OCCURRENCE AND PRE- AND POSTNATAL TRANSFER OF PBDES, PCBS AND OH-PCBS IN HUMANS

Daiva Meironyté Guvenius[1,2], Åke Bergman[2] and Koidu Norén[2]

[1]Department of Medical Biochemistry and Biophysics, Karolinska Institutet, S-171 77 Stockholm, Sweden
[2]Department of Environmental Chemistry, Stockholm University, S-106 91 Stockholm, Sweden

Introduction

A number of persistent organohalogen compounds are present in the environment. Such contaminants may be retained in the human body as such or may be metabolised to more polar compounds and excreted or retained in the body. A mother's exposure to persistent organohalogen contaminants will result in the exposure of the foetus if these contaminants cross the placenta. Additionally, new-born children are exposed via breast milk. In the present study, we have investigated the occurrence and distribution of polybrominated diphenyl ethers (PBDEs), polychlorinated biphenyls (PCBs) and hydroxylated metabolites of PCBs (OH-PCBs) in humans and the transfer of these compounds from mother to foetus.

Methods and Materials

Samples

Paired samples of human liver and abdominal adipose tissue were obtained at the autopsy from five Swedish subjects, one woman and four men, who had suffered a sudden death at 47 and 66-83 years of age, respectively.

Maternal blood, cord blood and breast milk samples were collected from 15 mothers living in the Stockholm region. All mothers delivered by caesarean surgery. The average age of the mothers was 32 years, 53% of them gave birth to their first infant. Maternal blood samples were collected at the mother's arrival at Karolinska hospital, cord blood at partus and breast milk 2-77 days after delivery.

Analytical procedure

Previously described analytical methods were used for simultaneous analysis of organohalogen compounds in human liver, adipose tissue, breast milk, maternal and cord blood [1-6]. The analytical method is based on liquid-gel partitioning with Lipidex 5000. Biological tissues were homogenised and extracted with organic solvents before partitioning [1,2].

At the partitioning with Lipidex 5000, organohalogen compounds together with lipids were transferred into the gel. Separation of analytes from lipids was partly achieved by eluting the gel with solvents of different polarity. Organohalogen compounds together with a part of lipids were eluted with acetonitrile and the rest of lipids with a mixture of methanol/ chloroform/ hexane [1-5].

The sum of the dry weight of these two fractions determined the lipid amount in the sample.

The residue of the acetonitrile fraction was applied on an aluminium oxide column. Organohalogen contaminants were eluted with hexane and further separated on a silica gel column. PCBs were eluted from the column with hexane and PBDEs with a mixture of dichloromethane/hexane [1-5]. The fraction containing PBDEs was purified by gel permeation chromatography (Bio-Beads X-S3) [1] or on a sulphuric acid prepared silica gel column [5]. PCBs were determined by GC/ECD and PBDEs by GC/MS.

Phenolic compounds were eluted from aluminium oxide column with acidified methanol and methanol, derivatised by diazomethane, purified on a column of silica gel impregnated with sulphuric acid and determined by GC/MS [2,5]. Selected ion monitoring was applied for all GC/MS analysis. The quantification was made by comparison to authentic reference standards [2,4].

Results and Discussions

PBDEs

PBDEs were present in all samples. BDE-47, BDE-99 and BDE-153 were the dominant congeners in all sample matrices. In breast milk, blood plasma and adipose tissue BDE-47 was the predominant congener, while in liver samples BDE-99 occurred at similar relative levels as BDE-47 (Figure 1).

The levels of PBDEs ranged 5-18 ng/g lipids and 4-8 ng/g lipids in liver and adipose tissue, respectively.

Pre- and postnatal exposure to PBDEs was investigated by analysing maternal blood plasma, cord blood plasma and breast milk from the same mothers. The PBDE levels were similar in maternal blood plasma and breast milk (0.6-7.7 and 0.7-8.4 ng/g lipids, respectively), while lower levels were found in cord blood plasma samples (0.5-4 ng/g lipids). In cord blood plasma the levels of higher bromianted PBDEs were significantly lower, indicating that these PBDE congeners cross the placenta to a lower extent.

