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Environmental PHAHs as developmental toxicants

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PHAH-, particularly PCB-associated neurodevelopmental adversity has been observed at environmental levels of exposure. Despite some discrepancies in detail neurocognitive delay or deficit was found to be associated with PCB-exposure in most pertinent cohort studies. Our own experience is based on two birth cohorts recruited between 1993/1995 and between 2000/2002. In the earlier cohort pre-/perinatal exposure was measured in terms of indicator PCBs (IUPAC-Nos. 138, 153, 180), whereas in the later cohort a larger spectrum of PCBs and PCDD/Fs was measured in maternal blood and milk. Using the Bayley- and the Kaufman-Scales neurodevelopmental adversity was observed at a median PCB-level of 404 ng/g milk fat in the earlier study. At a median PCB-level of 177 ng/g in the 2nd study no exposure-related neurodevelopmental adversity was seen, any more. Thus PCBs or PCB-associated PHAHs may have an adverse impact on neurocognitive development at elevated levels of exposure clearly exceeding 177 ng/g milk fat, only. The mechanistic basis of such adversity is unclear. Endocrine dysfunction is being discussed in this regard. Therefore, in the recent cohort, both thyroid and gonadal hormones were measured and related to PCBs and dioxins in maternal blood and milk. No consistent associations were seen between PHAHs and thyroid hormones, but robust negative associations were found between PCBs/dioxins and both testosterone and estradiol in cord serum. The clinical implications of these findings, which have not been described before, are unknown and must be clarified in follow-up studies.