

ENVIRONMENTAL EXPOSURE OF CHILDREN TO POLYCHLORINATED BIPHENYLS AND DEFICITS IN COCHLEAR STATUS

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Background and aims: The mammalian auditory system forming a part of CNS, with its complex development, is a typical target of neurodevelopmental toxicity. Polychlorinated biphenyls (PCBs), a member of neurodevelopmental toxicants, can induce hearing impairment in rats (Crofton 2000) and humans (Trnovec 2008, 2010). The underlying process is a damage of the outer hair cells in cochlea. A mode of action based on deficit of thyroid hormones proposed for rodents does not hold for humans (Crofton and Zoeller 2005). In our cross-sectional studies the level of otoacoustic emissions (OAE), a marker of cochlear status, was associated with actual PCB exposure and not related to thyroid hormones. However, no data on prenatal exposure, a marker of which is PCB cord blood concentration, were available (Trnovec 2008, 2010).

Methods: Longitudinal cohort study, complex audiological examination.

Results: In a random cohort of 485 infants exposed to PCBs from Michalovce district, eastern Slovakia (Hertz-Picciotto 2003), children were audiotically examined at the age of 45 months. The transient evoked (TE) OAE and distortion product (DP) OAE were related to PCB serum concentration at birth, 6, 16 and 45 months of age. The gender specific deficits were manifested in decreased amplitudes of both TEOAE and DPOAE in a similar way as with noise trauma or effect of cochleotoxic substances. For DPOAE two primary tone f₂ frequencies, 3364 and 2828 Hz for left and right ears, resp., showed highest sensitivity from 11 tested frequencies. The BMDL ($p_0=0.1$; BMR=0.05) for this effect in left ears of males is 419.3 ng PCB/g lipids and an alarming MOE = 1.11 at median PCB serum concentration of 377.4 ng PCB/g lipids.

Conclusions: Multivariate regression has shown that the OAE amplitudes were significantly related to PCB exposure at 16 and 45 months. There was no relationship with neurodevelopmentally relevant prenatal time period.