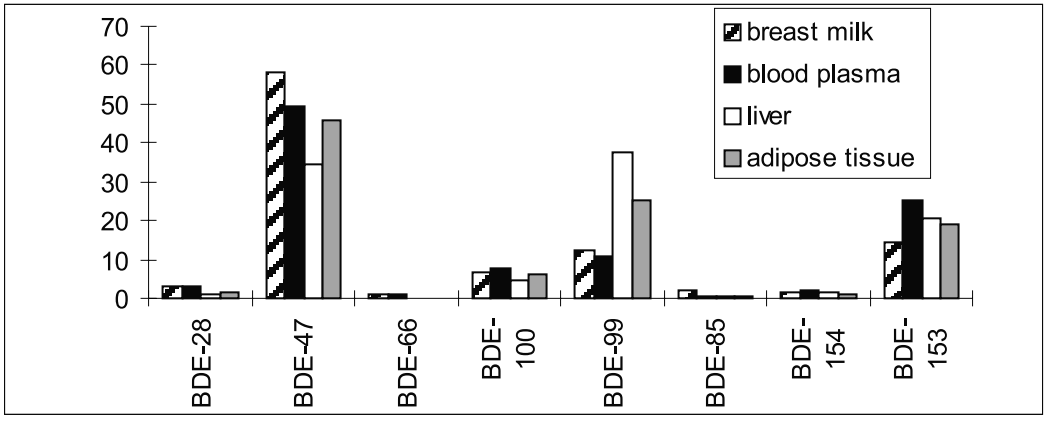

Figure 1. The mean distribution of PBDE congeners in percent of the sum of congeners in breast milk and maternal blood plasma (n=15), liver and adipose tissue (n=5).

PCBs

All sample matrices contained PCBs with CB-153, CB-138 and CB-180 as the dominating PCB congeners. The distribution of PCB congeners was slightly different in the samples. The relative concentrations of e.g., CB-180 were higher in liver and adipose tissue than in milk and blood plasma, while, those of CB-138 and CB-153 were higher in milk and blood plasma than in liver and adipose tissue (Figure 2). The differences in congener distribution may be related to the sample matrices and/or the age of the subjects.

The sum of PCB congeners was similar in liver and adipose tissue (459-2085 and 561-2343 ng/g lipids, respectively) and in maternal blood plasma and breast milk (104-598 and 77-547 ng/g lipids, respectively). Slightly lower levels of PCBs were found in cord blood samples (67-330 ng/g lipids).

The presence of PCBs in cord blood plasma and breast milk samples confirms the pre- and postnatal exposure to these compounds.

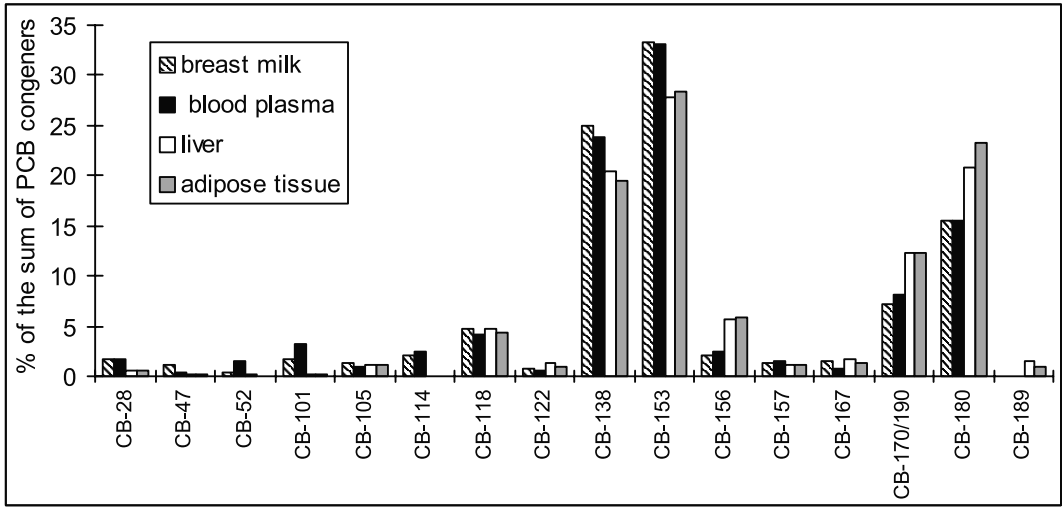

Figure 2. The mean distribution of PCB congeners in percent of the sum of congeners in breast milk and maternal blood plasma (n=15), liver and adipose tissue (n=5).

