, inhalation is considered to be a minor route of exposure to environmental dioxins and it is accumulation in food, and subsequent consumption, which is considered to be the important route [53].

91. In 2001, we recommended a Tolerable Daily Intake (TDI) for dioxins and dioxin-like polychlorinated biphenyls of 2 pg WHO-TEQ kg bw^{-1} day^{-1}, based on effects on the developing male reproductive system mediated via the maternal body burden [54]. This TDI is appropriate for assessing the risk to health of intakes via all routes, including inhalation and oral intakes.

92. Background concentrations in food can be obtained from analysis of samples of the food groups that made up the 2001 Total Diet Survey. The estimated average intakes of the sum of dioxins and dioxin-like PCBs from the UK diet for adults and schoolchildren were 0.9 pg WHO-TEQ kg bw^{-1} day^{-1} and 0.7-1.8 pg WHO-TEQ kg bw^{-1} day^{-1}, respectively, with younger children being at the upper end of the range for schoolchildren [55]. (Dioxin-like PCBs were monitored in the main monitoring study but none was detected above the typical detection limit of 5 fg m^{-3}).

Discussion

93. The exposures of local residents to dioxins in food estimated from the concentrations detected at Site A are similar to background levels for 2001 and below the TDI of 2 pg WHO-TEQ kg bw^{-1} day^{-1} for all age groups. At Site B, the estimated exposures were considerably higher as a consequence of a single high measurement. The reason for this high concentration is unknown, but we note that it occurred only once in over 60 measurements. To provide context, the level of dioxins and furans at a distance of 20 metres from a bonfire burning domestic waste was recorded to be up to 580 fgTEQ/m^3, about a third of the single high value recorded at Site B [56]. If this value is excluded, estimated exposures are similar to those at Site A. The main concern about dioxins is accumulated exposure leading to a high body burden. The reliability of the DREAM model has not been tested under the circumstances of a single high exposure, as recorded at Site B, and consequently the validity of the model findings are subject to some uncertainty.

Chemicals with no project-specific HCV

<u>2-methyl furan</u>

94. The maximum concentration of 2-methyl furan found in the main study was 2.0 ug m^{-3} [18]. 2-methyl furan is used in food as a flavouring agent and was evaluated together with other furan-substituted aliphatic hydrocarbons by the WHO/FAO Joint Expert Committee on Food Additives (JECFA) in 2008 [57]. JECFA noted that furan is carcinogenic and its carcinogenicity is believed to involve a reactive genotoxic metabolite formed by epoxidation and opening of the furan ring. It also noted that there is evidence from studies *in vitro* and *in vivo* that 2-methyl furan undergoes bioactivation to a reactive ring-opened metabolite that binds covalently to both protein and DNA. However, there are no standard toxicological studies on 2-methyl furan by the oral or inhalation routes and the few available genotoxicity data have produced conflicting results [57]. We confirm that it is not possible to set a health-based reference concentration for this compound and, therefore, it is not possible to assess the significance of the levels of 2-methyl furan measured in the study.

<u>Acenaphthylene, Benzo(ghi)perylene and Phenanthrene</u>

95. The maximum concentrations of acenaphylene, benzo(ghi)perylene and phenanthrene found in the main study were 1.72, 0.48 and 15.11 ng m^{-3}, respectively [18]. These compounds are PAHs but it is not known whether they are carcinogenic. JECFA reviewed these PAHs in 2005. Benzo(ghi)perylene was considered to be genotoxic *in vitro*, but there were no *in vivo* studies. The genotoxicity results for phenanthrene were considered to be equivocal and acepnapthylene was considered to have an inadequate database [58]. There are inadequate data to evaluate other potential toxicological effects, including carcinogenicity. We confirm that it is not possible to set health-based reference concentrations for these substances nor to assess the significance of the concentrations measured in the study.

