PUBLIC INTEREST CONSULTANTS

Comments on the "Report on the analysis of PCDD/PCDF and Heavy Metals in Soil and Egg samples related to the Byker incinerator"

by
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on behalf of the
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Summary

1. The hard and valuable work undertaken by Dr Pless Mulloli and her co-workers in undertaking the sampling and review of the contamination for the main report is fully recognised.
2. The contamination of allotments in Newcastle by high levels of dioxins and heavy metals from the Byker incinerator could be one of the most serious dioxin contamination events in the UK (and possibly even in the world). This is because it is nearly unprecedented to have high levels of dioxin contaminated material been introduced so directly onto land used for personal food supplies including eggs for a large number of people.
3. It is unlikely that the Newcastle Study covers the full extent of ash distribution in the Newcastle area. More work and research will need to be undertaken to establish the extent of other locations which may have received ash.
4. The scope of the Newcastle University study has meant that it has been outside the remit of the report to review current exposure levels, particularly vulnerable individuals and exposure standards for dioxins. The current study has also not presented any data for dioxin like PCBs nor for other halogenated, particularly brominated and chloro-bromo, dioxins. No assessment has been made of the possible exposure to workers handling the ash (either in the plant or on allotments).
5. It is noted that the initial contamination by incinerator residues and the extent of the contamination has had to be drawn to the attention of the regulatory authorities by the public rather than being discovered as a result of regulatory action.
6. In these circumstances it is recommended that the appropriate response to the reduction in public confidence in the regulatory authorities requires full and frank public consultation and debate on the risks associated with the contamination and the appropriate regulatory and medical response to the incidents.
7. It is appreciated that some of the sampling has been undertaken by different agencies and that the protocols may not be available to the Newcastle University team. However this means that the levels reported by Newcastle University are probably not the worst cases. At the Blucher allotment, for example, levels of 9,500 ng I-TEQ / kg have been found.
8. There are some inconsistencies in the draft of the University report which was supplied for these comments. The main implication is that the assessment of metal contamination is not as conservative as would seem desirable for material spread onto allotments.
9. Whilst the assumptions for exposure intake calculations have been made clear in the University report these important details are all not included in the reports from the Environment Agency and Food Standards Agency.
10. The only pathways for human exposure to dioxins considered in the University report is via egg consumption. The assessment of impacts on body burdens has only been carried out for 35 and 70 kg persons but not for small children / babies who are likely to be at greater risk from exposure. Maximum exposure from eggs has been assessed on the basis of one egg per day / person. This may underestimate consumption for those who keep their own poultry. The University calculation is not, in any case, a worst case as a dioxin level of twice the level assumed[1] has been measured in Bantam eggs from Branxton A.

[1] 56pg / g compared with the 30 pg / g used in the Executive Summary calculation.

11. The Food Standards Agency has also failed to assess dioxin intakes from eggs for children upto 10 years old.
12. The Environment Agency emphasised the importance of this pathway in an earlier draft report but omitted it completely from the final version.
13. None of the reports include any measured data on PCB levels in Newcastle eggs.
14. None of the reports considered dioxin exposure from eating contaminated poultry meat
15. None of the reports consider exposure for children with Pica[2] behaviour. The Environment Agency do examine soil ingestion as a pathway for their assessment of Dinnington Park but at much lower levels than have been reported in the literature and even in other Environment Agency reports. The assessment of soil ingestion for allotments is based on a much lower intake than even the Dinnington Park assessment.
16. The highest level of allotment soil contamination considered by the Environment Agency is 167 ng/kg. Allotment holders have reported seeing children putting ash in their mouths directly from paths. This material is an order of magnitude, or more, higher contamination.
17. None of the reports consider that those exposed from eggs on allotments may also receive higher than average levels of dioxin intake through other parts of their diet (such as eating fish or fish oil supplements).
18. All the reports quote contamination levels as I-TEQ rather than as WHO –TEQ. Whilst it is appreciated that I-TEQ makes comparison with other data more straightforward it should be acknowledged that the intake levels in the report will need to be increased by about 10% when they are compared with WHO -TDI exposure standards.
19. All the reports have avoided consideration of plausible worst case scenarios - for reasons including those noted above. The current risk/exposure assessments by both the Environment Agency and the Food Standards Agency do not appear to be protective given the higher levels of contamination recorded on some sites, the uncertainties associated with the extent of contamination; the exposures to mixtures of dioxins and heavy metals etc.
20. However the intakes that have been calculated by the Environment Agency and Food Standards Agency still bring exposures within the ranges at which adverse effects may occur. Gray and co-workers, for example, found decreased sperm counts in rats at the equivalent of 14 pg/kg bw/day daily intake. It is considered that this is a matter of great concern.

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[2] High levels of soil eating

Introduction

21. The hard and valuable work undertaken by Dr Pless Mulloli and her co-workers in undertaking the sampling and review of the contamination for the main report is fully recognised. BAN Waste Group regret however that their comments on the University report, always provided by the appropriate deadlines, have not been included with the final version.

22. These notes and comments are based on the data in the report and on issues which may still need to be considered or addressed.

23. The information available when these comments were prepared was the 3rd October draft Report, the 7th November draft Executive Summary and the 30th November Draft Environment Agency Review and the (undated) Food Standards Agency 'Preliminary contribution to Annexes'.

24. It is unclear, at the time of drafting this note, which suggestions made earlier in the process have been included in the final report as no further drafts post 3rd October have been made available. There has been no feedback on the earlier comments and information which was submitted for discussion even though this was also copied this directly to the Environment Agency and the Food Standards Agency. These bodies did not reciprocate with copies of their own drafts (which were provided after the deadlines set by the working group) but final copies of their submissions have been obtained at a late stage and these comments extend to those reports.

25. The contamination of allotments in Newcastle by high levels of dioxins and heavy metals from the Byker incinerator could be one of the most serious dioxin contamination events in the UK (and possibly even in the world). This is because it is nearly unprecedented to have high levels of dioxin contaminated material been introduced so directly onto land used for personal food supplies including eggs for a large number of people.

26. The comparison with other incidents is addressed in the 24th May Stage 1 Report which indicates that:

    "A consideration for future risk assessment of the situation created by using ash on footpaths will be the bioavailability of any contamination. From the literature there is only one case of contamination, which may possibly resemble the situation in the current study. This is the usage of the copper slag ' Kieselrot' on playgrounds and sports fields in Germany (24, 25). 800,000 tones of slag from ore mining had been used during the 1950s and 1960s. This material was found to contain 64,500 ng/kg I-TEQ. In garden soil in the vicinity of a highly contaminated sports field a concentration of 154 ng/kg was detected. However, the bioavailability was found to be low: when the body burden of people with extensive recreational and occupational exposure to dust from Kieselrot slag was analysed only slightly elevated levels were detected (26)."

27. We are not aware of any cases of contamination which resemble the situation of putting material contaminated with high levels of dioxin directly onto allotments used for subsistence agriculture. The Kieselrot garden soil levels were much lower than the Byker samples. Other UK examples which could be considered include[3] Rechem Pontypool, Coalite, Derbyshire, ICI Runcorn and Walsall Birmingham but none presented such a large or widespread risk to the foodchain. The same is true for international incidents like Times Beach, Missouri or Seveso.

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[3] Levels of off site contamination were generally quite low compared with Byker– Walsall Copper Smelter 209 ngTEQ/kg, 54 ng TEQ /kg on farmland near Coalite and up to 115 ng TEQ /kg near ICI Runcorn – See ENDS 251, December 1995.

28. It is therefore the view of the Byker and Newcastle Waste Group that at the earliest possible stage the public should be informed about the extent of the current knowledge of the contamination – and of the limitations in the scope of the work undertaken to date.

