Exposure to persistent organochlorine pollutants in relation to weight and height at 4 and 7 years of age

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Introduction

There are epidemiological studies indicating that in utero exposure to persistent organochlorine pollutants (POP) are negatively associated with both birth weight and postnatal growth.[1-4] However, there are also studies showing no negative effect on growth and even a positive effect on growth after exposure to polychlorinated biphenyls (PCB).[5, 6]

In Sweden a main exposure route for POP is through consumption of fatty fish from the Baltic Sea (off the eastern coast).[7-9] Fishermen's wives from the Swedish east and west coasts have reported that they consume more than twice as much fish as compared with women from the general population.[10] During the period 1973-1991 infants born to fishermen's wives from the Swedish east coast had an increased risk for lower birth weight as compared with a corresponding group from the Swedish west coast [11], where the fish has been much less contaminated.[12] Case-control studies among the infants born to fishermen's wives from the Swedish east coast indicated an increased risk of lower birth weight among infants born to mothers who reported a relatively high current intake of fish from the Baltic Sea, as well as among infants born to mothers with a relatively high concentration of 2,2',4,4',5,5'-hexachlorobiphenyl (CB-153) in plasma.[13, 14]

The aim of the present study was to investigate whether intrauterine exposure for POP may have negative impact on children's weight and height at 4 and 7 years of age, respectively.

Materials and Methods

The study included 174 fishermen’s wives from the Swedish east coast who had given birth to singleton infant during the period 1973-1991 with either low (55) or normal (119) birth weight, and 88 and 206 corresponding women from the Swedish west coast. A birth weight between 1500 and 2750 g was defined as low birth weight (LBW) and a birth weight between 3250 and 4500 g was defined as normal birth weight (NBW).

Information about the children’s weight and height in the four groups at about 4 and 7 years of age (exact ages for the measurements were always obtained) was collected in two ways. First, the mothers were contacted by telephone and asked to provide this information. Second, child health centers and school health services were contacted and asked to provide the requested information. At the telephone interview the mothers were also asked about smoking habits, education, lactational experiences, and their own height.

Comparisons between the east and west coast cohorts were performed regarding the weight and height of the children at 4 and 7 years of age. In addition, in year 1995 blood samples were collected among 157 east coast women (48 LBW and 109 NBW) and the concentrations of 2,2’,4,4’,5,5’-hexachlorobiphenyl (CB-153) was estimated for the year of childbirth.[13]

Results and Discussion

There were no significant differences between the east and west coast cohorts regarding weight and height at 4 and 7 years of age. However, the adjusted effects on weight and height at 7 years of age was nearly significant for normal birth weight children born in 1973-1980 (difference [east – west]: weight: -1.28 kg, 95% CI –2.58, 0.01; height -1.46 cm, 95% CI –3.06, 0.13, Table).

Table. The effect of cohort affiliation (east versus west coast) on weight and height (at 4 and 7 years of age, respectively) among children with LBW. Corresponding effect among children with NBW. Effect estimates (*ß*) with

95% confidence intervals (CI) obtained from linear regression models are shown. Moreover, the results are divided in two calendar year periods.

	Weight At 4 years of age	**At 7 years of age**
	ß [a] 95% CI	ß [a] 95% CI
LBW	0.17 -0.56, 0.90	0.06 -1.54, 1.66
born 1973-1991	-0.12 -1.07, 0.84	0.13 -1.86, 2.12
- Born 1973-1980	0.20 -0.99, 1.39	-0.14 -2.83, 2.54
- Born 1981-1991	-0.27 -0.84, 0.31	0.17 -0.79, 1.13
NBW	-0.62 -1.60, 0.36	-1.28 -2.58, 0.01
born 1973-1991	-0.06 -0.80, 0.69	1.18 -0.22, 2.58
- Born 1973-1980		
- Born 1981-1991		

