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Prenatal and Postnatal Exposure to Persistent Organic Pollutants and Infant Growth: A Pooled Analysis of Seven European Birth Cohorts


Table S1. Description of the birth cohorts with biological PCB-153 and p,p’-DDE exposure biomarkers included in the present study.*

Table S2. Chemical-analytical methods and detection/quantification limits of the birth cohorts.*

Figure S1. A) Conceptual representation of the pharmacokinetic model and B) examples of blood POP levels in mothers and infants.*

*Adapted from Verner et al. 2013. Reproduced with permission from Environmental Health Perspectives. AUC area under the curve. Simulations were carried out with a maternal daily dose of 10 ng/kg body weight/day. Model assumptions: exclusive maternal exposure through diet; complete gastrointestinal absorption; exclusive and homogenous distribution of POPs in maternal and child lipids with unlimited transplacental diffusion (due to lipophilicity). POPs elimination (e.g., fecal excretion, metabolism) was based on published half-life values. Breast milk consumption rate was based on exclusive/partial breastfeeding data from the general population (Arcus-Arth et al. 2005).
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Figure S2A. Directed acyclic graph of the association between infant growth and total exposure.

Figure S2B. Directed acyclic graph of the association between infant growth and prenatal exposure.*

*Birth weight and gestational age are intermediate variables between prenatal POP exposure and infant growth. We are interested in the effect of exposures on infants’ postnatal growth, not one that may be merely a continuation of prenatal growth mediated via birth weight. We included these variables in the model to close the pathway from prenatal exposure to infant growth via birth weight so that the model estimates only the direct association between prenatal exposure and postnatal growth.

Figure S2C. Directed acyclic graph of the association between infant growth and postnatal exposure.

Table S5. Biomarker concentrations and cord blood POPs concentrations estimated in the pharmacokinetic model (ng/g lipid).

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References