washing by flow water. Another source of Aluminum comes from the discharge of stations of water purification that contain huge amount of aluminum sulfate (alum) which are used as a coagulant for suspended solid particles. In addition, the extensive use of the clay in the industry of packed bricks along Sabal drainage canal is considered as anther sources of Aluminum. The field and laboratory studies of water samples and different fish tissues (muscles, liver and ovary) collected over one year from Sabal drainage revealed the following items: 1. Water samples collected from Sabal drainage canal have higher levels of Aluminum if compared to those collected from other localities. 2. The levels Aluminum in the muscle, liver and the ovary of Tilapia zillii collected from Sabal drain are higher in comparison to those detected in fish from other localities. Moreover, the level of Al exceeds the international permissible limits. From a public health standpoint, the increased concentrations of Aluminum in water samples and the muscles, liver and the ovary of T. zillii from Sabal drain is a matter of concern as this metal represent a health risk for fishermen and human consumers. Therefore, the following suggestions are important: 1. Constant elimination of illegal aluminum industries in the area of Sabal drainage canal is necessary. 2. It is very important to replace the smaller and old stations of sewage treatment by another modern type capable of collecting and treating huge amount of sewage, with high efficiency of treatment and purification.

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Multi-organ effects of prenatal alcohol exposure: long-term implications

R. Harding[1], K. Kenna[1], V. Stokes[1], R. DeMatteo[1], H. Parkington[1], M. Tare[1], S. Gray[1], A. Bocking[2], J. Brien[3], F. Sozo[1], S. Rees[4], D. Walker[1]

[1]Biomedical Sciences, Monash University, Melbourne Australia; [2]Dept BGYN, University of Toronto, Canada; [3]Dept Pharmacol & Toxicol, Queen's University, Kingston, Canada; [4]Dept Anatomy, University of Melbourne, Australia

Objective: During human pregnancy, episodic exposure of the fetus to ethanol (EtOH) is not uncommon. However, the effects of episodic alcohol exposure on organ development and functional outcomes are not well understood. As fetal EtOH exposure may be a potent but little recognised cause of developmental programming, we have used a sheep model to explore the effects of prenatal EtOH exposure on a range of tissues both before and after birth. Our objective was to determine the effects of repeated EtOH exposure during the last trimester equivalent on the development and postnatal function of major organs.

Methods: Pregnant ewes with implanted jugular vein catheters were infused with either saline or EtOH (0.75 g/kg) for one hour daily from 95 days of gestational age (DGA) until 133 DGA (term ~147 DGA). At 125 DGA, the animals underwent surgery for the insertion of a fetal arterial catheter for blood sampling and arterial pressure/heart rate recording, and an amniotic catheter. One group of animals (alcohol exposed and controls) underwent necropsy at 134 DGA, while another group were born and studied at 8 weeks after birth.

Results: Maternal and fetal plasma EtOH concentrations reached maximal values of ~0.11 g/dL at 1 hour after infusion onset, but returned to baseline within 8hours. EtOH exposure resulted in transient mild hypoglycemia in the ewe and fetus as well as maternal acidemia and delayed fetal hypoxemia. EtOH exposure did not affect fetal body or organ weights, and no gross anomalies were seen. In fetal lungs, EtOH exposure led to increased collagen mRNA expression and collagen deposition, and reductions in mRNA expression of surfactant proteins A and B to ~33% of control levels. mRNA expression of pro-inflammatory cytokines IL-1β and IL-8 was reduced by ~90% compared with controls[1]. In the fetal kidneys, there was an 11% reduction in nephron endowment following EtOH exposure[2]. In fetal arteries from several vascular beds, EtOH led to altered smooth muscle reactivity and endothelial function, and a profound increase in arterial stiffness. In the fetal brain, we found that EtOH led to thinning of the corpus callosum and a decreased number of microglia in white matter.

Conclusions: Daily exposure of the fetus to EtOH during the later stages of gestation can impact upon the development of multiple organs. Mechanisms may include fetal hypoxemia, hypoglycemia and oxidative stress. If the alterations in organ structure persist after birth, they could impair health in later life. On-going studies are examining the postnatal effects of prenatal EtOH exposure.