	Height At 4 years of age	**At 7 years of age**
	ß [a] 95% CI	ß [a] 95% CI
LBW	0.09 -1.43, 1.61	0.26 -1.80, 2.31
born 1973-1991	-0.50 -2.73, 1.74	-1.33 -3.61, 0.96
- Born 1973-1980	-0.03 -2.32, 2.26	1.51 -1.98, 4.99
- Born 1981-1991	-0.55 -1.53, 0.43	-0.33 -1.42, 0.76
NBW	-1.46 -3.13, 0.22	-1.46 -3.06, 0.13
born 1973-1991	0.09 -1.17, 1.35	0.64 -0.88, 2.17
- Born 1973-1980		
- Born 1981-1991		

[a] Mean differences (cm) between east and west coast cohort children, adjusted for the children's exact age (in month) and height of the mother (<160, 160-169, and ≥170 cm).

The median maternal plasma concentration of CB-153 for year of childbirth was for the mothers who had had a children with LBW 295 ng/g lipid (5th and 95th percentiles; 102, 599) and for the mothers who had had a children with NBW 243 ng/g lipid (5th and 95th percentiles; 97, 719). Although not statistically significant, the association between the estimated plasma concentrations of CB-153 during year of childbirth and the outcomes were in the hypothesized.The NBW children whose mothers had CB-153 concentrations above 250 ng/g lipid tended to have lower birth weight at 4 years of age (adjusted mean difference -0.65 kg, 95% CI -1.36 0.07) as well as at 7 years of age (adjusted mean difference -1.34, 95% CI -2,71, 0.03) than the NBW children whose mothers had lower concentrations of CB-153.

Adding the results from present study to previous studies do not give any clear evidence that in utero or postnatal dietary POP exposure may result in long term growth retardation in humans.

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References

1. Patandin S., Koopman-Esseboom C., de Ridder M.A.J., Weisglas-Kuperus N. and Sauer P.J.J. (1998) *Pediatr Res.* 44: 538-545.

2. Fein G.G., Jacobson J.L., Jacobson S.W., Schwartz P.M., and Dowler J.K. (1984) *J Pediatr.* 105: 315-320.

3. Jacobson J.L., Jacobson S.W. and Humphrey H.E. (1990) *Neurotoxicol Teratol.* 12: 319-326.

4. Blanck H.M., Marcus M., Rubin C., Tolbert P.E., Hertzberg V.S., Henderson A.K. and Zhang R.H. (2002) *Epidemiology.* 13: 205-210.

5. Rogan W.J., Gladen B.C., McKinney J.D., Hardy P., Thullen J., Tingelstad J. and Tully M. (1987) *Am J Public Health.* 77: 1294-1297.

6. Gladen B.C., Ragan N.B. and Rogan W.J. (2000) *J Pediatr.*136: 490-496.

7. Svensson B.G., Nilsson A., Hansson M., Rappe C., Åkesson B. and Skerfving S. (1991) *N Engl J Med.* 324: 8-12.

8. Svensson B.G., Nilsson A., Jonsson E., Schütz A., Åkesson B. and Hagmar L. (1995) *Scand J Work Environ Health.*21: 96-105.

9. Asplund L., Svensson B.G., Nilsson A., Eriksson U., Jansson B., Jensen S., Wideqvist U. and Skerfving S. (1994) *Arch Environ Health.* 49: 477-486.

10. Rylander L., Strömberg U. and Hagmar L. (1995) *Scand J Work Environ Health* 21: 368-75.

11. Rylander L. and Hagmar L. (1995) *Scand J Work Environ Health* 21: 419-426.

12. Bergqvist P. and Bergek S., Hallbäck H., Rappe C., Slorach S. (1989) *Chemosphere.* 19: 513-516.

13. Rylander L., Strömberg U., Dyremark E., Östman C., Nilsson-Ehle P. and Hagmar L. (1998) *Am J Epidemiol* 147: 493-502.

14. Rylander L., Strömberg U. and Hagmar L. (1996) *Scand J Work Environ Health.* 22: 260-266.