29. The scope of the Newcastle University study has meant that it has been outside the remit of the report to review current exposure levels, particularly vulnerable individuals and exposure standards for dioxins. Annex A of this note considers the particular sensitivities of the unborn, babies and infants. The current study has also not presented any data for dioxin like PCBs nor for other halogenated, particularly brominated and chloro-bromo, dioxins. The relevance and the importance of obtaining data on these contaminants is addressed in Annex B to this note. No assessment has been made of the possible exposure to workers handling the ash (either in the plant or on allotments).

30. It is noted that the initial contamination by incinerator residues and the extent of the contamination has had to be drawn to the attention of the regulatory authorities by the public rather than being discovered as a result of regulatory action.

31. It is further noted that Environmental Data Services reported[4] "Regulatory foul-ups contributed to Byker ash affair" in which it is reported *that "The Agency says it knew nothing about the allotment deposits until told about them by a resident last August*." And "*The episode has put Contract Heat (sic) and the council in the spotlight - but the regulators' performance also does not withstand critical scrutiny. care" legislation on waste rather than breaches of the IPC authorisation. …… the regulators' own contribution -including their failure to unearth anything amiss during routine inspections of the incinerator - is a significant embarrassment."*

32. The role of the public in highlighting the incident was explicitly recognised by the Minister, Mr Mullin[5], when he replied that:

    The possibility that ash from the plant might have been used for the construction of footpaths and other purposes was first raised by a local resident during discussions with the Environment Agency on another mater in August 1999.

33. In these circumstances it is recommended that the appropriate response to the reduction in public confidence in the regulatory authorities requires full and frank public consultation and debate on the risks associated with the contamination and the appropriate regulatory and medical response to the incidents.

34. It is a matter of concern that the reports prepared by the Environment Agency and Food Standards Agency do not, for the reasons outlined below, take the conservative approach to the risks that they claim. In spite of this it is clear that allotment holders and their children have probably been exposed to intakes of dioxins which exceed World Health Organisation Tolerable Daily Intakes (TDIs) and are higher

Dioxin Contamination:

35. The Byker contamination with PCDD/PCDF is of the order of magnitude that would be expected in varying mixtures of bottom ash and fly ash from the refuse derived fuel

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[4] ENDS 304, May 2000

[5] Hansard 6 Jun 2000 : Column: 195W

plant[6]. In some cases, such as the Blucher allotment where levels of 9,500 ng I-TEQ/kg have been found, the contamination seems likely to be almost all fly ash rather than mixed ash. It is appreciated that some of the sampling has been undertaken by different agencies and that the protocols may not be available to the Newcastle University team. It is also not clear that all the data is yet available[7].

36. Congener/homologue profiles of dioxins can be helpful but they should be used carefully. It is not clear, for example, that the profiles can be used to make categorical statements in the report without giving considerably more thought to a range of factors including the ratios of fly ash to bottom ash, the date of depositing the ash and any subsequent photolytic dechlorination and/or evaporation of more volatile congeners (which could even vary depending upon the temperature on the date of sampling).

37. It is confusing[8] to assert that there is a "*bell shaped deposition pattern characteristic of deposition from industrial processes such as incinerator emissions*". The deposition from incinerator emissions includes fly ash and so reinforces the points in 16 above that it is important to consider the ratios of fly ash to bottom ash in any samples.

38. It is not clear that the high OCDD levels found in the 'Byker plus compost' congener profiles are compost derived. No evidence for this is presented - particularly at very high TEQ levels. Industrial wood combustion gives high OCDD (probably from Pentachlorophenol not pentachlorophenole as in the early full draft) as can industrial oil boilers such as Byker running on standby but there seems too little evidence to make conclusive statements on this and no reference is cited[9] to indicate that compost could possibly produce very high TEQ levels.

39. There does not seem to have been any analysis carried out on an ash sample from RDF that had been sold and burned in an open grate or greenhouse heater – it is very likely that the profile from these sources would be very different from that affected by an electrostatic precipitator and this may explain some of the variations.

Metal Contamination

40. The section headed 'Results and Recommendations for further action' in the draft executive summary compared the sample results with some German and ICRCL guidelines. The way that this has been done appeared to be inconsistent[10]. In particular:

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[6] There is little data on fly ash contamination levels as the Environment Agency has not generally been collecting this information for incineration processes. However LondonWaste report 10,800 ng/kg I-TEQ for fly ash from Edmonton in 1997[6]. Bottom ash concentrations are significantly lower and are reported to be around 50 ng/kg I-TEQ.

[7] The Environment Agency review, for example, refers to EA report No. B001608 dated 16th August 2000 which is not in the public domain.

[8] See, for example, *Cleverly, D.; Schaum, J.; Schweer, G.; Becker, J.; Winters, D. 1997. The congener profiles of anthropogenic sources of chlorinated dibenzo-p-dioxins and chlorinated dibenzofurans in the United States. presentation at Dioxin '97, the 17th International Symposium on Chlorinated Dioxins and Related Compounds, held August 25-29 in Indianapolis, IN, USA. Short paper in, Organohalogen Compounds, Volume 32:430-435.* for a more detailed assessment of congener profiles form varying industrial sources

[9] I have not been able to find any support for these levels in the literature

[10] The final report may have been changed to reflect some, or all, of these comments – which were first submitted in November 2000 but no revised draft has been provided before the final version of these comments was written.

- The results sometimes refer to the German guidelines (Arsenic 40 vs. 10, Cadmium 2 vs. 3, chromium) and sometimes to ICRCL (copper 130 vs. 50, mercury 1 vs. 2 , lead 500 vs. 200-300). For Nickel the 50 – 70 range reflects a combination of the Dutch and ICRCL guidelines.
- For Arsenic, copper and lead the higher guidelines have been taken.
- Where the guideline is given as a range the higher end of the range has been taken as the threshold for further sampling to assess remediation.
- Although the Dutch guidelines are listed in Table 2 these (generally lower) values are not considered as the basis for future assessments. In the case of lead this means that levels of contamination ten times higher than the Dutch guidelines are being left without further consideration.
- There is no explanation for the use of 500 mg/kg as a threshold for further investigation of zinc. This is 2.5 times the Dutch levels; and 1.6 times both the German and ICRCL guidelines. If a significantly higher threshold is to be included it should be fully explained and properly justified.

41. It is suggested on the basis of comparison with average ash concentrations that there is "no clear link between soil contamination and deposition of Byker ash". The basis for this assessment and conclusion is of questionable validity – the ash is heterogeneous and heavy metal contamination will vary over time – as will leaching of the more soluble metal species such as chlorides. The linkage would not, therefore, be expected to be as simplistic as suggested in the summary but would depend upon initial ash concentrations, time of deposit and subsequent leaching, level of mixing of fly ash and bottom ash etc etc. At least the report should include the maximum and minimum ash concentrations in Table 2 – comparison with simply a median level is quite unrealistic.
42. The FSA have attempted to apply standards to allotments which have been derived for agriculture[11]. This is not considered to be appropriate, as standards for allotments should be significantly tighter for allotments than for agricultural land (Table 1 in the Executive Summary illustrates this for established thresholds). This is because agricultural produce from a particular location is rarely, if ever, consumed as the bulk of a subsistence diet. Produce from allotments, by contrast can provide the bulk of the family food intake for allotment holders.
43. There is a long time lag in introducing standards for land contamination and it is therefore recommended that the appropriate threshold to use is the lower (more protective) level from ICRCL or other accepted authorities. This approach is also recommended in the light of the reduced levels of public confidence in the regulatory authorities noted above and considering the mixture of metals and PCDDs/PCDFs present on these sites.

