ANTHROPOMETRIC AND PSYCHOMETRIC EXAMINATIONS OF CONSCRIPTS BORN TO MOTHERS WITH A HIGH INTAKE OF FISH CONTAMINATED WITH PERSISTENT ORGANOCHLORINES

Lars Rylander, Lars Hagmar

Department of Occupational and Environmental Medicine,
Institute of Laboratory Medicine, University Hospital, SE-221 85 Lund, Sweden

Introduction

In Sweden, the main exposure route for persistent organochlorine compounds (POC), such as polychlorinated biphenyls (PCB), polychlorinated dibenzo-*p*-dioxins (PCDD) and dibenzofurans (PCDF), is through consumption of fatty fish from the Baltic Sea (east coast; 1-3). Exposure to POC have been associated with reduced birth weight and with negative effects on neonatal behavioural performance tests (4-10). Prenatal exposure to PCB from contaminated fish from Lake Michigan also seem to have a long-term impact on intellectual functions (11). The most highly exposed children were at 11 years of age about three times as likely to have low average intelligence scores and about two times as likely to be at least two years behind in reading comprehension, compared to less exposed children. Whether prenatal exposure to POC may cause even more prolonged long-term effects is, however, unknown.

It is known that wives and sisters to Swedish fishermen eat more fish than women from the general population (12, 13). The present study aimed to investigate whether boys born to fishermen's wives and fishermen's sisters from the Swedish east coast at conscript examination (at 18 years of age) had increased risks for growth retardation or for having lowered psychometric scores. For relevant comparison, we used a similar cohort from the Swedish west coast, where the contamination of the fish have been considerably less. In addition, comparisons were made with expected values based on conscript examination data for the general population in the same geographical areas.

Material and methods

Cohorts

By linkage to the Swedish Medical Birth Register (MBR) infants born to fishermen's wives and fishermen's sisters from 1973 and onwards were identified. During the period 1973-1975 there were 287 live-born boys in the east coast cohort and 556 live-born boys in the west coast cohort. By linkage to the Military Service Enrolment Register (MSER) data from the examination of the conscripts during the period 1991-1994 were obtained for 268 (93.4%) of the east and 510 (91.7%) of the west coast cohort boys.

Outcome variables

The MSER included data from medical as well as psychometric examinations of the conscripts. In the present study the following outcome variables were studied:

- Body height, body weight, and body mass index (BMI; kg/m^2).
- Psychometric function classified into nine categories, where 9 is optimal ability.

Statistics

The observed distributions were compared with the expected distributions, which were based on data from MSER for the regional reference populations (i.e. all boys from the east and west coast counties, respectively). Moreover, direct comparisons were performed between the east and west coast cohorts. The results for dichotomized variables were then presented as odds ratios (OR) with confidence intervals. For statistical testing we used t-test for the continuous variables and Mann-Whitney test for the ordinal data. All tests employed were two-tailed.

Results

Body height, body weight, and BMI

At the conscription, the boys in the east coast cohort were somewhat shorter than the boys in the west coast cohort (178.8 cm *versus* 180.1 cm, p=0.01, table 1), whereas no significant differences were seen between the cohorts regarding body weight or BMI. The boys in the east coast cohort had increased BMI as compared with their regional reference population (22.6 kg/m^2 *versus* 22.1 kg/m^2, p=0.03), which was mainly explained by that they tended to be somewhat shorter (178.8 cm *versus* 179.5 cm, p=0.08). Also the boys in the west coast cohort had increased BMI as compared with their regional reference population (22.6 kg/m^2 versus 22.0 kg/m^2, p<0.001). The reason for this was that they were heavier than the regional reference population (73.4 kg versus 71.4 kg, p<0.001).

Table 1 Body height, body weight, and body mass index (BMI) at the conscript examination in the east and west coast cohorts, respectively. Moreover, the expected values (mean) based on data for the regional reference populations are given.

	West coast			**East coast**		
	Mean	(SD)[1,2]	Expected[2]	Mean	(SD)[1,3]	Expected[3]
Height (cm)	180.1	(6.6)	180.0	178.8	(6.2)	179.5
Weight (kg)	73.4	(11.7)	71.4	72.2	(13.0)	71.2
BMI (kg/m^2)	22.6	(3.2)	22.0	22.6	(3.7)	22.1

[1] Direct comparisons between the east and west coast cohorts (Height p=0.01; Weight p=0.2; BMI p>0.2).

[2] Comparisons between the observed and the expected distributions for the west coast cohort (Height p>0.2; Weight p<0.001; BMI p<0.001).

[3] Comparisons between the observed and the expected distributions for the east coast cohort (Height p=0.08; Weight p>0.2; BMI p=0.03).

