Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment

COT Statement on a toxicological evaluation of chemical analyses carried out as part of a pilot study for a breast milk archive

Introduction

1. The Food Standards Agency has received the results of the SUREmilk pilot studies, which explored alternative methods for the recruitment, collection, storage and management of an archive of breast milk samples[1]. The report includes analytical data on a range of environmental contaminants (dioxins, polychlorinated biphenyls, organochlorine pesticides, phthalates and heavy metals). The Committee was invited to advise on whether these data raised toxicological concerns for the breast-fed infant, which would require urgent action or indicate a need for further research. The data were collected primarily to explore the viability of sample collection methods, they do not constitute a rigorous survey.

Study design

2. Primiparous pregnant women and mothers were recruited within six NHS Trusts in Yorkshire during 2001-2002. The age range was 16 to 42 (mean 28) years. Samples of breast milk were collected on one or more occasions at 1 to 16 weeks. The research was designed as pilot studies of issues associated with recruitment and storage, and not to investigate the range of contaminant concentrations present at different lactation times, or in different ages of mother, and cannot be assumed to be representative of the UK population.

3. There were three cohorts of women, defined by criteria related to recruitment and not to potential exposure to contaminants.

- Cohort 1 – the 322 women in this group expecting/having healthy full-term babies were asked to provide two samples - one at a fixed time point between 1 and 16 weeks, the other at a random point. This would give a cross-section for each time point (1, 2, 4 ,8 ,12-16 weeks).

- Cohort 2 – 54 self-selected volunteers, also expecting/having healthy full-term babies and especially committed to breast feeding, were asked to provide samples at 1, 2, 4, 8 and 12 weeks to create a longitudinal series.

- Cohort 3 – an opportunistic approach. Eighteen mothers of babies admitted to a Special Care Baby Unit were asked if they would be prepared for any unused expressed milk stored in the unit to be taken for the study once they had been discharged and there was confirmation that they did not require it. Cohort 3 represented only 5% of the recruits.

4. Concentrations of dioxins, polychlorinated biphenyls, organochlorine pesticides, phthalates and heavy metals were analysed to explore the range of variation in breast milk and to demonstrate stability on storage. For dioxins, the analysis required a sample size of approximately 300mL, therefore samples had to be pooled and do not provide information on the range of variation. A stability study indicated that data on the phthalates were unreliable and these were not considered further. Based on the results of the stability study and prior experience, the data on other contaminants were considered to be of an acceptable quality.

Intake estimations

5. There is no universally accepted methodology for assessing consumption of breast milk in different ages of infant, allowing for different nutritional needs and progressive weaning. Some researchers assume consumption of a fixed amount (e.g. 600ml or 800ml per day) regardless of age. The method employed in this evaluation is based on the results of a study which tracked the consumption of breast milk and growth of 48 infants in Cambridge [2]. Breast milk consumption was highest for infants under 2 months of age, with an average daily intake of 160g/kg bodyweight (bw). These data were previously used in estimating intake of dioxins and polychlorinated biphenyls (PCBs) by breastfed infants in the UK [3,4]. The estimates of intake from breast milk, and from infant food where available, have been compared with previous COT opinions and available tolerable intakes recommended by the Joint FAO/WHO Expert Committee on Food Additives (JECFA), the Joint FAO/WHO Meeting on Pesticide Residues (JMPR), the EU Scientific Committee on Food (SCF) or the Expert Group on Vitamins and Minerals (EVM).

PCBs and organochlorine pesticide residues in milk

PCBs

6. Seven ortho-PCBs were analysed in individual milk samples. One of these (PCB118) is considered to have weak dioxin-like activity, the others are non-dioxin-like. There is currently no accepted method of risk assessment for the non-dioxin-like PCBs, and there are no established tolerable intakes. These substances are currently being evaluated by a co-operation between

the European Food Safety Authority (EFSA) and the World Health Organisation (WHO), which is expected to report at the end of 2004.

Pesticides

7. Fifteen organochlorine pesticides were analysed in 92 individual milk samples from 48 donors. Alpha-HCH, beta-HCH, heptachlor, hepox (cis), hepox (trans), oxychlordane, chlordane (trans), chlordane (cis) and p,p-TDE were not detected. Hexachlorobenzene (HCB) and p,p'-DDE were detected in almost all samples (47 of 48 donors), p,p'-DDT in 26% (15 donors) and dieldrin in 16% (11 donors). o,p'-DDT was detected in the milk of one woman who also had a relatively high concentration of p,p'-DDE.