Assessment of Exposure via Egg Intakes

44. The only pathways for human exposure to dioxins considered in the report is via egg consumption. The assessment of impacts on body burdens has only been carried out for 35 and 70 kg persons but not for small children/babies who are likely to be at greater risk from exposure. Nor has any assumption been made about dioxin like PCBs in the eggs. Many people with hens are likely to eat, and have eaten for some time, more than one egg per day – this should be acknowledged. A 60 kg person eating one egg/ day would

[11] Such as from the MAFF Code of Good Agricultural Practice for the Protection of Soil or from the Sludge (Use in Agriculture) Regulations 1989 (SI.1989No 1263)

exceed the upper limit WHO TDI without any consideration of PCBs. It is recommended that this should be included in the Key Findings. It should also be made clear in the key findings what proportion of the Byker Eggs exceed EU standards as per fig 11 in the draft full report. It is not precautionary to consider average background exposure when it is known that some diets such as high fish consumption or use of fish oil supplements dramatically increase dioxin intakes above the average intake levels.

45. The key finding that "*regular consumption of Byker eggs is likely to have contributed to an elevation of the body burden of PCDD/PCDF to levels equivalent to those in the general population approximately 10 years ago*" is inappropriate. It is clear that levels ten years ago were far too high and that there is good evidence that even current levels are affecting the health of some people. It is quite wrong to attempt to normalise a level of contamination by comparing it with an historically unacceptable situation. Furthermore this makes no allowance for the consumption of more than one egg per day nor for very long-term exposure or for those with higher dioxin levels in their diet from other sources. The appropriate comparator is the lower end of the WHO TDI and the more stringent standards of the Scientific Committee on Food (see below).

46. The Environment Agency report does not consider exposures from eggs or chickens[12], unlike the earlier draft which said:

    "we consider it [exposure from the consumption of contaminated poultry and contaminated eggs] to be an important pathway for some of the allotment sites and therefore a further model (called CalTOX) was used to provide additional daily intakes from these two routes."

    And

    "There may also be the possibility that the chickens could have access/come in contact with the path (and feed on the grains, worms etc. along the path). If they did, then ingestion of dioxins from the soil may become more important for the soil to chicken/egg to human exposure pathway, meaning that the eating of eggs (or the chickens themselves) could become a main exposure pathway for people"

47. In is difficult to see how the Environment Agency can justify the claim that the risk assessment is 'highly conservative' when the pathway the Agency has accepted is 'important' and which on the basis of the FSA and Newcastle data[13] is indeed 'a main exposure pathway for people' has been completely omitted. This serious omission must bring into question the validity of the conclusions reached in the Environment Agency review.

48. The FSA assessment considers at children aged between 10-15 years and weighing 43.4 kg. These children are described as 'schoolchildren' in the results tables. It is unclear why the assessment has not been extended to include younger children and babies (for whom eggs can be amongst their first foods). If these more vulnerable children were included then the daily intakes would be similar to older children - particularly when their parents had a ready source of eggs from their own poultry. Because of the lower bodyweights the daily intakes could be two or more times the intakes calculated in the FSA tables 2a and 2b. There is no reason to believe that those who keep bantams, at

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[12] on the grounds that "None of the sites looked here were used to keep poultry. The Food Standards Agency has included a discussion of dioxin contamination of eggs and poultry on other allotments sites in its comments on the main report". No explanation is given for the omission of any sites with poultry as part of the Environment Agency assessment nor for the omission of the earlier sections emphasising the importance of this pathway.

[13] There are several sites where poultry seem to have had direct access to ash

Branxton A, for example, would not eat all the eggs from those birds or that they would necessarily mix the intake of bantam eggs with other eggs.

49. The FSA estimates include a scenario with dioxin like PCBs although only equal to the level of dioxins whilst in practice the PCB levels are often greater than equivalent dioxin levels.

50. The consequence of omitting younger children from the assessment is that actual exposures from eggs alone may have been four or more times a 10 pg/kg bw/day TDI or 10 or more times the WHO upper limit of 4 pg/kg bw/day and 40 or more times the November 2000 intake recommendations of the EU Scientific Committee on Food (see below).

Dioxin Exposure from Meat

51. None of the reports has made any assessment of the additional exposure from the consumption of poultry reared on the allotments. This pathway may be important. ENDS[14] reports that a routine survey of dioxin levels in meat in south-western Germany uncovered a highly contaminated pheasant originating from the UK. The bird contained 235pg of dioxins per gram of fat, expressed as a toxic equivalent (TEQ) of the most toxic dioxin, 2,3,7,8 TCDD. The report said:

    "This is over 300 times the level found in farmed chicken, and over 50 times the average level in other game birds. The source of the bird was close to the Coalite works in Derbyshire where off site contamination levels are much lower than in Byker.

    The levels found were clearly a health concern. A 100g portion of the contaminated pheasant would contain 587 times the maximum tolerable daily dioxin intake recommended by the World Health Organisation."

52. It is recommended that this pathway should be reviewed as a matter of urgency.

Dioxin Exposure via Soil Intake

53. No reference is made to Pica[15] behaviour in any of the reports. However this can be a very significant exposure route for affected children at the levels found on these allotments – eating, for example, unwashed radishes grown on an allotment could extend this exposure to non-pica children. This is a serious omission which should be corrected. The levels of dioxin contamination in the allotments, parks, recreational land and footpaths are those at which a child with 'Pica' behaviour very significantly exceeds even a 10 pg/kg bw/day TDI by contamination from soil alone.

54. The threshold values in the 'Public Health Recommendations' section of the 24/5/00 Stage 1 report, copied below, were determined pre-1995 before WHO revised (in 1998) downward dioxin TDIs. As TDIs have been reduced by a factor of ten (it is appropriate to use the lower end of the range as WHO make clear that there is a long term objective to reduce intakes still further below 1 pg/kg bw/day) these values should at least be caveated as being potentially non protective. The levels should in any case be taken as I-TEQ or, preferably, WHO TEQ (about 10% higher than I-TEQ) including dioxin like PCBs. This is likely to have a significant impact on the numbers of sites which exceed the 40 ng/kg and 100 ng/kg thresholds.:

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[14] ENDS January 2000, Issue No. 300

[15] High levels of soil eating

"The public health recommendations for PCCD/PCDF agreed in the protocol were based on those established by the joint working group on dioxins in Germany for PCDD/PCDF levels in soil (3):

< 5ng I-TEQ/kg soil target value,

5-<40 ng/kg I-TEQ/kg soil unrestricted cultivation of food stuff, avoidance of critical land use

40-<100 ng/kg I-TEQ/kg soil limitation to defined agricultural and horticultural use, unlimited cultivation only of plants with minimum dioxin transfer

>100 ng/kg I-TEQ/kg soil remediation in playgrounds (sealing, decontamination or soil exchange)

>1000 ng/kg I-TEQ/kg soil remediation in residential areas

The protocol outlined that our interpretation of these limits in the local context would be to advise against the holding of poultry if levels were found to be 5-<40ng/kg I-TEQ, and to advice against the consumption of root vegetables if levels were 40-<100ng/kg."

55. On the basis of this advice it is quite possible that the existing levels of contamination could already have led to elevated body burdens of dioxin. The remit of the report does not cover collection of lipid samples of exposed local residents and allotment holders who may already be contaminated with dioxins and hence no data is available. Nor have there been any proposals, so far as we are aware, for monitoring tests to determine body burdens or breast milk contamination with dioxins for these residents.

56. The most obvious exposure pathways are through food uptake and is via soil ingestion. The USEPA outline the issues associated with soil eating in their Exposure Factor Handbook[16]:

    "The ingestion of soil is a potential source of human exposure to toxicants. The potential for exposure to contaminants via this source is greater for children because they are more likely to ingest more soil than adults as a result of behavioral patterns present during childhood. Inadvertent soil ingestion among children may occur through the mouthing of objects or hands. Mouthing behavior is considered to be a normal phase of childhood development. Adults may also ingest soil or dust particles that adhere to food, cigarettes, or their hands. Deliberate soil ingestion is defined as pica and is considered to be relatively uncommon. "

57. Allotment holders have confirmed at public meetings that they have had to remove clinker from the mouths of their children whilst they have been playing on allotment paths.