Psychometric scoring
There was no significant difference between the cohorts regarding the psychometric score distributions (p=0.09, table 2). Analyzed as a dichotomized variable, the east coast cohort did not have increased risk of having scored below 5 as compared with the west coast cohort (OR 1.0; 95% CI 0.7-1.5). Moreover, none of the cohorts differed from their regional reference population in this respect.

Table 2 Results of the psychometric tests in the east and west coast cohorts, respectively. Moreover, the expected distributions (%) based on data for the regional reference populations are given.

Score	West coast			East coast		
	N	%[1,2]	Expected[2]	N	%[1,3]	Expected[3]
1	16	3.4	1.7	5	2.0	2.4
2	33	7.0	7.0	19	7.6	6.2
3	57	12.0	11.6	33	13.1	10.7
4	103	21.7	20.6	48	19.1	15.2
5	109	23.0	21.4	45	17.9	21.6
6	91	19.2	19.8	46	18.3	18.8
7	44	9.3	13.1	27	10.8	16.5
8	20	4.2	4.1	24	9.6	6.9
9	1	0.2	0.7	4	1.6	1.7

[1] Direct comparison between the east and west coast cohort distributions (p=0.09).
[2] Comparison between the observed and expected distributions in the west coast cohort (p=0.11).
[3] Comparison between the observed and expected distributions in the east coast cohort (p>0.2).

Discussions
As compared with the west coast cohort, the boys in the east coast cohort were, at conscript testing, somewhat shorter, whereas the cohorts did not differ in the psychometric test. The mean height in the east coast cohort did, however, not significantly differ from the mean height among their regional reference population.

The major weakness in the present study is the lack of individual exposure data. It is, however, known that wives and sisters to Swedish fishermen have a relatively high intake of locally caught fish (4, 13). Fishermen's wives from the Swedish east coast had, in the mid nineties, been shown to have about 30% higher concentrations of PCB in plasma as compared with women from the general population (14). In addition, in the early seventies the fish from the Baltic Sea contained much higher concentrations of PCB (15). This taken together, gives circumstantial evidence that a large number of the boys in the east coast cohort had had a substantial prenatal exposure for POC.

Prenatal exposure for PCB have been associated with negative long-term impact on intellectual function, at least until 11 years of age (11). We have for the presently studied boys no data on their intellectual function at that age, but the results do not indicate that prenatal exposure for POC through their mothers consumption of fish from the Baltic Sea affects the psychometric function at 18 years of age. Whether the pattern is the same for prenatally exposed girls remains unknown.

Acknowledgements
This work was financed by grants from the Swedish Foundation for Strategic Environmental Research, Swedish National Environmental Protection Board, the Swedish Work Life Council, and the Medical Faculty, Lund University. We are also grateful to professor Bengt Källén and Dr Gunnel Sandegård for valuable help.

References

1. Svensson BG, Nilsson A, Hansson M, Rappe C, Åkesson B, Skerfving S; *N Engl J Med* **1991,** 324, 8-12.
2. Svensson BG, Nilsson A, Jonsson E, Schütz A, Åkesson B, Hagmar L; *Scand J Work Environ Health* **1995**, 21, 96-105.
3. Asplund L, Svensson BG, Nilsson A, Eriksson U, Jansson B, Jensen S, et al; *Arch Environ Health* **1994**, 49, 477-86.
4. Rylander L, Strömberg U, Hagmar L; *Scand J Work Environ Health* **1995**, 21, 368-75.
5. Rylander L, Strömberg U, Hagmar L; *Scand J Work Environ Health* **1996**, 22, 260-6.
6. Rylander L, Strömberg U, Dyremark E, Nilsson-Ehle P, Östman C, Hagmar L; *Am J Epidemiol* **1998**, 147, 493-502.
7. Taylor PR, Stelma JM, Lawrence CE; *Am J Epidemiol* **1989**, 129, 395-406 (1989).
8. Fein GG, Jacobson JL, Jacobson SW, Schwartz PM, Dowler JK; *J Pediatr* **1984**; 105, 315-20.
9. Jacobson SW, Fein GG, Jacobson JL, Schwarz PM, Dowler JL; *Child Dev* **1985**, 56, 583-60.
10. Patandin S, Koopman-Esseboom C, de Ridder MAJ, Weisglas-Kuperus N, Sauer PJJ; *Pediatr Res* **1998**, 44, 538-45.
11. Jacobson JL, Jacobson SW; *N Engl J Med* **1996**, 335, 783-9.
12. Rylander L, Hagmar L; *Scand J Work Environ Health* **1995**, 21, 419-26.
13. Rylander L, Strömberg U, Hagmar L; *Chemosphere* (in press).
14. Grimvall E, Rylander L, Nilsson-Ehle P, Nilsson U, Strömberg U, Hagmar L, et al; *Arch Environ Contam Toxicol* **1997**, 32, 329-36.
15. Bignert A, Olsson M, Persson W, Jensen S, Zakrisson S, Litzén K, et al; *Environ Pollution* **1998**, 99, 177-98.