8. HCB concentrations ranged from 0.1 to 3.03 μg/kg (mean 0.66 μg/kg). Consumption of breast milk at the highest detected concentration of HCB would result in infants of 7 months and younger exceeding the TDI of 0.16 μg/kg bw/day [5], with a maximal 3-fold exceedance for infants below 2 months of age. A mean concentration of 1 μg/kg (range <1-5 μg/kg) was previously reported based on 193 samples taken between 1989 and 1991 in the UK [6].

9. HCB has been shown to induce liver tumours in rats, but from an overview of the predominantly negative mutagenicity data, it is considered not to be genotoxic. A WHO taskforce recommended a health-based guidance value of 0.16 μg/kg bw/day, which is used as the TDI for HCB [5]. This was based on the lowest reported no observed effect level (NOEL) of 0.05 mg HCB/kg bw/day, for primarily hepatic effects observed at higher doses in studies on pigs and rats exposed by the oral route, incorporating an uncertainty factor of 300 (× 10 for interspecies variation, × 10 for intraspecies variation, and × 3 for severity of effect). The COT considered that the toxicity profile of HCB did not justify the use of the 3-fold factor for severity of effect, and therefore the TDI was likely to be over-precautionary.

10. Studies involving lactational exposure in mink, rats and cats have shown effects on the offspring at maternal doses of 0.16 to 4 mg/kg bw/day. A NOEL of approximately 1 mg/kg bw/day was derived from a four-generation reproductive toxicity study in Sprague-Dawley rats. The lowest observed effect levels (LOELs) and NOELs represent maternal doses that are higher than the NOEL that provided the basis for the TDI. However, the dose to the pups is not known.

11. WHO (1997) noted that mean intakes of HCB by nursing infants have been estimated to range from < 0.018 to 5.1 μg/kg bw/ day in various countries [5]. Thus, the maximal intake of HCB estimated from the SUREmilk project (0.485 μg/kg bw/day) is at the lower end of the WHO range. The results of most studies on the levels of HCB in foods and human tissues over time indicate that exposure of the general population to HCB declined from the 1970s to the mid-1990s in many locations.

12. Based on representative levels of HCB in air, water and food, WHO (1997) estimated the total intake of HCB by adults in the general population to be between 0.0004 and 0.003 µg/kg bw/day, predominantly from the diet [5].

13. Overall, the exceedances above the TDI are undesirable, but the significance of this should be considered in the context of the use of maximum levels in the intake calculations, the precautionary derivation of the TDI and that the period of exceeding the TDI from breast-feeding is relatively short. The SUREmilk data do not highlight a new problem with respect to HCB levels in breast milk.

Dieldrin

14. Consumption of breast milk containing dieldrin at the highest detected concentration of 0.54 µg/kg, would result in an intake of 0.087 µg/kg bw/day in an infant of less than 2 months, which is below the PTDI of 0.1 µg/kg bw/day.

15. In 1977 JMPR recommended an ADI of 0.1 µg/kg bw (combined total for dieldrin and the closely related substance aldrin) [7]. Aldrin is readily metabolised to dieldrin in plants and animals. Only rarely are aldrin residues present in food or in the great majority of animals, and then only in very small amounts. Therefore, national and international regulatory bodies have considered aldrin and dieldrin together. The ADI was based on NOAELs of 1 mg/kg of diet in the dog and 0.5 mg/kg of diet in the rat, which are equivalent to 0.025 mg/kg bw/day in both species. JMPR applied an uncertainty factor of 250 based on concern about carcinogenicity observed in mice.

16. This ADI was reaffirmed in 1994 by JMPR [8], and was converted into a Provisional Tolerable Daily Intake (PTDI) because aldrin and dieldrin were no longer used as pesticides and are therefore viewed as environmental contaminants. Although levels of aldrin and dieldrin in food have been decreasing, dieldrin is highly persistent and bioaccumulates.

17. WHO reported that dieldrin has been detected in breast milk at a mean concentration of 0.5–11 µg/kg of milk in Europe and the USA. Breast-fed babies receive doses of approximately 1 µg/kg bw/day when mothers' milk contains 6 µg of dieldrin per litre. Although concentrations in breast milk decreased from an average of 1.33 µg/kg of milk in 1982 to 0.85 µg/kg of milk in 1986, higher concentrations (mean 13 µg/litre) have been found in breast milk from women whose houses were treated annually with aldrin [9].

