Hexachlorobenzene and risk of small for gestational age
Merete Eggesbo* (Norwegian Institute of Public Health, Oslo, Norway), Hein Stigum (Norwegian Institute of Public Health, Oslo, Norway) Anuschka Polder (National Veterinary Institute, Oslo, Norway), Matt Longnecker (National Institute of Environmental Health Sciences Research Triangle Park, USA), Magne Aldrin (University of Oslo, Norway).

Background: Experimental studies have reported dose-dependent destruction of ovarian primordial germ cells even at low doses of Hexachlorobenzene (HCB), a fungicide and an ubiquitous environmental contaminant. Population based studies have reported conflicting results. The aim of this study was to investigate the association between HCB and SGA with more extensive control for possible confounders than most previous studies.

Methodology: A case control study consisting of 66 SGA cases and 402 controls from the Norwegian HUMIS study. HCB, p,p'-DDE and the PCB congeners #28,52,74,101,170,118 were measured in mothers' milk. HCB was studied as a continuous variable, since no threshold effects were observed (GAM model).

Results: Median HCB levels 11 μg/kg lipid (range 4-42). The risk of SGA increased with a factor of 1.7 for a 10 unit increase in HCB (OR 1.7, CI 1.1 to 2.5). Adjusting for maternal age, siblings, maternal educational level, prepregnant weight and height and smoking increased the effect: OR 2.3 (1.1 to 4.5). Including PCBs and DDE further increased the estimates: OR 4.1 (1.6 to 10.6).

Implications: This study indicates that HCB may increase the risk of SGA. This could be due to a toxic effects to the ovary, if so, the effect may have occurred in the past when the levels were higher. The current levels in the mothers may reflect past HCB levels, since their dietary habits may be assumed to have been rather consistent. Since environmental toxicants coexist in exposure sources, the possibility of confounding by unidentified toxicants cannot be excluded.