Recent epidemiological studies by the Small Area Health Statistics Unit (SAHSU)

Geographic density of landfill sites and risk of congenital anomalies in England [59]

96. This study investigated the risk of congenital anomalies in relation to an index of geographic density of landfill sites across 5 x 5 km grid squares in England using postcoded data from the National Congenital Anomalies Database and on terminations of pregnancy for serious congenital anomalies. In total, 8,804 landfill sites were included in the study; these had been operational at some time between 1982 and 1997. A landfill exposure index was calculated for each 5 x 5 km grid square which gave a measure of the proportion of births in the square that was within 2 km of a landfill site and which was weighted for the number of sites in the square. A value of one in this index could arise, for example, where all births in that square were within 2 km of a single landfill site, or where exactly half the births were within 2 km of two landfill sites. Each increment of the exposure index consequently represented the equivalent of adding one site within 2 km of all births. The total number of congenital anomalies in each 5 x 5 km grid cell was also computed by summation. Analysis was carried out separately for landfill sites handling special, and non-special or unknown, waste. For each group of landfills, the index was classified into four categories of intensity, and risks for the second, third and top categories were compared to the bottom category, which comprised 5 x 5 km grid squares which had no such landfill site within 2 km of any birth. For special waste sites[10], after adjustment for confounding, there was a small but significantly increased risk of all anomalies combined and of cardiovascular defects for the third category [odds ratios 1.08 (95% credible interval[11] 1.02, 1.13) and 1.16 (1.00, 1.33) respectively] but not the top category; and of hypospadias and epispadias for the third and top categories [odds ratios 1.11 (1.02, 1.21) and 1.12 (1.02, 1.22)] respectively. No excess risk was found in relation to other types of landfill sites.

97. This ecological study suffered from the same limitations that the COT highlighted in its 2001 statement. In particular, the National Congenital Anomalies Database used in this study suffers from poor ascertainment [60]. This has potential to introduce substantial bias into epidemiological studies. It is difficult to know what effect, if any, this might have on the observed results but there is no evidence that ascertainment problems vary systematically with landfill site locations. We understand that the national database now includes data from most of the local and regional congenital anomaly registers. Results from the study were similar when analysis was restricted to areas covered by a local register, but these areas accounted for only a small proportion of all births in England. We recommend that a high quality national register should be established, to facilitate good quality epidemiological research on the aetiology of congenital anomalies.

98. It is always difficult to interpret the results of epidemiological studies which report an apparent small increased relative risk, no matter how well conducted, because of the possibility of uncontrolled bias and/or residual confounding. Bias

[10] Special waste sites were landfill sites which received wastes now classed as both hazardous and non-hazardous waste.

[11] An interval that includes the true parameter with probability 0.95; the Bayesian analogue to the frequentist 95% confidence interval.

may arise from the data sources used, as described above, or because exposure to the key hazard has not been assessed accurately by the methods used in the study [59]. Confounding occurs when another variable is associated with the exposure of interest and independently determines risk of the health outcome of concern. Confounding factors can be taken into account at the design stage or the analysis stage but complete adjustment is not always possible, particularly in ecological studies. We note that the few increased risks reported in this study were small, and that an exposure-response relationship was only evident for one category of anomaly. Occasional positive findings of this sort can be expected to occur simply by chance. Therefore, we conclude that the results of this study do not give grounds for any specific concerns or recommendations relating to the health of pregnant women or those wishing to start a family who live in the vicinity of a landfill site.

Down syndrome in births near landfill sites [61]

99. This study found no excess risk of giving birth to a child with Down syndrome in populations living within 2 km of a landfill site in England and Wales. Again, there are limitations to the study due to its ecological design and the possibility of residual confounding. The study used a good quality Down syndrome register as the source of health data but did not include data on miscarriages because there was no adequate database. It is estimated that over 50% of fetuses with trisomy 21 are spontaneously aborted and, therefore, it is unfortunate that these data could not be included in the study. Nevertheless, despite the limitations, we regard these results as reassuring.

Overall conclusions and recommendations

100. A considerable body of work has been carried out in relation to landfill sites since the initial publication of the EUROHAZCON study in 1997. In overall terms, we consider the findings of this work reassuring. In particular, we have found no causes for concern for the health of families with infants or for couples who live in the vicinity of landfill sites and who are considering having a baby.