DDT and DDE

18. Consumption of breast milk containing the highest detected concentrations of p,p'-DDE (38.7 µg/kg), o,p'-DDT (0.43 µg/kg) and p,p'-DDT (1.72µg/kg), would result in intakes of 6.19, 0.069 and 0.275 µg/kg bw/day, respectively in an infant of less than 2 months. Thus total intakes of DDT (both isomers) and DDE would be below the PTDI of 10 µg/kg bw/day set by JMPR for any combination of DDT and its metabolites DDD and DDE [10].

Metals

19. Thirteen elements were analysed in 91-114 individual milk samples. In most cases, the reporting does not allow identification of numbers of donors that these samples represent. Worst-case intake from breast milk has been calculated, using the maximum detected concentration. Total intakes of these metals were estimated from the maximum intakes from breast milk at different ages together with maximum estimated intakes from non-soya infant foods recently reviewed by the Committee [11]. The non-soya data are used because it is assumed that breastfed infants will not be fed on soya formula.

20. Cadimum, chromium, antimony and mercury concentrations were below the limit of quantitation in all samples, with very few above the limit of detection. The data are therefore considered insufficiently accurate for further analysis but are not of toxicological concern.

21. Aluminium was above the limit of quantitation in 24% of 91 samples, with a maximum concentration of 186 ìg/kg. Estimated intakes from breast milk ranged from 30 ìg/kg bw/day below 2 months of age to 7 ìg/kg bw/day at 8-10 months. The total estimated intakes of aluminium ranged from 44 to 184 ìg/kg bw/day and are considerably below the JECFA PTWI [12], which is equivalent to 1000 ìg/kg bw/day.

22. Cobalt was above the limit of quantitation in 7% of 91 samples, with a maximum concentration of 0.31 ìg/kg. Estimated intakes from breast milk ranged from 0.05 ìg/kg bw/day below 2 months of age to 0.01 ìg/kg bw/day at 8-10 months. These are considerably below the EVM guidance level, which is equivalent to 23 ìg/kg bw/day [13]. No estimates are available of intake from infant foods.

23. Nickel was above the limit of quantitation in 57% of 91 samples (6 donors), with a maximum concentration of 39 ìg/kg. Estimated intakes from breast milk ranged from 6.2 ìg/kg bw/day below 2 months of age to 1.4 ìg/kg bw/day at 8-10 months. The total estimated intakes of nickel ranged from 6.2 to 9.0 ìg/kg bw/day. This is up to 80% above the TDI of 5 ìg/kg bw/day set by WHO [14]. The COT previously concluded that the exposure estimates from infant food were likely to be over-estimates due to use of upper bound concentrations and worst case scenario consumption, and therefore the exceedance of the TDI was unlikely to be of significance. Furthermore, the COT noted that ingestion of nickel may exacerbate eczema in pre-sensitised individuals, and infants are less likely than adults to be sensitised to nickel [11].

24. Arsenic was above the limit of quantitation in 7% of 91 samples, with a maximum concentration of 4.0 ìg/kg. Estimated intakes from breast milk ranged from 0.64 ìg/kg bw/day below 2 months of age to 0.15 ìg/kg bw/day at 8-10 months. The total estimated intakes ranged from 0. 65 to 2.05 ìg/kg bw/day, which are all within the JECFA PTWI [15], equivalent to 2.14 ìg/kg bw/day. The COT has previously concluded that there are no appropriate safety guidelines for inorganic or organic arsenic, and that exposure to

inorganic arsenic should be As Low As Reasonably Practicable [16]. No data are available relating to the species of arsenic, or the sources of maternal exposure leading to the levels detected in breast milk. The majority of dietary exposure is organic arsenic from fish, and the small number of samples in which arsenic was quantifiable may be consistent with a small proportion of the women eating larger amounts of fish. However, no information is available on the form of arsenic present in the breast milk.

25. Tin was above the limit of quantitation in only two of 91 samples, with a maximum concentration of 6 ìg/kg. The estimated intakes from breast milk ranged from 0.96 ìg/kg bw/day below 2 months of age to 0.22 ìg/kg bw/day at 8-10 months. The total estimated intakes ranged from 1.5 to 19 ìg/kg bw/day. All estimated intakes are considerably below the EVM guidance value [13], which is equivalent to 220 ìg/kg bw/day.