58. It is surprising, and worrying, therefore that the Environment Agency model does not take PICA behaviour into account on the allotments – exposures from soil are taken as only about 0.5% of total intake – lower levels than even the Dinnington Park assessment.

59. Furthermore the Environment Agency risk and exposure assessment for Byker uses a maximum soil concentration of only 321 ng I-TEQ/kg. This is far lower than many of the contaminated sites and even Environment Agency sampling cited in the main report indicates levels of more than seven times higher than this (2418 ng I-TEQ/kg for Walkergate B allotment. It is not clear, therefore why the Environment Agency have relied upon such low results a level at least ten – and probably twenty – times higher would have been more appropriate.

60. The Agency model uses the following parameters of exposure[17]:

    Allotments

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[16] Exposure Factor Handbook, p 4-1, USEPA August 1996

[17] Reported in 'Notes from the Byker Steering Group meeting, 16th November 2000'

Ingestion of soil	*0.5% of all intake*
Consumption of vegetables	*98%*
Dust inhalation	*Negligible*
Dermal (skin) contact	*1.5%*
Parks/Recreational Land	
Ingestion	*73%*
Dermal (skin) contact	*27%*

61. Environment Agency assessments of PICA on other sites have tended to use very low levels of consumption. A recent Environment Agency IPC 'decision document' for the Portsmouth incinerator[18], for example, suggests that:

    "Other possible ingestion of dioxins may occur from eating soil. This is a rare syndrome known as "pica". Studies into this condition have identified a mean ingestion rate of 21 mg/kg/day".

62. An arithmetic check on the Dinnington Park results from the Environment Agency model indicates that the levels of soil ingestion used by the Environment Agency in modelling the Byker intakes are very likely the same (321 ng/kg gave 9 pg/kg intake, if 73% of this is due to ingestion then the ingestion rate is about 21 mg/day). It is clear that Pica exposure has not been considered on the allotment sites even though children are likely to play on paths whilst their parents tend the allotments and gardeners have reported soil eating behaviour in their own children whilst on the allotments.

63. The Environment Agency approach cannot be considered to be correct – and it is certainly not precautionary. Pica may reasonably be described as a rare syndrome but soil ingestion is common and the levels quoted relate to normal ingestion behaviour and not to Pica where ingestion levels are very much greater.

64. USEPA recommend that for Pica levels of intakes are assumed to be 10g/day. Pica intake is therefore closer to 1 g/kg/day i.e. nearly 50 times greater than the Environment Agency suggest.

65. Even these levels may not represent the highest exposures. More recent work by Calabrese[19], for example, reports that:

    "Several soil ingestion studies have indicated that some children ingest substantial amounts of soil on given days. Although the EPA has assumed that 95% of children ingest 200 mg soil/day or less for exposure assessment purposes, some children have been observed to ingest up to 25-60 g soil during a single day".

66. It therefore seems that whilst about 5% of all children will ingest levels higher than those assumed by the Environment Agency some children will be exposed to more than 100 times those levels.

67. Pica is not just a short-term exposure - over the first five years it is estimated that the average child affected by pica consumes about 8,000 g of soil although consumption is not equally spread over the period[20].

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[18] Decision document for IPC Application No BF9409, undated, page 73

[19] Soil Ingestion: A Concern for Acute Toxicity in Children Edward J. Calabrese, Edward J. Stanek, Robert C. James, and Stephen M. Roberts Environmental Health Perspectives Volume 105, Number 12, December 1997

[20] See, for example, R. D. Kimbrough and others, "Health implications of 2,3,7,8-tetra-chloro-dibenzo-dioxin (TCDD) contamination of residential soil," JOURNAL OF TOXICOLOGY AND ENVIRONMENTAL HEALTH Vol. 14 (1984), pgs. 47-93

68. Whilst Pica may be relatively uncommon the USEPA cite[21] Bruhn and Pangborn (1971) reported the incidence of pica for "dirt" to be 19 percent in children, 14 percent in pregnant women, and 3 percent in non-pregnant women. However, "dirt" was not clearly defined.

69. A model-based prediction developed by Calabrese[22] indicated that "*the majority (62%) of children will ingest >1 g soil on 1-2 days/year, while 42% and 33% of children were estimated to ingest >5 and >10 g soil on 1-2 days/year, respectively*". They concluded that these estimates were qualitatively significant "*because they suggest that soil pica is not restricted to a very small percentage of the normal population of children, but may be expected to occur in a sizable proportion of children throughout the course of the year*". They also concluded that "*if soil pica is seen as an expected, although highly variable, activity in a normal population of young children, rather than an unusual activity in a small subset of the population, its implications for risk assessment become more significant*."

70. The draft USEPA dioxin review suggests:

    " Soil ingestion for older children and adults were not considered, which may have underestimated lifetime soil ingestion exposures. Pica soil ingestion patterns were not evaluated in this assessment. The ingestion rates (200 mg/day for central scenarios and 800 mg/day for high end scenarios, during ages 2-6) considering this appear reasonable."

71. The Environment Agency in the Portsmouth incinerator decision document calculated possible exposures using this 'high-end' figure:

    "an average ingestion rate of 800mg/day was suggested as representing the upper limit for children with a high tendency to eat soil. "

72. Recalculating the Environment Agency results for Byker on this basis gives an ingestion intake of 25.7 pg/kg/day from ingestion alone. Even the inconsistency between different Environment Agency regions, therefore, would indicate significant cause for concern.

73. The Environment Agency report (and that of the FSA[23]) have been improved from the previous versions by making proper reference to the WHO TDIs rather than simply comparing the (unreasonably low) exposure results with a Tolerable Daily Intake of 10 pg/kg/day[24].

74. The old standard is no longer scientifically robust and is also inappropriate in policy terms. The Government has confirmed[25], that the new WHO standard will also be used as a comparator for exposure data.

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[21] Exposure Factor Handbook, p 4-18, USEPA August 1996

[22] Soil Ingestion: A Concern for Acute Toxicity in Children, Edward J. Calabrese, Edward J. Stanek, Robert C. James, and Stephen M. Roberts Environmental Health Perspectives Volume 105, Number 12, December 1997

[23] The Food Standards Agency is reported in the 7th November minutes of the Working Group as indicating that the Agency is "*still using the UK standards and questioning evidence for the WHO standards*".

[24] On the grounds that the 1998 WHO TDI of 1-4 pg TEQ/kg bw/day "has not been endorsed by the Department of Health. As the Agency takes advice from the Department of Health we must use values recommended by them as being the most authoritative for the UK at the moment."

[25] Hansard 22 Jun 1999 : Column: 335

Mr. Caton: To ask the Secretary of State for the Environment, Transport and the Regions what assessment he has made of the revised World Health Organisation tolerable daily intake for dioxins (1-4 pg TEQ/Kg bw day). [87843]

Mr. Meale: The Department of Health Advisory Committee on the Toxicity of Chemicals in Food, Consumer Products and the Environment (COT) has not yet had the opportunity to review the data used by the recent WHO consultation which recommended the Tolerable Daily Intake (TDI) for dioxins of 1-4 pg TEQ/kg

75. Using levels of soil uptake adopted in other Environment Agency reports –such as for dioxin contamination around the Portsmouth Incinerator -indicates that the ingestion intakes can be more than 2.5 times even the 10 pg/kg/bw level, more than 6 times the WHO upper limit TDI more than 25 times greater than the WHO target TDI. It is important to note also that this:

- Does not allow for any other intake of dioxin or dioxin like substances, such as PCBs, and mixed halogenated dioxins (see below) either from food or as contaminants of the ingested soil
- Egg contamination is not calculated together with the other uptakes even though allotment holders with hens are likely to be exposed to all pathways.
- Assumes that the measured concentration is the highest level to which any child would be exposed – whilst in practice it is unlikely that the sampling would be at precisely the point of highest concentration.
- Makes no allowance for any increased body burdens that children in the area may carry as a result of the long term exposure of their parents to high levels of dioxin emissions from the incinerator or from their allotments.