101. We welcome the monitoring work by the EA which, we believe, comprises the most detailed survey to date of chemicals to be found at the boundaries of landfill sites. Although many of the chemicals found have been reported as components of landfill gas, it cannot be known for certain that they were emitted from the landfill sites; they may have been emitted, either wholly or in part, from other sources. However, the results indicate the types and concentrations of chemicals to which individuals at the boundaries of landfill sites such as those studied could be exposed. In considering the relevance of the results to earlier epidemiological studies, we note that Sites A and B had gas control measures, in line with all new sites which accept biodegradable waste. Older sites, which have not implemented such measures, should already have passed peak methane emission levels. The results of this research may not necessarily apply to hazardous waste sites or those which accepted both hazardous and municipal waste (co-disposal sites). However, we are informed that hazardous waste is unlikely to degrade biologically to generate significant quantities of gaseous emissions and that emissions are likely to vary according to what is deposited in the site. Co-disposal, which could lead to production of substantial quantities of gas, possibly carrying with it hazardous waste,

ceased in July 2004. Sites A and B could not necessarily be considered representative and there are other, larger sites which could have greater emissions. Nevertheless, the landfill gas composition at these two sites was reported to be broadly typical of the composition in the national database in respect of compounds which are of particular concern because they have the potential to cause reproductive or developmental effects [7].

100. There are a number of limitations to this monitoring work and our assessment of it. Only a small number of sites have been surveyed and only a limited number of measurements were made at Sites C and D. Little or no information is available on the reproductive toxicity of several of the monitored chemicals and a small number have too few toxicity data overall for us to assess the significance of the concentrations found. Nevertheless, in general, we consider that the results of this study are reassuring. The monitoring work indicated that the levels at which most of the measured substances were found would not be expected to cause developmental or other chronic health effects, and there were no major concerns in relation to any findings. We have formed specific conclusions and recommendations in relation to the following chemicals:

Arsine: It is difficult to provide an informative assessment on arsine from the limited data available. We recommend that more sensitive sampling or analytical methods be developed and then further monitoring data obtained from the boundaries of landfill sites. Also, there is a need for quantitative information on the conversion of arsine to arsenic in an appropriate experimental species.

Chromium: It is not possible to draw reliable conclusions because of a lack of information on speciation and inadequacies in available measurement techniques. We recommend that, should techniques be developed to measure chromium (VI) in air at lower detection limits, it would be appropriate to use these in future monitoring.

Dimethyl sulphide and dimethyl disulphide: On the basis of the limited toxicological data available on these compounds, no adverse health effects are expected for the concentrations measured in the study. However, there are no data on reproductive or developmental toxicity nor on carcinogenicity and, therefore, it is not possible to give definitive advice.

Formaldehyde: The results raise no concerns about chronic health effects or adverse reproductive or developmental effects, but the maximum concentrations reported might result in some short-term irritant effects in some individuals at the boundary of landfill sites.

Methyl mercaptan: On the basis of the limited toxicological data available on this compound, no adverse health effects are expected from the concentrations measured in the study. However, there are no data on reproductive or developmental toxicity nor on carcinogenicity and, therefore, it is not possible to give definitive advice.

Polycyclic aromatic hydrocarbons: The concentrations measured in the study are not a major concern with respect to the critical toxic effect, which is carcinogenicity.

However, we note that only limited reproductive and developmental toxicity data are available on these compounds.

Stibine: There are insufficient toxicity data to assess the significance to health of the results.

Dioxins: The results indicate that estimated intakes from eating locally grown produce are, in most cases, comparable to background exposures, below the tolerable daily intake for these compounds, and unlikely to be of concern. The reason for a single high concentration at Site B has not been identified but we note that it occurred only once in over 60 measurements.

101. In addition, we recommend:

- that a high quality national register should be established, to facilitate good quality epidemiological research on the causes of congenital anomalies,

- the development of improved sampling and analytical techniques for monitoring the concentrations of trace substances emitted to the environment generally.

102. We have now reviewed a number of studies of ecological design which have investigated the association between adverse health outcomes and landfill sites. The risk estimates which were derived from these studies are small and, as explained above, it is not possible to discriminate effects due to confounders and bias from those which might be causally associated with the hazard under investigation. We therefore consider that there would be little value in undertaking further studies of this type.

103. In 2001, the COT recommended that case-control or cohort studies could be carried out. It has also been suggested that studies on personal uptake of pollutants by residents living near landfill sites and, possibly, on exposure of workers on landfill sites could usefully inform such studies. However, the findings in this study do not point to any specific health outcomes or exposures that merit further investigation as the focus of such studies.