26. Lead was above the limit of quantitation in 7% of 114 samples, with a maximum concentration of 2.6 ìg/kg. The estimated intakes from breast milk ranged from 0.42 ìg/kg bw/day below 2 months of age to 0.1 ìg/kg bw/day at 8-10 months. The total estimated intakes ranged from 0.44 to 0.69 ìg/kg bw/day. All estimated intakes are below the JECFA PTWI [17], which is equivalent to 3.6 ìg/kg bw/day.

27. Copper was above the limit of quantitation in all 114 samples, with a maximum concentration of 896 ìg/kg. The estimated intakes from breast milk ranged from 143 ìg/kg bw/day below 2 months of age to 33 ìg/kg bw/day at 8-10 months. The total estimated intakes ranged from 109 to 184 ìg/kg bw/day. All estimated intakes are below the JECFA PTWI [18], which is equivalent to 500 ìg/kg bw/day, but total estimated intakes for the younger age groups marginally exceeded the EVM Safe Upper Level [13], which is equivalent to 160 ìg/kg bw/day. The dietary exposure estimates are likely to overestimate actual intakes, and since copper is an essential element, extrapolation to infants on a bodyweight basis may not be appropriate.

28. Zinc was above the limit of quantitation in all 114 samples, with a maximum concentration of 9424 ìg/kg. The estimated intakes from breast milk ranged from 1508 ìg/kg bw/day below 2 months of age to 349 ìg/kg bw/day at 8-10 months. The total estimated intakes ranged from 1411 to 2264 ìg/kg bw/day. All estimated intakes exceed the JECFA PTWI [19], which is equivalent to 1000 ìg/kg bw/day, by 40-140%. Zinc is an essential element, and extrapolation to infants on a bodyweight basis may not be appropriate.

29. Selenium was above the limit of quantitation in all samples, with a maximum concentration of 28 ìg/kg. The estimated intakes from breast milk ranged from 4.5 ìg/kg bw/day below 2 months of age to 1.0 ìg/kg bw/day at 8-10 months. The total estimated intakes ranged from 3.1 to 5.8 ìg/kg bw/day All estimated intakes are below the EVM Safe Upper Level [13], which is equivalent to 7.5 ìg/kg bw/day. Selenium is an essential element and extrapolation to infants on a bodyweight basis may not be appropriate.

Dioxin and dioxin like-PCBs

30. Based on analysis of 15 pooled samples of breast milk, the total TEQ content, including the dioxin-like PCBs was estimated to range from 12 to 28 pg WHO-TEQ/g fat, corresponding to 0.26 to 0.78 WHO-TEQ/g whole weight.

31. Table 1 compares the estimated dioxin and dioxin-like PCB intakes derived from the SUREmilk data, with intakes from milk collected from women in Birmingham, Glasgow and Cambridge in 1993/4 [3, 20], and from infant foods [21]. The data indicate that intakes by breastfed infants have decreased by over 50% since 1993/4. It should be noted that the accuracy of these estimates at different ages is unclear, since all data are derived from pooled milk and do not allow for the decreasing concentrations of dioxins and PCBs with duration of lactation.

Table 1 Dietary intakes of dioxins and dioxin-like PCBs by breast-fed infants

Age (months)	**Mean consumption of breast milk [2] (g/kg bw/d)**	**Estimated *upper bound* dietary intakes of dioxins + PCBs (pg WHO-TEQ/kg bodyweight/day)**		
		Human milk		**Baby foods**
		Suremilk Pools A-Z	**1993/94 [3,20]**	**2003 [21]**
< 2	160	46-125 (mean 83)	186	
2-3	140	41-109 (mean 72)	163	
3-4	124	36-97 (mean 64)	144	
4-5	103	30-80 (mean 53)	120	
5-6	79	23-62 (mean 41)	92	0.3-0.4
6-7	63	18-49 (mean 33)	73	
7-8	42	12-33 (mean 22)	49	
8-10	37	11-29 (mean 19)	43	0.4

32. The majority of the TEQ derives from 1,2,3,7,8-pentachlorodibenzodioxin (24%), 2,3,4,7,8,-pentachlorodibenzo-furan (20.6%), PCB126 (15%), PCB156 (8.4%), 1,2,3,6,7,8-hexachlorodibenzodioxin (8.0%) and 2,3,7,8-tetrachlorodibenzodioxin (7.5%). The half-lives of the dioxin and furan congeners in humans have been estimated to be 5.3-16, 5.0-19.6, 3.5-14 and 4-11 years, respectively, and the estimated half-life for PCB156 is 4.2 years[22]. Half-life data are not available for PCB 126 and there is a need for generation of these data to support the risk assessment.

