

3rd WHO-COORDINATED EXPOSURE STUDY

TEMPORAL AND SPATIAL TRENDS OF PCDDS/PCDFS AND PCBs IN NORWEGIAN BREAST MILK – RESULTS FROM THREE ROUNDS OF WHO CO-ORDINATED STUDIES

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Introduction

Ever since Laug et al.¹ detected DDT in breast milk from non-occupationally exposed mothers, human milk monitoring programs have been performed in many countries for elucidating the infants' intake of persistent organochlorine compounds (OCs) from nursing, for comparing levels of environmental pollution by these chemicals in different areas within or between countries, and for investigating temporal trends in exposure levels. In Norway, human milk surveys for PCBs and chlorinated pesticides have been performed periodically since 1970².

Several parameters may affect the levels of OCs in breast milk, such as age, duration of lactation, parity, intake of highly contaminated food, area of residence and smoking habits. In order to keep the inherent variability of breast milk samples to a minimum and improve comparability of results from different investigations in 1986, WHO/EURO developed a protocol for analytical field studies of PCBs and PCDDs/PCDFs in breast milk and invited institutions world-wide to participate in the study. The main purpose was to gain as comprehensive a picture as possible of the situation regarding breast milk contamination levels by PCBs and PCDDs/ PCDFs in different geographical areas. This first field study was completed in 1987 and since then, two more WHO co-ordinated rounds were performed in 1992-93 and 2000-01^{3,4}. Norway has participated in all three rounds using the WHO developed protocols, and breast milk samples were collected from the same three different geographical areas representing a coastal area in the North (Tromsø), a rural inland area (Hamar) and an industrialised area in the South with known dioxin source (Skien/Porsgrunn). In this contribution we compare the results of these three studies with respect to temporal and spatial trends of PCDDs/PCDFs and PCBs in Norwegian breast milk.

Method and Materials

Breast milk from 10-12 primiparous mothers living for at least 5 years in the particulate area was collected between 2 weeks and 2-3 months after delivery. The age of the mothers was very similar for both the three geographical areas and the three time periods (mean 26 years). A questionnaire covering basic personal data, occupation, previous places of residence, dietary and smoking habits, was filled out by each mother.

Analyses were performed by solvent extraction, clean-up and determination by GC-ECD (organochlorine pesticides and PCBs) or GC-HRMS (PCDDs/PCDFs) as described elsewhere⁵⁻⁷. In the first study in 1985/86 10-12 individual samples were analysed⁵. Because of the high cost and time consumption of the analyses and requirements for relative large sample volumes from the donors, in the 2nd and 3rd round, samples from 10 donors of each area were pooled.

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Levels expressed in 2,3,7,8-TCDD toxic equivalents (TEQs) were calculated using the toxic equivalency factors established by WHO in 1998.

Results and Discussion

PCDDs/PCDFs

The concentrations of PCDDs/PCDFs for all three locations and study rounds are given in Figure 1. Relative standard deviations for the mean PCDD/PCDF levels of individual breast milk samples collected during the 1st round in 1985/86 at the three locations were 23, 33, and 39 % for Tromsø, Hamar and Skien/Porsgrunn, respectively. Thus, the small geographical differences found in TEQ levels were not statistically significant. However, for some penta- and hexaCDFs, the levels were found significantly higher in the Skien/Porsgrunn area than in the two other areas. These differences seem to be related to the known source of PCDFs emissions (Mg production) in Porsgrunn. Also in the 2nd round, levels of some PCDF were somewhat higher in Porsgrunn, however, no clear differences were seen in 2000/01. This is consistent with a dramatic reduction in emissions of OCs from the Mg production plant during the late 1980s.