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ABBREVIATIONS

ATSDR	US Agency for Toxic Substances and Disease Registry
B(a)P	benz(a)pyrene
BMC	benchmark concentration
$BMCL_{10}$	lower 95% confidence limit of the benchmark concentration for a 10% response
bw	body weight
CICAD	Concise International Chemical Assessment Document
COM	Committee on the Mutagenicity of Chemicals in Food, Consumer Products and the Environment
COC	Committee on the Carcinogenicity of Chemicals in Food, Consumer Products and the Environment
DB(a,l)P	dibenz(a,l)pyrene
DH	Department of Health
DMS	dimethyl sulphide
DMDS	dimethyl disulphide
EA	Environment Agency
EPAQS	Expert Group on Air Quality Standards
EU	European Union
FAO	Food and Agriculture Organisation
fg	femtogram i.e. 10^{-15} grams
HCV	health criteria value
HSE	Health and Safety Executive
JECFA	Joint FAO/WHO Expert Committee on Food Additives
kg	kilogram(s) i.e. 10^{3} grams
km	kilometre(s) i.e. 10^{3} metres
LOAEC	lowest observed adverse effect concentration
LOAEL	lowest observed adverse effect level
m^{-3}	per cubic metre
mg	milligram(s) i.e. 10^{-3} grams
MOE	margin of exposure
NAQS	National Air Quality Standard
ng	nanogram(s) i.e. 10^{-9} grams
NOAEL	no observable adverse effect concentration
NOAEL	no observable adverse effect level
NOx	oxides of nitrogen
PAH	polycyclic aromatic hydrocarbon
PCB	polychlorinated biphenyl
PCDD	polychlorinated dibenzo-*para*-dioxin
PCDF	polychlorinated dibenzofuran
PM_{10}	particulate matter with a diameter of less than 10 micrometres
ppb	parts per billion
pg	picogram i.e. 10^{-12} grams
SAHSU	Small Area Health Statistics Unit
TEQ	toxic equivalent
TWA	time weighted average
UF	uncertainty factor
µg	microgram(s) i.e. 10^{-6} grams
USEPA	US Environmental Protection Agency

VOC	volatile organic compound
WEL	workplace exposure limit
WHO	World Health Organisation
WHO-TEQ	toxic equivalent calculated using factors published by WHO

LIST OF CHEMICALS

Chemicals measured in main landfill study

1,1,1-Trichloroethane	Dichlorodifluoromethane
1,1-Dichloroethane	Dichlorofluoromethane
1,2-Dichloroethane	Dichloromethane
1,2-Dichloroethene	Dimethyl disulphide
1,3-butadiene	Dimethyl sulphide
2-butanone	Ethyl mercaptan
2-Ethyl-1-hexanol	Ethylbenzene
2-Methylfuran	Fluoranthene
Acenaphthene	Fluorene
Acenaphthylene	Formaldehyde
alpha-Terpinene	Indeno (123-cd) pyrene
Anthracene	Lead
Antimony	m+p Xylene
Arsenic	Manganese
Arsine	Mercury
Benzene	Methyl mercaptan
Benzo (a) anthracene	Naphthalene
Benzo (a) pyrene	Nickel
Benzo (b/k) fluoranthene	Nitromethane
Benzo (ghi) perylene	o Xylene
Cadmium	Phenanthrene
Carbon disulphide	Polychlorinated biphenyls (PCBs)
Chlorobenzene	Polychlorinated dibenzo-*p*-dioxins, polychlorinated dibenzofurans (PCDDs and PCDFs)
Chlorodifluoromethane	Pyrene
Chloroethane	Stibine
Chloroethene	Styrene
Chloroform	Tetrachloroethene
Chloromethane	Thallium
Chromium	Tin
Chrysene	Toluene
Cobalt	Trichloroethene
Copper	Trimethylbenzene
Dibenzo (ah) anthracene	Vanadium
Dichlorobenzene	

Chemicals screened out because the maximum concentrations measured were below 1% of the project-specific HCV

Acenaphthene	Dichlorodifluoromethane
Anthracene	Fluoranthene
Antimony	Fluorene
2-Butanone (methyl ethyl ketone)	Mercury
Chlorobenzene	Naphthalene
Chloroethane	Nitromethane
Chlorodifluoromethane	Pyrene
Chloromethane	Thallium
Dichlorobenzene	Tin
1,1-Dichloroethane	Trimethylbenzene
1,2-Dichloroethene	Vanadium
Dichlorofluoromethane	