Previous COT evaluations

33. COT last discussed concentrations of dioxins and PCBs in human milk in 1997, when the TDI for dioxins and dioxin-like PCBs was 10 pg TEQ/kg bw/day [4]. At that time the mean total intake of breast-fed infants was reported to range from 170 pg Int-TEQ/kg bw/day at 2 months of age to 39 pg TEQ/kg bw/day at 10 months of age. Since the toxicity equivalency factors have been

revised in the intervening period, these data have been converted to the WHO-TEQ in Table 1. The Committee noted a number of factors, including:

- infants may be more susceptible to these compounds
- a high level of exposure during a critical period of early postnatal development may have adverse effects, whereas exposure to the same level in later life would not
- the period of breast-feeding is short compared with the time needed to accumulate these compounds in the body
- there are known benefits to breast-feeding.

34. Overall, the Committee concluded that, although intakes of dioxins and PCBs by breast-fed babies were higher than is desirable, breast-feeding should continue to be encouraged on the basis of convincing evidence of the benefits of human milk to the overall health and development of the infant. The Committee recommended that the levels of dioxins and PCBs in human milk should be surveyed at regular intervals and, if levels did not continue to fall, that a review should be instigated to investigate whether inputs of these pollutants to the environment can be reduced further, so as to reduce human exposure. The COT also noted that it would be desirable to investigate the potential for accumulation of the dioxin and PCB congeners which occur in breast milk, that is their disposition in and elimination from the body, especially in early life.

35. In 2001, the COT revised the Tolerable Daily Intake (TDI) for dioxins and dioxin-like PCBs [23]. The Committee noted the advice of the COC, that TCDD should be regarded as a "probable human carcinogen", but that the negative genotoxicity data and evidence from mechanistic studies suggested that a threshold approach to risk assessment was appropriate. The COT considered that, because of the long half-life of TCDD, the risk assessment should be based on the body burden. A TDI of 2 pg WHO-TEQ/kg bw/day was established, based upon effects on the developing male reproductive system mediated via maternal body burden. The body burden of TCDD at steady state is about 2000-fold higher than the average daily intake, and therefore occasional exceedance of the TDI would not be expected to result in harmful effects, provided that intake averaged over a prolonged period is within the TDI.

36. In 2003, the FSA published the results of the 2001 Total Diet Study of dioxins and dioxin-like PCBs [24]. The key observations were:

- Estimated total dietary intakes of dioxins and dioxin-like PCBs by all age groups fell by around 50% between 1997 and 2001, continuing the reduction in intakes seen from the previous surveys.
- The percentage of school-aged children estimated to exceed the TDI fell from 62% in 1997 to 10% in 2001.
- The percentage of toddlers (aged 1.5 – 4.5 years) estimated to exceed the TDI fell from 97% in 1997 to 37% in 2001.

Comments on new data

37. Data on dioxins and PCBs in breast milk may not be directly comparable because of the factors that influence the levels. Dioxin content of breast milk varies with maternal age, and previous breast-feeding history. The method of milk collection will influence the concentrations detected because the fat content varies during the course of a single feed, and dioxin levels tend to decrease with duration of lactation. However, the SUREmilk data indicate that concentrations have decreased, which would be consistent with the decreasing dietary exposure to dioxins and PCBs.

38. In infants under 2 months of age, the estimated intakes of dioxins and dioxin-like PCBs from breast milk could result in daily intakes of 20-60 times the TDI. The TDI is set at an intake that would prevent accumulation of dioxins in a woman's body to a body burden that could be harmful to the fetus. The half-life of TCDD is considered to be in the region of 7.5 years, and the other predominant congeners also have half-lives of several years. It could therefore take in the region of 30 years or more for the body burden to reach steady-state.

39. The infant's body burden is lower than that of its mother because of its low body fat. Weisglas-Kuperus *et al*. (2000) reported on concentrations of marker PCBs in blood of children aged 42 months [25]. The data indicated that the average children's PCB body burdens were 15-20% of the average maternal body burden, and the PCB body burden of breast-fed children was about 3-4 times that of formula-fed children. Similar differences might be expected for the TEQ body burden, but data are not available because of the large volume of blood needed for the analysis.

