Comparison of prenatal and postnatal exposure to persistent organochlorine compounds in male infants born 1997-2001 in two Nordic countries

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Introduction

Due to the high persistence and bioaccumulation of organochlorine compounds along the food chain humans are almost exclusively exposed through their diet. Persistent pollutants accumulate over time in human adipose tissue and can be transferred to the fetus through placenta and to the newborn baby by breastfeeding. Here we present exposures to pentachlorobenzene (PeCB), hexachlorobenzene (HCB), hexachlorocyclohexanes (α -, β -, γ -, δ -, ϵ -HCH), DDT-related compounds (p, p'DDT, o, p'DDT, p, p'DDD, o, p'DDD, p, p'DDE and o, p'DDE), octachlorostyrene (OCS), pentachloroanisole (PCA), aldrin, dieldrin, *cis-, trans*-chlordane (c-CHL and t-CHL), heptachlor (HC), oxychlordane (OXC), *cis-, trans*-heptachlor epoxide (c-HE and t-HE), methoxychlor (MOC), mirex, and endosulfan-I and –II (END-1 and END-2) in male infant cohorts from Denmark and Finland. Exposures were measured in placenta and breast milk.

Material and Methods

The sample collection, preparation, cleanup procedures and HRGC-HRMS analysis conditions have been described elsewhere¹. Organochlorine compounds in 112 placenta and 65 milk samples from Turku, Finland and 168 placenta and 65 milk samples from Copenhagen, Denmark were analysed. For the Finnish placenta, which were analysed in the early phase of the study, OCS values were calibrated according to the added ¹³C aldrin whereas values for Danish placenta samples and the milk samples from both countries were calibrated according to ¹³C OCS. Matlab 6.5 (*MathWorks Inc.*) was used for analyzing the difference between the sample groups. Lilliefors test was used for goodness of fit to a normal distribution (0.05 levels) of the data. Parametric two sample T-test was used for data that were normally distributed at a 0.05 significance level. The nonparametric Wilcoxon rank sum test was used for data that were not significantly normally distributed.

Results and Disscussion

The Lipid contents of the Finish samples were higher than of the Danish samples for both placenta and breast milk. These data have a nearly normal distribution¹. Dieldrin, *p*, *p*²DDE, *p*, *p*²DDT, β-HCH, HCB, END-1, OXC and c-HE were the major pollutants detected. The data of these 8 compounds were not normally distributed but could be better described by γ-distribution. α -, γ-HCH, PeCB, PCA, mirex, *p*, *p*²DDD and MOC could be detected in almost all samples. *o*, *p*²DDT, *o*, *p*²DDD, *o*, *p*²DDE, δ-HCH, t-CHL and c-CHL were detected in some samples but at lower levels than the 8 major pollutants. Aldrin, HC and END-2 were detected only in few samples and their statistic analysis was not done. Significant differences between levels in placenta from the two countries were observed for *p*, *p*²DDE, *p*, *p*²DDD, *p*, *p*²DDT, β-HCH, HCB, dieldrin, OXC, and c-HE and between levels in breast milk samples for PeCB, α -HCH, PCA, *o*, *p*²DDT, t-CHL and c-CHL (p<0.05). Generally, compound levels were higher in the Danish samples than in the Finnish samples. Only mean concentration of mirex was higher in Finnish samples than in the Danish (Table 1). END-1 and OCS were not significantly different between the cohorts (the slight difference in OCS levels in placenta samples might originate from the change in the calibration method). The concentrations of END-1 were linearly correlated to that of OXC (Table 2) in placenta and milk samples in each cohort separately. The slope value for the END-1 /OXC correlation in the Danish samples (1.69 and 1.54) indicates a difference to that in the Finnish samples (2.25 and 2.53), which might reflect some cohort specific character of

OXC vs. END-1 exposure. This might imply a correlation of the sources of these two compounds.

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	Danish placenta		Finish placenta			Danish milk		Finish milk		
	Mean	N ^a	Mean	Na	p-Value	Mean	Na	Mean	Na	p-Value
PeCB	0.56	168	2.55	112	0.24	0.38	65	0.27	65	<0.0001
α-HCH	1.02	168	9.19	112	0.01	0.50	65	0.18	65	<0.0001
β-НСН	9.91	168	5.58	112	< 0.0001	20.55	65	11.68	65	<0.0001
γ-HCH	0.84	168	3.51	112	0.14	0.76	65	0.59	65	0.002
δ-HCH	0.12	124	8.48	96	<0.0001	0.07	41	0.05	25	0.002
PCA	0.21	168	0.18	112	0.49	0.15	65	0.04	64	<0.0001
НСВ	8.00	168	4.83	112	<0.0001	12.82	65	8.51	65	<0.0001
OCS	0.11	168	0.14	112	0.0001	0.20	65	0.20	65	0.57
OXC	1.19	168	1.01	112	0.02	5.20	65	3.93	65	0.0001
c-HE	0.99	168	0.71	112	<0.0001	3.08	65	2.37	65	<0.0001
o,p'-DDE	0.03	164	0.03	110	0.45	0.09	65	0.05	65	<0.0001
p,p'-DDE	47.37	168	21.95	112	< 0.0001	148.73	65	79.04	65	<0.0001
o,p'-DDD	0.07	167	0.08	112	0.37	0.03	61	0.03	55	0.03
p,p'-DDD	0.87	168	0.58	112	<0.0001	0.50	65	0.39	65	0.04
o,p'-DDT	0.07	167	0.07	112	0.65	0.58	65	0.31	64	<0.0001
p,p'-DDT	0.62	168	0.38	112	<0.0001	7.19	65	4.26	65	<0.0001
DDE/DDT	97.58	168	69.27	112	<0.0001	25.12	65	18.49	65	0.001
t-CHL						0.06	55	0.04	53	0.005
c-CHL	7					0.04	32	0.02	30	0.2
END-1	2.18	168	2.26	112	0.56	7.70	65	7.01	65	0.07
Dieldrin	2.74	168	1.39	112	<0.0001	5.84	65	2.78	65	<0.0001
MOC	0.12	168	0.07	112	0.0003	0.06	64	0.11	64	0.0002
Mirex	0.19	168	0.25	112	0.002	0.23	65	0.32	64	0.23
Lipid ^b	1.09	168	1.22	112	<0.0001	2.99	65	4.52	65	<0.0001

Table 1: Exposure differences of organochlorine compounds measured in placenta and milk samples
from Denmark and Finland

Note: ^aPotential numbers of Danish and Finish placenta samples are 168 and 112; Danish and Finish milk samples are both 65, respectively; ^bparametric two sample T-test is used (non-parametric Wilcoxon rank sum test is used for the other all data).

Table 2: Correlations of OXC vs. END-1 (R: correlation coefficient; N: sample number)

Sample	END-1 = Intercept (SE) + Slope (SE) * OXC	R	p-Value	Ν
Danish milk	Y=-1.09(0.55)+1.69(0.10)*X	0.90	<0.0001	65
Danish placenta	Y=0.35(0.09)+1.54(0.07)*X	0.87	<0.0001	168
Finish milk	Y=-1.83(0.57)+2.25(0.13)*X	0.91	<0.0001	65
Finish placenta	Y=-0.30(0.15)+2.53(0.13)*X	0.88	<0.0001	112

Literature:

1. H. Shen, K.M. Main, M. Kaleva, H. Virtanen, A.-M. Haavisto, N.E. Skakkebaek, J. Toppari and K.-W. Schramm. Prenatal organochlorine pesticides in placentas from Finland: exposure of male infants born during 1997–2001. *Placenta* 2005, 26 (6) 512-514.