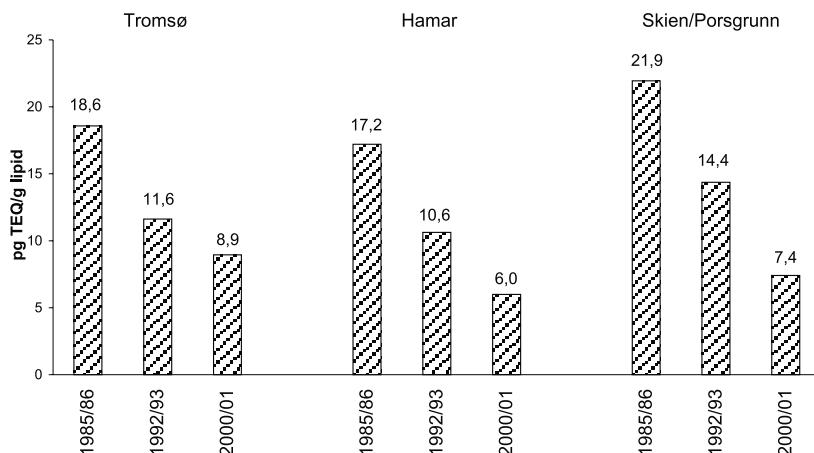


Figure 1. PCDD/PCDF TEQs in Norwegian breast milk sampled during the three rounds of WHO co-ordinated surveys.

As the same protocol was used in all three surveys, sample variability due to other factors than dietary intake is expected to be minimal, and results for the different time periods should well reflect changes in exposure. An average reduction of PCDD/PCDF TEQs in breast milk of about 37 % has been observed between consecutive surveys separated by a period of 7-8 years. Similar decreases in breast milk contamination have been found in other countries. This is in accordance with the general reduction in emissions of PCDDs/PCDFs and their occurrence in the environment and in food. In total, the PCDD/PCDF reduction for the 15 years between the first and the last round is 61 %.

The congener profiles for the three rounds were very similar for the country average with 2,3,4,7,8-PeCDF, 1,2,3,7,8-PeCDD, 2,3,7,8-TeCDD and 1,2,3,6,7,8-HxCDD contributing between 86 and 88% to the PCDD/PCDF TEQ for all locations and time periods.

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PCBs

For the determination of PCBs in the 1st round, packed column GC with Aroclor 1254 pattern comparison was used ("total PCB"). In order to compare the results of total PCB levels with later results obtained by congener specific determinations, both methods were employed in the second round. The sum of seven indicator PCBs (CB-28, 52, 101, 118, 138, 153, 180) as determined by capillary GC accounted on the average for 62 % (56-70 %) of the total PCB levels determined by packed-column GC. This relationship was used for the calculation of total PCB in the 3rd round.

The comparison of total PCB levels for the three studies is given in Figure 2. As with PCDDs/PCDFs, there is no obvious geographical difference in total PCB levels. Furthermore, there is no large temporal change in concentrations of PCB in breast milk from 1985/86 to 1992/93 (overall mean 515 ng/g lipid). In contrast, total PCB levels have decreased by an average of 58 % (48-67 %) during the 8 years interval from 2nd to 3rd round.

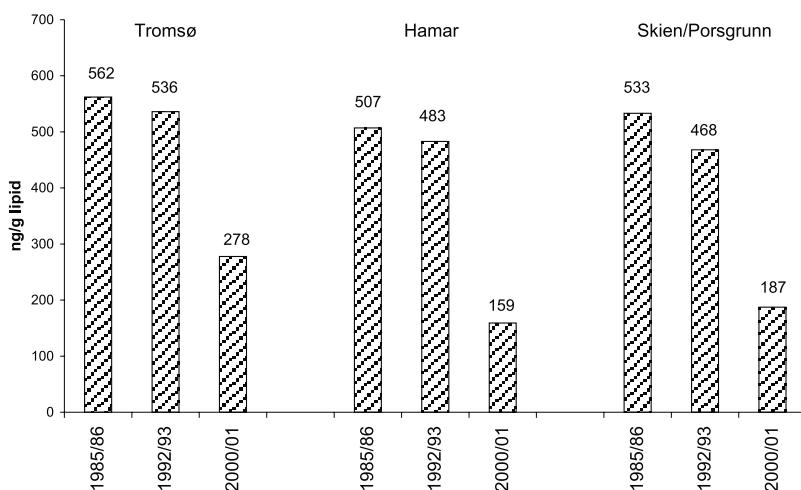


Figure 2. Total PCB levels in Norwegian breast milk sampled during the three rounds of WHO co-ordinated surveys

As there are no congener specific PCB data available for the 1985/85 study, contributions of dioxin-like PCBs to 2,3,7,8-TCDD toxic equivalents can only be calculated for the 2nd and 3rd round. The major contributors to PCB TEQs were CB-126, CB-156, CB-118 and CB-114. CB-123 and CB-167 have not been determined in our studies, however, due to their low abundance and/or TEF value, it is not expected that these congeners contribute significantly to the total TEQ.