76. Adding even low levels of dietary intake increases the exceedances.

Levels of Dioxin Intakes compared with Tolerable Daily Intakes

77. The levels of exposure that can be calculated for those exposed to Byker ash are of concern when compared with the impacts upon which the WHO standards[26] are based and the low margins incorporated in even the WHO standards:

STUDY	RESPONSE (LOAELs)	MATERNAL BODY BURDEN* (ng/kg bw)	RELATED HUMAN EDI (pg/kg bw/day)
Gray et al., 1997a	RATS: decreased sperm count in offspring	28	14
Gehrs et al., 1997b; Gehrs & Smailowicz 1998	Immune suppression in offspring	50	25
Gray et al., 1997b	Increased genital malformations in offspring	73	37
Schantz and Bowman, 1989	MONKEYS: Neurobehavioural (object learning) effects in offspring	42	21
Rier et al., 1993	Endometriosis	42	21

78. These are adverse effects and are within, and in some cases lower than, the range of expected exposures.

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bw/day. Therefore this TDI has not been adopted formally by the Government. The COT will undertake a review when a full report of the consultation is available. In the interim, current exposure data will be considered using both the current UK TDI (10 pg TEQ/kg bw/day) and the recently recommended WHO TDI. [my emphasis]

[26] Table 4 WHO Assessment of the health risk of dioxins: re-evaluation of the Tolerable Daily Intake (TDI)Executive Summary http://www.who.int/pcs/pubs/dioxin-exec-sum/exe-sum-final.html

79. Both reports have now been modified to include the recent developments in Europe. ENDS reports[27] for example, that "*Scientific advisers to the European Commission have recently called for renewed efforts to cut human exposures to PCBs and dioxins. They suggest yet more emphasis on cutting emissions - and effectively recommend that fish products of European origin be removed from animal feeds because they are too contaminated.*"

    The idea of limits on dioxins and PCBs in food surfaced following the Belgian dioxin crisis - the largest of several dioxin-related food scares in Europe. Food is the most important source of human exposure to dioxins, and a 1999 report by AEA Technology, ordered by the Commission and the Department of the Environment, Transport and the Regions, concluded that many EC citizens - and particularly breast-fed infants - have exposures above international safety limits (ENDS Report 298, pp 3-4).

    Against this background, the Commission asked the Scientific Committee on Food (SCF) to advise on the scientific basis for limits on dioxins and PCBs in food. It also asked its Scientific Committee on Animal Nutrition (SCAN) to advise on the contribution of feeds to dioxin and PCB contamination in human food. Both committees' opinions were released in November.

    The SCF established a tolerable weekly intake of dioxins and PCBs of 7 picograms per kilogram bodyweight, expressed as a toxic equivalent (TEQ) of the most toxic dioxin, 2,3,7,8-TCDD. This is equivalent to the lower bound of the World Health Organisation's recommended tolerable daily intake of 1-4pg TEQ/kg.

80. Now that WHO and, more recently, the EU Scientific Committee on Food have established much more stringent standards than those still relied upon in the UK it is clear that the exposure levels that arise from Byker are very greatly in excess of the TDIs.

Current Exposures from Food:

81. Whilst average exposure to dioxins in the UK is likely to be within the WHO targets there are certain particularly vulnerable and more highly exposed groups. The Committee on Toxicity of Chemicals in Food Consumer Products and the Environment (COT [28]has shown that those taking fish oil supplements can have high intakes:

Age	Daily intake
Breast fed infants - up to	181 pg TEQ kg bw / day.
Toddlers $1\,^1/_2$ - $2\,^1/_2$ yrs	19.6 pg TEQ kg bw / day.
Toddlers $3\,^1/_2$ - $4\,^1/_2$ yrs	25.3 pg TEQ kg bw / day.
Schoolchildren	13.7 pg TEQ kg bw / day.
Adults	10.8 pg TEQ kg bw / day.

    These results exceed even the 10 pg / kg / bw standard for the whole lifetime of the more exposed adults. The levels are particularly worrying when compared with the WHO targets of less than 1.0 pg TEQ / kg bw / day .

82. Those who eat large quantities of fish may already be highly exposed from that source. MAFF / FSA have reported that consuming fish more than twice / week can lead to the WHO upper limits being exceeded – simply adding average exposures onto calculated intakes, as per the Environment Agency assessment therefore ignores the likelihood that

[27] ENDS December 2000, Issue No. 311

[28] Statement by the committee on toxicity of chemicals in food, consumer products and the environment on the health hazards of polychlorinated biphenyls April 1997

some allotment holders and highly exposed children will also have higher than average dioxin intakes from other sources.

Uncertainties:

83. The Key findings box should include the statement on page 4 that "*it needs to be pointed out that hot spots can not be ruled out on the basis of the analyses that were carried out*."

Missing Data and information:

84. There is a paucity of good UK data on background levels of contamination of eggs. We understand that the FSA has collected some other data on egg contamination , including for free range eggs, but this does not seem to have been placed in the public domain yet. This data should be made available so that that comparative levels can be more fully assessed.

85. The report should indicate that there are likely to be other sites around Newcastle which are contaminated but which have not yet been identified. Future investigations should include areas where ash has been tipped / utilised and also where RDF was sold and the residues have been disposed of (possibly with even higher dioxin levels as the combustion is likely to have been even less efficient). It is significant that many other uses[29] of the processed fuel seem to have been tried over the past 20 years and the distribution of the waste that has been caused by this needs detailed investigation – some of the materials will have been burned and will present ash problems in other cases it is likely that the raw materials will be contaminated with metals and, possibly, products of incomplete combustion from processing line fires.

Conclusions

86. The current risk / exposure assessments by both the Environment Agency and the Food Standards Agency do not appear to be protective given the higher levels of contamination recorded on some sites, the uncertainties associated with the extent of contamination; the exposures to mixtures of dioxins and heavy metals etc.

87. In at least some of the cases the allotments will need to be cleared completely with new topsoil being supplied. It is not protective to use the current risk assessments to justify leaving the contaminated material in place.

88. More data is required for dioxin like PCBs, halogenated dioxins and other products of incomplete combustion.

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[29] World Bank Technical Paper no 37, 1985 reports, for example, that:

"An attempt is being made to expand the market for the pelletized fuel, but, as is the case elsewhere, many users have been reluctant to buy a new and as yet unproven fuel. Other uses of the material have been explored, for example, there have been some test runs using the fuel fraction in an unpelletized form as core material in the manufacture of chipboard or fiberboard. The pellets themselves have been used for making paper for carpet underpadding and even for bingo tickets, but only on an experimental basis.