OH-PCBs

Matrix specific differences were observed in OH-PCB congener distribution. 4-OH-CB187 and 4-OH-CB-146 were the most dominant congeners in blood plasma, while 3'-OH-CB138 and 4'-OH-OH-CB130 dominated in liver, and 3'-OH-CB138 and 4-OH-CB-193 in adipose tissue (Figure 3).

The sum of OH-PCB congeners in liver and adipose tissue samples was 12-358 ng/g lipids and 2-9 ng/g lipids, respectively. However, large variations were noticed for OH-PCB congeners between the adipose tissue samples.

The concentrations were slightly lower in cord blood plasma (35-271 pg/g plasma) than in maternal blood plasma (82-328 pg/g plasma). OH-PCBs constituted up to 30% of the PCB levels in maternal blood plasma and 50% in cord blood plasma. The levels of OH-PCBs in breast milk were very low (0-5 pg/g plasma).

These results indicate that OH-PCBs pass the placental barrier. Because of the low levels of OH-PCBs in breast milk the postnatal exposure to OH-PCBs in the present study subjects may be considered of less importance.

Acknowledgments

We are sincerely grateful to Prof. Lars Grimelius, Dr. Anette Aronsson and Prof. Gunvor Ekman-Ordeberg for supply of samples, Prof. Mats Hamberg for the supply of diazomethane. The study was

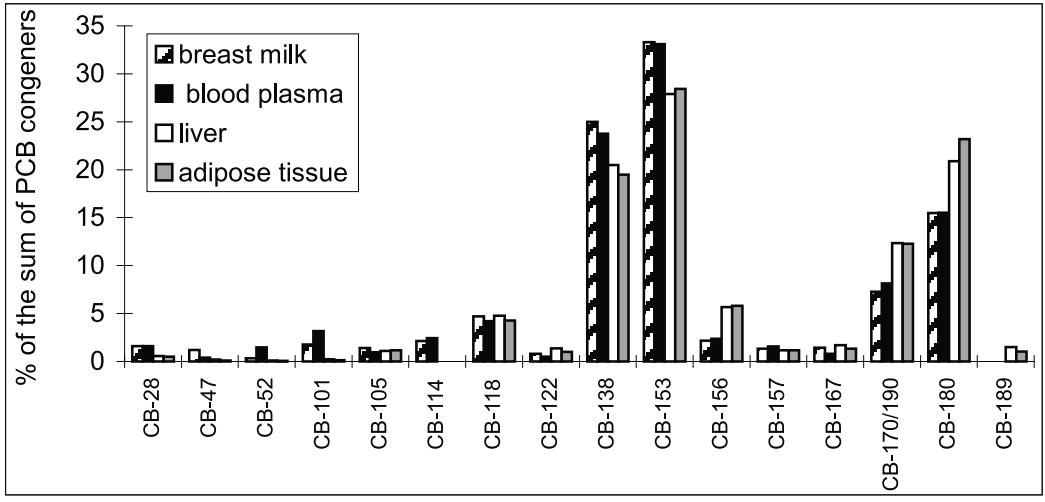

Figure 3. The mean distribution of OH-PCB congeners in percent of the sum of congeners in maternal blood plasma (n=15), liver and adipose tissue (n=5).

supported by Formas, Swedish Council of Work Life Research, MISTRA (Foundation for Strategic Environmental Research) and Karolinska Institutet.

References

1. Meironyté Guvenius D., Bergman Å. and Norén K. (2000) Arch Environ Contam Toxicol. 40, 564
2. Meironyté Guvenius D., Hassanzadeh P., Bergman Å. and Norén K. (2002) Environ Toxicol Chem. Accepted
3. Weistrand C. and Norén K. (1998) J Toxicol Environ Health. 53, 293
4. Meironyté D., Norén K. and Bergman Å. (1999) J Toxicol Environ Health. 58, 329
5. Meironyté Guvenius D., Aronsson A., Ekman-Ordeberg G., Bergman Å. and Norén K. (2002) Environ Health Perspect. Submitted
6. Meironyté Guvenius D. (2002) Organohalogen contaminants in humans with emphasis on polybrominated diphenyl ethers (Thesis), Karolinska Institutet, ISBN 91-7349-140-3