Neurophysiological Measures of Developmental and Chronic exposure to Methylmercury

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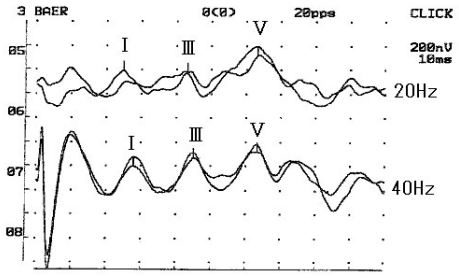


Background

Neurophysiological methods have been applied to detect subclinical changes associated with exposures to developmental neurotoxicant in asymptomatic children. A review of neurophysiological findings in methylmercury neurotoxicity in children and adolescents suggested differences between the effects of prenatal and postnatal exposures. We therefore examined a group of adult members of the Faroese whaling society with high life-time exposure to methylmercury.

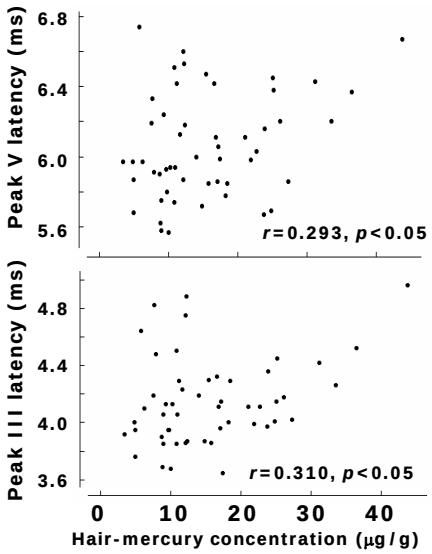
Methods

Brainstem auditory evoked potential (BAEP) latencies were examined in 50 male whalers aged 21-81 (mean, 56) years, who donated a hair sample for mercury analysis six years previously. Hair, toenails, and whole blood were collected for mercury analysis at the time of clinical examination.

In recording BAEP, click stimuli were presented monaurally at a rate of 20 or 40 Hz through shielded earphones. The peaks of I, III, and V primarily represent volume-conducted electrical activities from the acoustic nerve, pons, and midbrain, respectively.

Results

Delays in the peak III and peak V latencies of the BAEP were significantly related to the prior hair-mercury concentration (range 3.4 - 43.7 μg/g; median 13.2 μg/g) in the male whalers (see Figure below). These significant associations remained after adjustment for age, thus suggesting a possible adverse effect of life-time methylmercury exposures.



Correlations between hair-mercury concentrations 6 years before and peak III and peak V latencies of brainstem auditory evoked potential in adult male whalers

Discussion

In the Faroese whalers, peak III and V latencies of the BAEP were significantly though weakly associated with a measure of methylmercury exposure. This finding is consistent with the results observed in Faroese children[a], Madeiran children[b], and Ecuadorian children[c]. Accordingly, both postnatal and prenatal exposures to methylmercury appear to affect the auditory pathway from the acoustic nerve to the midbrain.

In reviewing the neurophysiological effects of methylmercury, the auditory pathway from the acoustic nerve to the pons appear to be permanently affected by prenatal methylmercury exposure, and the change in the auditory pathway from the pons to the midbrain may also be sensitive to postnatal methylmercury exposure at adolescence and adulthood (see Table below). Although no prenatal exposure information is available. it is likely that the Faroese whalers were exposed to methylmercury prenatally, but the postnatal exposure is known only from a sample 6 years ago and current samples. The relative impact of prenatal and postnatal exposures therefore cannot be determined.

[a]Murata et al: Neurotoxicol Teratol 1999;21:471-2.[b]Murata et al: Neurotoxicol Teratol 1999;21:343-8. [c]Counter: J Occup Environ Med 2003;45:87-95.

Implications

Although the sample size of this pilot study was small and it is probable that these whalers had been exposed to methylmercury prenatally, it adds to the evidence that delays in the brainstem evoked potentials may function as objective measures of methylmercury neurotoxicity.

Neurophysiological Findings on Methylmercury Neurotoxicity in Humans

Outcome parameters	Exposure	Effects[a]	References
Somatosensory evoked potentials (mainly, N19 latency)	Prenatal	↑(MD)	Inayoshi et al, 1988
	Postnatal	C(MD)	Tokuomi et al, 1982
Visual evoked potentials	Postnatal	↑(MD)	Iwata 1980; Imai et al, 1991
	Pre- & postnatal	↑(CS)	Weihe et al, 2002
BAEP:			
I-III interpeak latency	Prenatal	↑(MD,Co)	Hamada et al, 1982; Murata et al, 1999a, 2004b
III-V interpeak latency	Pre- & postnatal	↑(CS)	Weihe et al, 2002; Counter 2003; Murata et al, 2004a
	Postnatal	↑(Co)	Murata et al, 2004b
Event-related potential (P300)	Postnatal	↑(MD)	Kondo et al, 1995
Heart rate variability	Prenatal	↓(MD,Co)	Oka et al, 2002; Grandjean et al 2004; Murata et al, 2006

[a]The effect was expressed as significant increase (↑) and decrease (↓), and change in the waveform (C) among cases with Minamata disease (MD) and children of the cross-sectional (CS) or cohort (Co) study. (***Am J Ind Med* 2007 April 20)**