Annex A - Prenatal Intakes, babies and infants:

1. The unborn and breast fed children may be the most vulnerable to the impacts of dioxin contamination. Particularly where young mothers have been exposed for some time to eggs and other products from allotments. This group has not been considered by either the Environment Agency nor by the FSA.
2. Weisglas-Kuperus[30] reported that

   "… [P]renatal dioxin/PCB exposure can cause growth deficits, motor dysfunction, neurodevelopmental disabilities, learning problems and hearing disorders in humans. … PCBs and dioxins are present in background exposed mothers and their infants at concentrations that might be toxicologically relevant. These chemicals may interfere with endogenously produced hormones, neurotransmitters and growth factors and may change the course of prenatal human development. Cognitive and neuromotor changes, differences in immune response, reduced birthweight, microgenitalism, reduced fertility and change in the male/female ratio may all be associated with human prenatal PCB/dioxin exposure."
3. The Committee on Toxicity of Chemicals in Food Consumer Products and the Environment[31]. This indicates that young babies who are breast-fed may be exposed to as much as 170 times the WHO target tolerable daily intake of dioxin like chemicals in their body even without fish oil supplements.
4. Although the report stated the opinion that on balance of risk it was still preferable to breast-feed, the World Health Organisation published a report nearly five years ago on levels of PCBs, dioxins and furans in human milk which concluded that:

   "*a wealth of new information has become available since the last assessment of possible risks to infants*" and that "*the lowest observable adverse effects levels (LOAELs) for developmental, neurobehavioural and reproductive endpoints, based on body burdens of TEQs in animals, may be in the range of current background human body burdens in certain segments of the population."*
5. It seems unlikely that the official advice will be particularly reassuring to parents who are concerned that their babies are receiving hundreds of times more dioxin and PCBs than WHO intake standards and around 6,000 times more than the US ADI[32]. Those parental concerns are supported by the scientific literature, as shown below, and the approach recommended by the WHO report is likely to be more appealing. WHO concluded that rather than restricting breast feeding the:

   "Primary measures to control and reduce the input of these chemicals into the environment were considered the most effective way to limit and minimize exposure"
6. Patatndin[33] et al, for example investigated food habits from early ages until reproductive age (25 years) which, they said, important in order to assess exposure risk for the next generation. They said:

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[30] Weisglas-Kuperus, N. 1998. Neurodevelopmental, immunological and endocrinological indices of perinatal human exposure to PCBs and dioxins. Chemosphere 37: 1845-1853.

[31] Op Cit

[32] For example, if an infant was breast-fed for one year on milk containing 20 ppt TEQ its average daily dose would be 60 pg TEQ / kg / day

[33] Dietary Exposure to Polychlorinated Biphenyls and Dioxins from Infancy Until Adulthood: A Comparison Between Breast-feeding, Toddler, and Long-term Exposure Svati Patandin, Pieter C. Dagnelie, Paul G.H. Mulder, Eline Op de Coul, Juul E. van der Veen, Nynke Weisglas-Kuperus, and Pieter J.J. Sauer Environmental Health Perspectives Volume 107, Number 1, January 1999

"As in other industrialized countries in Western Europe, contamination of breast milk with PCBs and dioxins in The Netherlands has led to considerable public concern. The Dutch government launched a longitudinal neurodevelopmental study in 1989 aimed at investigating the adverse effects of background exposure to PCBs and dioxins on growth and development of healthy full-term infants. The period of observation was expanded to pre-school age in a European Community-funded European collaborative project.

Previously reported results showed that in Dutch infants, lower birth weights and decreased postnatal growth, delays in psychomotor development and neurodevelopment, alterations in thyroid hormone and immunological status were associated with prenatal PCB and dioxin exposure rather than with lactational exposure.

Given the results from the above mentioned studies and the bioaccumulation of PCBs and dioxins in the food chain, an estimate of dietary exposure to PCBs and dioxins from early ages until reproductive age is important in order to assess exposure risk for the next generation."

7. The study found that breast-feeding for 6 months contributed to the cumulative PCB / dioxin TEQ intake until 25 years of age, 12% in boys and 14% in girls. They reported that the daily TEQ intake per kilogram body weight is 50 times higher in breast-fed infants and three times higher in toddlers than in adults.
8. Other studies include:
   - Nagayama[34] et al reporting that "*our study suggests that exposure to background levels of the highly toxic organochlorine compounds [dioxins] through the breast milk influences the human neonatal immune system*."
   - Nagayama[35] in a second study reported that "*exposure to background levels of the highly toxic organochlorine chemicals through the breast milk may cause some effects on thyroid hormone status…*"
   - A Finnish study of the teeth of breastfed children in which Alaluusua[36] et al. found, "*Defects were clearly associated with the total exposure to toxic and furans*." Noting that the "*high frequency of hypomineralised dental defects among normal children may be a sign of exposure to PCDD/Fs [dioxins*]," they proposed that such defects "*may be the best available indicator of dioxin exposure*."
9. Koppe[37] et al found that the concentration of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in the fat of breast milk of 14 Dutch mothers varied from 5.35 to 17.0 ng / kg (nanograms per kilogram fat). Expressed as toxic equivalents the concentrations of the 17 different congeners of dioxin and furans in the fat of the breast milk were between 29.85 and 92.88 ng / kg. These levels are sufficient to induce enzyme formation in the livers of infants. Dioxin induces the same enzyme production in the liver as phenobarbital; therefore,

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[34] Nagayama, J., Tsuji, H., Iida, T., Hirakawa, H., Matsueda, T., Okamura, K., Hasegawa, M., Sato, K., Ma H., Yanagawa, T., Igarashi, H., Fukushige, J., Watanabe, T. 1998. Postnatal exposure to chlorinated dioxins and related chemicals on lymphocyte subsets in Japanese breast-fed infants. Chemosphere 37:1781-7

[35] Nagayama, J., Okamura, K., Iida, T., Hirakawa, H., Matsueda, T., Tsuji, H., Hasegawa, M., Sato K., Ma, H., Yanagawa, T., Igarashi, H., Fukushige, J., Watanabe, T., 1998. Postnatal exposure to chlorinated dioxins and related chemicals on thyroid hormone status in Japanese breast-fed infants. Chemosphere 37:1789-93.

[36] Alaluusua, S., Lukinmaa, P., Torppa, J., Tuomisto, J., Vartiainen, T. 1999. Developing teeth as biomarker of dioxin exposure. Lancet 353:206.

[37] Koppe JG; Pluim HJ; Olie K; van Wijnen J Breast milk, dioxins and the possible effects on the health of newborn infants. Department of Neonatology, Academic Medical Centre, University of Amsterdam, Netherlands. Sci Total Environment

because phenobarbital affects fat-soluble vitamins, such as vitamins D and K, a similar response was anticipated in response to exposure to dioxin. Consequently, one aim of this study was to investigate blood coagulation parameters. They found a statistically significant relationship with the mean concentration of TCDD in the breast milk of mothers whose babies suffered from bleeding problems.

10. It is clear that the exposures to dioxin and dioxin like compounds from the Byker contamination is likely to result in exposures which are in the range of real, and serious, health impacts.

Annex B - Others Contaminants of Concern

1. Kay and Steiglitz published a paper in Chemosphere[38] listing just some of these emissions from municipal waste incineration. Many, possibly all, of these contaminants, plus the majority of others which could not be recognised by the GC-MS library are likely to be present in the fly ash spread around Newcastle. It is important to recognise, therefore that the exposure to residents and allotment holders is not to single chemicals but to an extremely complicated mixture of hazardous chemicals.

   Whilst the risks associated with mixtures of these chemicals have not been assessed it is likely that the concentrations will have varied significantly with time given that waste is such a variable feedstock. Even if the chemicals were identified little, sometimes nothing, is known about the toxicology of the majority of them.

