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Influence of dioxins and PCBs on markers of immune function and respiratory infections in Swedish infants

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Early development of thyroid and immune systems in animals is sensitive to polychlorinated biphenyls (PCBs), dibenzo-p-dioxins (PCDDs), and dibenzofurans (PCDFs). These persistent organic pollutants (POPs) were measured in maternal serum in late pregnancy and in breast milk from 323 primiparous women from Uppsala County. In utero exposure of infants was estimated from POP levels in maternal serum, and post-natal exposure from levels in breast milk, number of days of nursing and extent of nursing each day. Infant thyroid hormones were measured 4 weeks and 3 months after delivery, and white blood cell counts and lymphocyte subsets 3 months after delivery. POP-exposure was not associated with thyroid hormone levels. However, a positive association between in utero exposure to PCB congeners CB 28, 52 and 101 (LPCB) and numbers of lymphocytes and monocytes was found. Furthermore, in utero exposure to mono- and di-ortho PCBs was negatively associated with percentage of CD8+ cytotoxic T-cells. The odds ratio for respiratory infections was significantly increased for infants with the highest in utero LPCB exposure. Infants with the highest in utero and post-natal di-ortho PCBs exposure had lowered risks of respiratory infections. Our results suggest that the immune system is more sensitive to POP exposure than the thyroid system. Observed alterations in immune markers were within normal range, and no obvious connection with risk of respiratory infections was evident. Respiratory infections early in life are risk factors for child asthma development. Follow-up of this cohort will show if POPs influence asthma development.