40. Pollitt (1999) calculated accumulated TEQ body burden, assuming a half-life of 9 years for all congeners, 100% absorption and a zero body burden at birth, using the previous UK data for dioxins and dioxin-like PCBs in breast-milk [26]. Breast-feeding for 6 months was predicted to result in increased body burden in early life, but the steady state level, reached at about 40-50 years of age, was not increased. The most recent JECFA monograph on dioxins and dioxin-like PCBs used a physiologically-based pharmacokinetic (PBPK) model to predict the effects of transplacental exposure and 6 months of breast-feeding on the body burden of the infant [22]. This model uses a number of different assumptions on the relative partition coefficients of milk fat and adipose tissue. It is unclear which of these are most appropriate, but the worst case predicts a higher body burden in young children than in adults.

41. The COT previously commented on studies of development of children in Rotterdam and Groningen, with cohorts sub-divided into groups that were breast-fed for at least 6 weeks or formula fed [23]. The researchers reported that dioxin exposure was associated with impaired cognitive development and changes in the children. The COT concluded that concerns over the known and potential confounders made it impossible to reach firm conclusions. However, if the effects were real, they were most likely to be due to pre-natal exposure, and breast-feeding ameliorated the effects. In these studies, the mean concentrations of dioxins and dioxin-like PCBs in breast milk were in the

region of 60-70 pg WHO-TEQ/g fat, i.e 2-5 times higher than those reported in the SUREmilk study.

42. The most sensitive effect of dioxins and dioxin-like PCBs are considered to be impairment of sperm production and morphology in the male offspring resulting from exposure *in utero.* It is unclear whether effects on sperm quality or other reproductive effects would be expected to result from post-natal exposure. There are no available studies of sperm quality in adult men who were breast-fed as infants compared with those who were formula-fed. However, compared with the previous COT evaluation on dioxins and PCBs in breast-milk (paragraph 33), there is less concern that infants could be more susceptible to these compounds. Any possible risks need to be considered in the context of the revised basis for the TDI, the decreasing concentrations of dioxins and dioxin-like PCBs in food and in breast milk, and the well-established benefits of breast-feeding [27].

Conclusions

43. We *note* that the SUREmilk samples were collected primarily to explore the viability of breast milk collection methods and do not constitute a rigorous survey. Nevertheless, it is possible to draw some conclusions on the toxicological implications of the data.

44. We *conclude* that the estimated intakes of metals and other elements associated with the highest detected concentrations in the breast milk samples do not raise toxicological concerns.

45. We *consider* that the data on non-dioxin-like PCBs should be reviewed after completion of the international risk assessment of non-dioxin-like PCBs.

46. We *consider* that although intakes of HCB by some breast-fed babies may be higher than is desirable, this relatively short period of exceeding the TDI is unlikely to result in adverse effects. We *consider* that the intakes of the other organochlorine pesticides measured in this study were within relevant tolerable intakes and do not raise toxicological concerns.

47. The intakes of dioxins and dioxin-like PCBs derived from the SUREmilk study appear to be less than half of those from breast milk sampled in 1993/4. This indicates that concentrations of dioxins and dioxin-like PCBs in breast milk have decreased; however we caution that the data may not be directly comparable to those reported previously or representative of the UK population.

48. Dietary exposure to dioxins and dioxin-like PCBs has decreased considerably over the past 20 years, and these substances are eliminated slowly from the body. The concentrations of these substances in breast milk are therefore expected to continue to fall.

49. The TDI is set to protect against the most sensitive effects of dioxins and dioxin-like PCBs, which occur in the male fetus as a result of the mother's accumulated body burden. There is uncertainty with respect to whether similar effects would arise from post-natal exposure, but there is currently no basis for assuming that the young infant is at increased risk.

50. Taking into account that the TDI is now set to protect against reproductive effects and the evidence that concentrations of dioxins and dioxin-like PCBs in breast milk are declining, the new data do not suggest any reason to alter Government advice that breast-feeding should continue to be encouraged on the basis of convincing evidence of the benefits of human milk to the overall health and development of the infant.

51. We *recommend* that monitoring of contaminants in breast milk should continue, particularly for HCB and the dioxins and PCBs, incorporating appropriate studies of stability and recovery. Data on other contaminants of potential concern, such as the brominated flame retardants, are also required.

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