Persistent environmental toxicants in human breast milk and infant growth

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Toxicants and metabolic outcomes

- Toxicants are passed to infants via breastmilk
- Most studies look at levels in maternal pregnancy serum or cord blood
Objective

• Explore levels of 26 toxicants in human breast milk and their association with rapid infant growth
Study design: HUMIS-NoMic Study

• Prospective cohort of 2,606
• Enrolled 2 weeks postpartum between 2002-2008
• Questionnaires completed on child’s weight and length, pregnancy information, mothers’ health status
  – Linked to Norwegian Medical Birth Registry
• 2400 have milk samples
  – Toxicants analyzed in 1300
  – Milk lipids analyzed in 800
• Also have collected data on gut microbiota, metabolomics, diet, neuropsychological outcomes, immune outcomes
Characteristics

- 800 milk samples analyzed
  - Obese women oversampled
- Excluded duplicates and twins
  - > 789 singleton infants
- 19.2% rapid growers
- Mean maternal age 29.6 years
- Median pre-pregnancy BMI 24.0
- 45.3% of mothers were overweight or obese
- Rapid growers more likely to be first-born
Exposures and outcomes

172 toxicants

Discarded those with >50% missing

14 PCBs
4 OCPs
6 BDEs
2 PFCs

Discarded those with incl. prob. <0.1

Multiple imputations
Variable selection (spike Slab GAM)

HCB, β-HCH, PCB74

Single imputations

Raid growth between 0 and 6 months of life

Logistic regression
Final exposures: Persistent organic pollutants (POPs)

- Now largely banned, but previously widely used for pest control, industrial uses
- Accumulate and biomagnify in food chain
- Highly lipophilic
- Endocrine-disruptors
Results: Logistic regression

- Adjusted for maternal smoking, parity, sex, cumulative breastfeeding, education, pre-pregnancy BMI, maternal age, gestational weight gain, birth weight, gestational age, preterm status
IQR-adjusted results

- Adjusted for maternal smoking, parity, sex, cumulative breastfeeding, education, pre-pregnancy BMI, maternal age, gestational weight gain, birth weight, gestational age, preterm status
β-HCH and growth outcomes

• Previous studies on β-HCH and growth outcomes have been equivocal
  – Prenatal exposure linked to increased BMI z-scores and risk of overweight at 7 years (Agay-Shay 2015)
  – Others have found no association with growth outcomes (Cupul-Uicab 2013, Mendez 2011, Eggesbø 2009)

• In adults, serum β-HCH linked to increased BMI, insulin resistance, diabetes (Arrebola 2014, Dirinck 2011, Everett 2010)
POP and programming

- HCB and PCBs in breast milk associated with lower birth weight, restricted fetal and infant growth (Iszatt 2015, Stigum 2015, Govarts 2012, Eggesbø 2009)
- Perinatal exposure to endocrine-disrupting chemicals leads to physiologic changes that predispose to obesity (Vafeiadi 2015, Tang-Peronard 2011)
β-HCH and programming

Perinatal β-HCH exposure

Critical window

Non-rapid or reduced growth

Later metabolic outcomes?
Conclusions

• β-HCH exposure via breast milk are associated with reduced odds of rapid growth in infancy
• May be related to decreased infant growth

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