SIGNIFICANT CHANGES OF SERUM CONCENTRATIONS OF LIPID AND PCB DURING PREGNANCY AND NURSING

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Introduction

Polychlorinated biphenyls (PCBs) are environmental pollutants with high persistence to biodegradation resulting in contamination of foods of animal origin. As a consequence, PCBs (and other organochlorine pollutants such as dioxins) are present at relatively high concentrations in human tissues. Of special concern is the transport of PCBs from mother to foetus, which is sensitive to the toxic effects of organochlorines. Exposure during breast-feeding is 10-100 times higher compared to other periods in life, which also is a cause of concern[1]. The concentration of PCBs in the blood of the pregnant women is the determining factor for transport to the foetus/baby[2]. Most often, the blood concentration is expressed as ng/g serum lipid. The lipid concentration is believed to be a relevant mirror of the total body burden and enable comparisons between lipid concentrations of organochlorines in different tissues[3]. The aim of the present study was to increase our knowledge on PCBs in serum during pregnancy and nursing. The results could be used to improve the timing of blood sampling as well as for a discussion about the best way to express (i.e. lipid weight or fresh weight) the concentrations of persistent organochlorine pollutants (POP) in blood samples.

Methods and Materials

11 primiparous women from the general population in Uppsala County, Sweden, were recruited for this study. They accepted repeated blood sampling during the pregnancy (start week 9-13) and during the breast-feeding period (end 3 months after delivery, week 52). Serum concentration of 10 PCBs and 11 organochlorine pesticides (only the PCBs are reported in this abstract) were analysed in serum of the mothers. The ten PCB congeners (IUPAC no 28, 52, 101, 105, 118, 138, 153, 156, 167 and 180) analysed are commonly found in human serum and tissue samples and some of the congeners cause toxicity in animals[4]. Results below the limit of determination (LOD) was set to half of the LOD. The congeners PCB 52, 101, 105 and 167 were not included in the analysis since over 80 % of the samples were below the LOD.

The PCBs were analysed at the Swedish National Food Administration and procedures for extraction of samples and sample clean up were basically the same as described elsewhere[5].

Values are presented as means ± S.E.M. Differences between groups were detected by one way ANOVA, followed by Tukey's post-test to detect individual differences.
A limited one way ANOVA followed by Tukey's post-test to detect individual differences was performed for week 35-38, week 43 and week 52 (* $P<0.05$) in figure 2B.

Results and Discussion

The study demonstrates significant changes of serum lipid content and serum concentrations of PCBs during pregnancy and lactation. The concentration of sum PCB expressed in ng/g serum lipid showed a significant decrease between week 9-13 and week 35-38 (227.2 ± 24.42, n=10; 147.3 ± 10.8, n=11, respectively; $P<0.05$)(fig. 2A). However, this decrease was not due to an

actual decrease of the PCBs in the blood but was caused by a significant increase in maternal blood lipid content during pregnancy (week 9-13: 0.41 ± 0.02 %, n=10, week 35-38: 0.78 ± 0.03 %, n=11, P<0.05) and which simply "dilute" the existing PCBs in the serum lipid (fig. 1). This is supported by our data presented in figure 2B, expressing the PCB concentration as pg/g fresh weight serum, showing no significant change during the pregnancy. Such a "lipid" dilution of POPs during pregnancy could protect the foetus against high concentrations of contaminants[6].

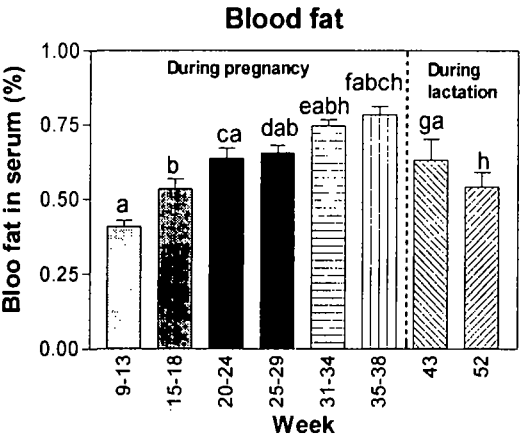

Figure 1. Average serum lipid content (%) in pregnant women from Uppsala, Sweden, during pregnancy and lactation. Week 9-13: n=10; week 15-18: n=10; week 20-24: n=9; week 25-29: n=6; week 31-34: n=11; week 35-38: n= 11; week 43: n=4; week 52: n=5. The second (and following) letter indicates a significant difference from the indicated group (P<0.05).

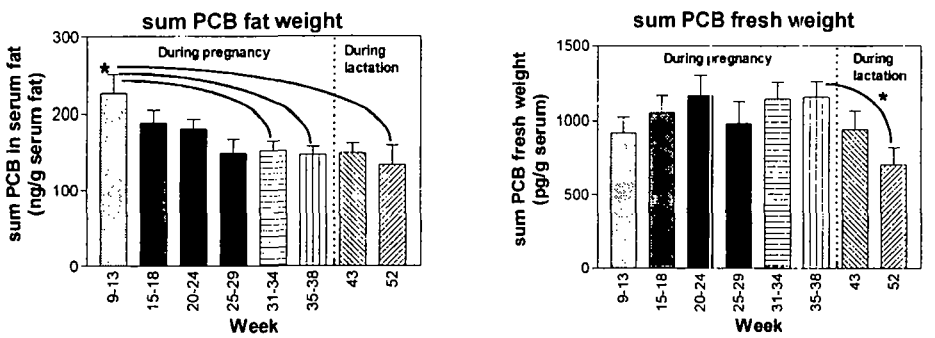

Figure 2. (A, B) Average serum lipid (A) and fresh weight (B) concentration of sum PCB (no 28, 118, 138, 153, 156, and 180) in pregnant women from Uppsala, Sweden, during pregnancy and lactation. See fig. 1 for sample sizes. Significant differences between different groups are indicated with lines and noted by an asterisk (* P<0.05).

Thus, the increase in serum lipid content during pregnancy seems not to be correlated with mobilisation of fat from adipose tissues (and the dissolved PCBs in it) since such a relationship would have resulted in an unchanged PCB concentration expressed as ng/g serum lipid. A possible explanation for this could be that the additional lipid in the blood comes directly from food intake during pregnancy and therefore do not contribute to a significant increase of PCBs. This possibility is of interest when food recommendations are given to pregnant women, advising to avoid food origins with high POP levels. In addition, the implication for epidemiological studies using blood levels during pregnancy to assess exposure is that the blood samples should be collected within a well-defined period during pregnancy and expressed in a way so both fresh and lipid weight can be calculated.

After delivery there is a relatively fast decrease of the serum lipid concentration (fig. 1). A corresponding increase of the PCB concentration in serum lipid does not occur and indicate that there is a net elimination of PCBs from the mother during the nursing period (fig. 2A). Again, this is supported by the data in figure 2B, showing a significant decrease of PCBs in the serum when expressed as fresh weight. The result is well in accordance with previous studies showing a decline of PCBs during nursing[7].

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