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[38] K. Jay and L. Steiglitz, "Identification and Quantification of Volatile Organic Components in Emissions of Waste Incineration Plants," CHEMOSPHERE Vol. 30, No. 7 (1995), pgs. 1249-1260 identified the following volatile organic chemicals emitted from a municipal waste incinerator: pentane; trichlorofluoromethane; acetonitrile; acetone; iodomethane; dichloromethane; 2-methyl-2-propanol; 2-methylpentane; chloroform; ethyl acetate; 2,2-dimethyl-3-pentanol; cyclohexane; benzene; 2-methylhexane; 3-methylhexane; 1,3-dimethylcyclopentane; 1,2-dimethylcyclopentane; trichloroethene; heptane; methylcyclohexane; ethylcyclopentane; 2-hexanone; toluene; 1,2-dimethylcyclohexane; 2-methylpropyl acetate; 3-methyleneheptane; paraldehyde; octane; tetrachloroethylene; butanoic acid ethyl ester; butyl acetate; ethylcyclohexane; 2-methyloctane; dimethyldioxane; 2-furanecarboxaldehyde; chlorobenzene; methyl hexanol; trimethylcyclohexane; ethyl benzene; formic acid; xylene; acetic acid; aliphatic carbonyl; ethylmethylcyclohexane; 2-heptanone; 2-butoxyethanol; nonane; isopropyl benzene; propylcyclohexane; dimethyloctane; pentanecarboxylic acid; propyl benzene; benzaldehyde; 5-methyl-2-furane carboxaldehyde; 1-ethyl-2-methylbenzene; 1,3,5-trimethylbenzene; trimethylbenzene; benzonitrile; methylpropylcyclohexane; 2-chlorophenol; 1,2,4-trimethylbenzene; phenol; 1,3-dichlorobenzene; 1,4-dichlorobenzene; decane; hexanecarboxylic acid; 1-ethyl-4-methylbenzene; 2-methylisopropylbenzene; benzyl alcohol; trimethylbenzene; 1-methyl-3-propylbenzene; 2-ethyl-1,4-dimethylbenzene; 2-methylbenzaldehyde; 1-methyl-2-propylbenzene; methyl decane; 4-methylbenzaldehyde; 1-ethyl-3,5-dimethylbenzene; 1-methyl-(1-pro-penyl)benzene; bromochlorobenzene; 4-methylphenol; benzoic acid methyl ester; 2-chloro-6-methylphenol; ethyldimethylbenzene; undecane; heptanecarboxylic acid; 1-(chloromethyl)-4-methylbenzene; 1,3-diethylbenzene; 1,2,3-trichlorobenzene; 4-methylbenzyl alcohol; ethylhex anoic acid; ethyl benzaldehyde; 2,4-dichlorophenol; 1,2,4-trichlorobenzene; naphthalene; cyclopentasiloxanedecamethyl; methyl acetophenone; ethanol-1-(2-butoxyethoxy); 4-chlorophenol; benzothiazole; benzoic acid; octanoic acid; 2-bromo-4-chlorophenol; 1,2,5-trichlorobenzene; dodecane; bromochlorophenol; 2,4-dichloro-6-methylphenol; dichloromethylphenol; hydroxybenzonitrile; tetrachlorobenzene; methylbenzoic acid; trichlorophenol; 2-(hydroxymethyl) benzoic acid; 2-ethylnaphthalene-1,2,3,4-tetrahydro; 2,4,6-trichlorophenol; 4-ethylacetophenone; 2,3,5-trichlorophenol; 4-chlorobenzoic acid; 2,3,4-trichlorophenol; 1,2,3,5-tetrachlorobenzene; 1,1'biphenyl (2-ethenyl-naphthalene); 3,4,5-trichlorophenol; chlorobenzoic acid; 2-hydroxy-3,5-dichlorobenzaldehyde; 2-methylbiphenyl; 2-nitrostyrene(2-nitroethenylbenzene); decanecarboxylic acid; hydroxymethoxybenzaldehyde; hydroxychloroacetophenone; ethylbenzoic acid; 2,6-dichloro-4-nitrophenol; sulphonic acid m.w. 192; 4-bromo-2,5-dichlorophenol; 2-ethylbiphenyl; bromodichlorophenol; 1(3H)-isobenzofuranone-5-methyl; dimethylphthalate; 2,6-di-tertiary-butyl-p-benzoquinone; 3,4,6-trichloro-1-methyl-phenol; 2-tertiary-butyl-4-methoxyphenol; 2,2'-dimethylbiphenyl; 2,3'-dimethylbiphenyl; pentachlorobenzene; bibenzyl; 2,4'-dimethylbiphenyl; 1-methyl-2-phenylmethylbenzene; benzoic acid phenyl ester; 2,3,4,6-tetrachlorophenol; tetrachlorobenzofurane; fluorene; phthalic ester; dodecanecarboxylic acid; 3,3'-dimethylbiphenyl; 3,4'-dimethylbiphenyl; hexadecane; benzophenone; tridecanoic acid; hexachlorobenzene; heptadecane; fluorenone; dibenzothiophene; pentachlorophenol; sulphonic acid m.w. 224; phenanthrene; tetradecanecarboxylic acid; octadecane; phthelic ester; tetradecanoic acid isopropyl ester; caffeine; 12-methyltetradecacarboxylic acid; pentadecacarboxylic acid; methylphenanthrene; nonedecane; 9-hexadecene carboxylic acid; anthraquinone; dibutylphthalate; hexadecanoic acid; eicosane; methylhexadecanoic acid; fluoroanthene; pentachlorobiphenyl; heptadecanecarboxylic acid; octadecadienal; pentachlorobiphenyl; aliphatic amide; octadecanecarboxylic acid; hexadecane amide; docosane; hexachlorobiphenyl; benzylbutylphthalate; aliphatic amide; diisooctylphthalate; hexadecanoic acid hexadecyl ester; cholesterol.

Dioxin Like Compounds

2. Whilst there are only 210 possible dioxin and furan congeners of a single halogen there are about 4,600 discrete bromo, chloro and mixed halogen derivatives possible. Some or all of these may be produced in municipal waste incinerators. Only the 17 chlorinated dioxins have been measured as part of this survey. Not even PCBs, known to be a significant part of the dietary uptake, and to be a contaminant of incinerator ash, has been measured.
3. The failure to consider brominated and mixed halogenated dioxins is a significant concern because:
   - Levels of brominated compounds in breast milk have increased massively in the past two decades
   - There is evidence that these compounds are as toxic as the chlorinated species
   - Brominated and other halogenated dioxins are formed in municipal waste incinerators
   - The level of brominated pre-cursors that are included in municipal wastes are increasing (as fire retardants etc).

   Some of the evidence for these statements is:

Levels of brominated compounds in breast milk have increased massively in the past two decades

4. A study[39] summarising the investigations of polychlorinated biphenyls (PCBs), naphthalenes (PCNs), dibenzo-p-dioxins (PCDDs), dibenzofurans (PCDFs), polybrominated diphenyl ethers (PBDEs) and pesticides (DDT, DDE, hexachlorobenzene, dieldrin) as well as methylsulfonyl metabolites of PCBs and DDE in human milk sampled during different periods up to 1997. During the course of 20-30 yr. the levels of organochlorine compounds in human milk have decreased to various extent. On the contrary to the organochlorine compounds, the concentrations of PBDEs have increased during the period 1972-1997, indicating a doubling of the levels every 5 yr. These levels reflect the environmental contamination and background levels in the population. The authors called for immediate measures to stop the environmental pollution and human exposure to the dioxin like PBDEs.

The level of brominated pre-cursors that are included in municipal wastes are increasing

5. The widespread use of brominated fire retardants in commercial products such as carpets, textiles and plastics together with their inevitable disposal – at least in part – by incineration, afford a potentially very significant source of bromine for the generation of brominated compounds.
6. More stringent fire regulations in many countries have resulted in large increases in the use of flame retardants over the past decade the total consumption of PBDEs more than doubled over the period from 1984 to 1992 and world-wide PBDE production is estimated at roughly 80 million pounds per year.