Chemicals whose maximum concentrations were ≥1% and average concentrations were below 75% of the project-specific HCV and which were not reviewed in depth by COT

1,3-butadiene	Dichloromethane
2-Ethyl-1-hexanol	Ethyl mercaptan
1,1,1-Trichloroethane	Ethylbenzene
alpha-Terpinene	Lead
Arsenic	m+p Xylene
Benzene	Manganese
Cadmium	Nickel
Carbon disulphide	o Xylene
Chloroform	Trichloroethene
Cobalt	Tetrachloroethene
Copper	

Chemicals whose average concentrations were at or above 75% of the project-specific HCV and which were reviewed in depth by COT

Arsine	Methyl mercaptan
Chromium	Potentially carcinogenic polycyclic aromatic hydrocarbons (PAHs)*
1,2-Dichloroethane	Stibine
Dimethyl disulphide	Styrene
Dimethyl sulphide	Toluene
Formaldehyde	

* Benzo(a)pyrene, benzo(a)anthracene, benzo(b,k)fluoranthene, chrysene, dibenz(a,h)anthracene, indeno(123-cd)pyrene

Other chemicals reviewed by COT

Chloroethene	Polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans
2-methylfuran	Benzo(ghi)perylene
Acenaphthylene	Phenanthrene

Other substances measured in study

Aspergillus fumigatus	Nitrogen dioxide
Endotoxins	PCBs
Entrobacteriaceae	Penicillia
Fibres	PM_{10}
Fungi and yeasts	Sulphur dioxide
Gram negative bacteria	Thermophilic bacteria
Hydrogen sulphide	Thermophilic fungi
Mesophilic aerobes	Yeasts
Moulds	

How we have reported measurements

1. In this statement, when reporting the concentrations of chemicals measured at the boundaries of the landfill sites, we have presented the 50th percentile value of the measurements made for each chemical at each site, together with the maximum concentration detected. The 50th percentile is the middle value of a set of numbers in order of their size. It is often used when the data are not evenly distributed about the average. For example, it is used when there are a few values that are much higher than the other measurements and when to quote the average (arithmetic mean) of the values would give undue weight to these high readings.

2. Consider a set of 9 numbers.

Number	5	27	44	7	87	55	73	18	172

The average of these would be 54. The 50th percentile is the middle value. If we arrange the numbers in ascending order, we can see this is 44.

Number	5	7	18	27	**44**	55	73	87	172

3. When concentrations of chemicals are measured, the instruments may not be sufficiently sensitive to measure some low levels. The lowest level we can measure is known as the limit of detection or detection limit (DL). This can vary over the course of the study, for example, as different techniques are used. In the example below, the DL was initially 100 micrograms per cubic metre (µg per m^3) and, in August, was reduced to 10 µg per m^3.

Date	21/5	23/6	20/7	20/8	21/9	19/10	20/11	18/12	23/1
True concentration	5	27	44	7	87	55	73	18	172
Detection limit (DL)	100			10					

4. Where a value is below the DL, it is not known whether the substance is or is not present. In our statement, we have reported the median concentrations both with the assumption that the substance was not present (concentration assumed to be 0) and with the assumption that the substance was present at the DL prevailing at the time. The true concentration of the chemical lies at or between these levels. This is shown in the table below.

Date	21/5	23/6	20/7	20/8	21/9	19/10	20/11	18/12	23/1
True value	5	27	44	7	87	55	73	18	172
Measurement	<100	<100	<100	<10	87	55	73	18	172
Assuming all non-detects are 0	0	0	0	0	87	55	73	**18**	172
Assuming all non-detects are at the DL	100	100	100	10	**87**	55	73	18	172

We can see that, if we report the values below the detection limit as 0, the 50th percentile will be 18. However, if we report the values as the prevailing limit of detection, the 50th percentile is 87. The true value lies at or between 18 and 87.

5. Note that if none of the values measured for a chemical were above the detection limit, the 50th percentile level calculated by assuming that all non-detected levels were present at the DL will not be zero. Instead, it will be the 50th percentile value of the detection limits when the measurements were made.