Total TEQ

The total TEQs for the three locations in 1992/93 and 2002/01 are shown in Figure 3. The contributions of PCDDs/PCDFs, non-*ortho* and mono-*ortho* PCB to the total TEQ are similar throughout the data set and average to 40 %, 25 % and 35 %, respectively.

Assuming a daily intake of 160 g milk/kg b.w. and using the mean fat content of 3.2 % from the 3rd round, the estimated weekly intake of dioxin-like compounds for a breast-fed infant in Norway

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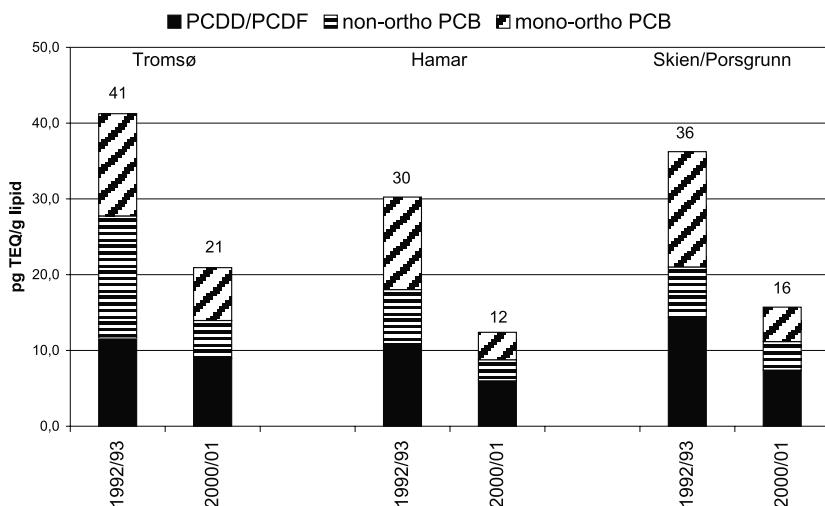


Figure 3. Total TEQs for PCDDs/PCDFs, non-*ortho* and mono-*ortho* PCBs in pooled samples from Norway

presently will be 590 pg/kg b.w. In spite of the continued decrease of levels of OCs in Norwegian breast milk, this exceeds the tolerable life-long intake (TWI) established by the EU (14 pg/kg b.w./week) by a factor of about 42. However, it should be kept in mind that TWIs are based on a life-long intake and are not directly applicable for breast-fed infants because of the relatively short nursing period and the rapid increase in amount of the infants' adipose tissue. It is generally agreed on that, due to its overall advantages, breast-feeding is the preferred nutrition for infants.

Acknowledgements

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References

1. Laug E.P., Kunze F.M. and Pitchett C.S. (1951) Arch Ind Hyg 3, 245.
2. Johansen H.R., Becher G., Polder A. and Skaare J.U. (1994) J Toxicol Environ Health 42, 157
3. WHO/EURO (1989). Environmental Health Series No. 34. World Health Organization – Regional Office for Europe, Copenhagen, Denmark.
4. WHO/EURO (1996). Environmental Health in Europe No. 3. World Health Organization – Regional Office for Europe, Copenhagen, Denmark.
5. Clench-Ass J., Bartonova A., Oehme M. and Lindström G. (1992) J Toxicol Environ Health 37, 73.
6. Becher G., Skaare J.U., Polder A., Sletten B., Rossland O.J. Hansen H.K. and Ptashekas J (1995) J Toxicol Environ Health 46,133.
7. Polder A., Odland J.O., Tkachev A., Føreid S., Savinova T.N., Skaare J.U. (2002) Sci. Total Environ., in press.