Brominated and other halogenated dioxins are formed in municipal waste incinerators

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[39] Noren K, Meironyte D. Certain organochlorine and organobromine contaminants in Swedish human milk in perspective of past 20-30 years. Chemosphere 2000 May-Jun;40(9-11):1111-23

7. Weber[40] and Greim report that:

   Brominated dibenzo-p-dioxins and dibenzofurans can be formed under laboratory conditions by pyrolysis of flame retardants based on polybrominated biphenyls and biphenyl ethers. Their occurrence in the environment, however, is due to combustion processes such as municipal waste incineration and internal combustion engines. As these processes generally take place in the presence of an excess of chlorine, predominantly mixed brominated and chlorinated compounds have been identified so far in environmental samples.

8. Heeb[41] et al. found that:

   "Incineration of municipal waste yields a variety of halogenated phenols including chlorinated, brominated and mixed chlorinated phenols in raw and stack gas. A total of 14 chlorinated, 3 brominated and 31 mixed brominated and chlorinated phenols could be characterised by high resolution gas chromatography high resolution mixed spectrometry."

   And continued:

   "The growing commercial production of brominated organics and their widespread application as flame retardants and fuel additives are suspected to contribute increasingly to environmental pollution. Incineration of bromine containing products will lead to substantial formation of mixed brominated and chlorinated aromatics [6]. Upon thermal treatment of halogenated material bromine radicals do form preferentially compared to chlorine radicals. Already a moderate molar fraction of bromine in the halogen load of a flue gas has been found to reduce the amount of chlorinated aromatics significantly and to favor (sic) the formation of mixed brominated and chlorinated aromatics [7]. Compared to the accumulated knowledge about polychlorinated aromatics only little is known about the fraction of mixed halogenated aromatics produced"

9. The increase in brominated material may, therefore, have reduced the chlorinated dioxin levels whilst increasing the (unmeasured) brominated dioxins levels.

There is evidence that these compounds are as toxic as the chlorinated species

10. The USEPA Draft dioxin review[42] highlights the probable underestimates of exposure levels associated with the use of the current TEF assessments because of other compounds which act in a similar way not being included in the TEF totals:

    "The WHO working group acknowledged that there are a number of other classes of chemicals that bind and activate the Ah receptor. The chemicals include, but are not limited to, polyhalogenated naphthalenes[43], diphenyl ethers, fluorenes, biphenyl methanes, quaterphenyls,

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[40] Weber LW, Greim H The toxicity of brominated and mixed-halogenated dibenzo-p-dioxins and dibenzofurans: an overview. : J Toxicol Environ Health 1997 Feb 21;50(3):195-215

[41] Heeb, N.V., I. Dolezal, T. Bührer, P. Mattrel, and M. Wolfensberger, "Distribution of Halogenated Phenols Including Mixed Brominated and Chlorinated Phenols in Municipal Waste Incineration Flue Gas," Chemosphere, 31, No. 4, pp 3033-3041, 1995.

[42] Chapter 9. Toxicity Equivalence Factors (TEF) for Dioxin and Related Compounds Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds Part II: Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds, page 9-7, NCEA-I-0836 May 2000 External Review Draft

[43] A study by Abad et al. [Abad E, Caixach J, Rivera J Dioxin like compounds from municipal waste incinerator emissions: assessment of the presence of polychlorinated naphthalenes., Dept. of Ecotechnologies, CID-CSIC, Barcelona, Spain. Chemosphere 1999 Jan;38(1):109-20] reports that polychloronaphthalenes (PCN) have been identified and quantified in emission samples collected from different municipal waste incinerators. Polychlorodibenzo-p-dioxins (PCDD) and polychlorodibenzofurans (PCDF) were also determined to find a possible relationship between these classes of organochlorinated compounds. The total levels of PCN varied

and others. In addition, a number of brominated and chloro/bromo-substituted dioxin analogues of the PCDDs and PCDFs have been demonstrated to cause dioxin-like effects. The WHO working group concluded that "at present, insufficient environmental and toxicological data are available to establish a TEF value for any of the above compounds" (van den Berg et al., 1998)."

11. Weber and Greim warned very clearly of the dangers of these compounds:

    "Brominated dibenzo-p-dioxins or dibenzofurans bind to the cytosolic Ah receptor about as avidly as their chlorinated congeners and induce hepatic microsomal enzymes with comparable potency. The same holds true for mixed brominated-chlorinated compounds. Gross pathologic symptoms-hypothyroidism, thymic atrophy, wasting of body mass, lethality-also occur at doses that, on a molar concentration basis, are virtually identical to those seen with the chlorinated compounds. Their potency to induce malformations in mice following prenatal exposure is equivalent to that of chlorinated dibenzo-p-dioxins and dibenzofurans. Possible activities as (co)carcinogens and endocrine disrupters have not been evaluated, but are likely to exist. Considering the overall similarity in action of chlorinated and brominated dibenzo-p-dioxins and dibenzofurans, environmental and health assessments should be based on molar body burdens without discrimination for the nature of the halogen."

12. Hornung[44] et al. reported that:

    "2,3,7,8-TBDD was more potent than 2,3,7,8-TCDD in a early life stage rainbow trout mortality assay and that other polybrominated dibenzo-p-dioxins were equipotent or less potent than identically substituted polychlorinated dibenzo-p-dioxins. The brominated furan, 2,3,7,8-TBDF was 9-fold more potent than 2,3,7,8-TCDF and both 3,3',4,4'-TBB and 3,3',4,4',5,5'-HxBB were 10-fold more potent than identically substituted polychlorinated biphenyl."

13. Till[45] et al. analysed fly ash extracts and with municipal waste incinerators samples in the EROD bioassay using rat hepatocytes in primary culture. They found that the bioassay of the extract resulted in a two- to fivefold higher estimate of TCDD equivalents (TEQ) than the chemical analysis of PCDDs / PCDFs and PCBs.

14. They suggested that the unexplained inducing potency in fly ash samples probably results from additional dioxin like components including certain PAHs not analysed in their study[46]. This study could not, however, confirm the hypothesis that emissions from municipal waste incinerators of hitherto unidentified dioxin like compounds are higher by orders of magnitude than emissions of potent PCDDs / PCDFs and dioxin like PCBs.

15. Work by Kim Hooper[47] of the California Environmental Protection Agency describes the emerging environmental challenge of PBDEs as having many parallels with PCBs and

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from 1.08 up to 21.36 ng / Nm3 (mono- to octachlorinated) and 0.33 to 5.72 ng / Nm3 (tetra- to octachlorinated), whereas the levels of PCDD / PCDF ranged between 1.14 and 276.26 ng / Nm3 (0.01 and 5 ng I-TEQ / Nm3), depending on the type of the MWI.

[44] Hornung MW, Zabel EW, Peterson RE. (1996) Toxic equivalency factors of polybrominated dibenzo-p-dioxin, dibenzofuran, biphenyl, and polyhalogenated diphenyl ether congeners based on rainbow trout early life stage mortality.Toxicol Appl Pharmacol 1996 Oct;140(2):227-234

[45] Dioxinlike components in incinerator fly ash: a comparison between chemical analysis data and results from a cell culture bioassay. Till M, Behnisch P, Hagenmaier H, Bock KW, Schrenk D, Institute of Toxicology, University of T-ubingen, T-ubingen, Germany. Environ Health Perspect 1997 Dec;105(12):1326-32

[46] Note that more recent work by Fernandez et al, reported in ENDS 304 p12 "Sunlight increases PAH toxicity 1,000 fold, researchers find" shows that the toxicity of these compounds may be subject to influences that have not even been contemplated in traditional toxiccological assessments.

[47] Hooper K, McDonald T, 'The PBDEs: An emerging environmental challenge and another reason for breast milk monitoring programs, Environ Health Perspect, Vol 108, No 5, May 2000.

other persistent organic pollutants and suggests that these compounds may present a significant environmental challenge in the future.

16. *The disastrous history of incineration and chlorinated dioxins should provide a stark warning about the risks associated with dioxin like substances.*