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Is pre- or post-natal secondhand smoke exposure associated with childhood overweight? Hong Kong's "Children of 1997" birth cohort

M.K. Kwok, C.M. Schooling, T.H. Lam, G.M. Leung

Department of Community Medicine and School of Public Health, Li Ka Shing Faculty of Medicine, The University of Hong Kong, Unit 624-627, Level 6, Core F, Cyberport 3, 100 Cyberport Road, Hong Kong, China

Objective: Paternal smoking,[1,2] and perhaps pre-natal secondhand smoke (SHS) exposure,[3] may be modifiable causes of childhood overweight. However, social patterns of smoking and childhood overweight make such observations open to residual confounding. Studies from non-Western socio-historical contexts such as Hong Kong, where most women do not smoke (<4%), are valuable in clarifying whether the association between SHS exposure and childhood overweight observed in

western societies is biologically-mediated or socio-economically confounded, and whether there are any critical windows for SHS exposure (i.e. prenatal vs. postnatal).

Methods: We used multivariable linear regression to examine the adjusted associations of SHS exposure with body mass index (BMI) and height z-scores at 7 years relative to the 2007 World Health Organization growth reference, among 6,713 children with non-smoking mothers (74% follow-up) from a population-representative, Hong Kong Chinese birth cohort, ''Children of 1997'', born in April and May 1997. We classified SHS into mutually exclusive categories representing increasing doses from different sources (paternal smoking or any household smoking). Appropriate institutional ethics committee clearance and participants' informed consent were obtained.

Results: Compared to infants with no pre-natal SHS exposure from non-smoking households, infants from non-smoking households exposed to pre-natal SHS had higher BMI z-scores, but not height, at 7 years (mean difference 0.08, 95% confidence interval (CI) 0.003 to 0.15) as did infants from households where fathers smoked daily (0.12, 95% CI 0.04 to 0.21), adjusted for sex, birth order, highest parental education and mother's place of birth.

Conclusions: Our findings, although preliminary, suggest that an association of pre-natal SHS exposure and paternal smoking with child overweight could possibly be biologically mediated, with the key exposure perhaps before birth. Given the known harms of smoking, reducing SHS exposure from conception as a precautionary action for childhood overweight might be warranted. *Acknowledgements*: We thank colleagues at the Student Health Service and Family Health Service of the Department of Health for their assistance and collaboration and Connie Hui for assisting the record linkage.

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Organochlorine compounds and rapid early growth: findings from the INMA Environment and Childhood Project

M.A. Mendez, R. Garcia-Esteban, M. Guxens, M. Kogevinas, J. Sunyer

Center for Research in Environmental Epidemiology (CREAL) and Municipal Institute of Medical Research (IMIM-Hospital del Mar), 88 Dr. Aiguader Street, Barcelona, Spain

Objectives: Rapid growth in the first months of life is a strong risk factor for long-term obesity, although relatively little is known about factors that promote this growth pattern. It has been hypothesized that chemicals with endocrine disrupting properties may influence obesity risk, but empirical data is limited. This paper explores associations between several organochlorine compounds (OCs) and rapid infant growth as a marker of long-term obesity risk.

Methods: Data come from the Spanish INMA-INfancia y Medio-Ambiente (Environment and Childhood) Project in Sabadell, Barcelona, a birth cohort which recruited 657 women during the 1st trimester of pregnancy with subsequent follow-up to assess infant health. Average daily weight gain during the first 6 months was estimated, and rapid growth defined as >0.67 SDs of weight gain/day. Multivariable logistic regression was used to evaluate odds of rapid growth associated with lipid-adjusted levels of several OCs measured in maternal first trimester serum (DDE, DDT, PCBs, bHCH and HCB), adjusting for covariates including birthweight, gestational age, child sex, breastfeeding duration, parity, and maternal age, education, smoking, and weight gain in pregnancy. Appropriate institutional ethics committee clearance and participants' informed consent were obtained.

Results: Overall, maternal OC levels were not significantly higher among rapid growers. Among mothers of normal pre-pregnancy weight (body mass index $<25\,kg/m^2$), however, the prevalence of rapid growth was significantly higher in the top (39.8%) vs. bottom (19.2%) quartile of maternal DDE ($P < 0.05$). While there was no association among infants of mothers overweight or obese prior to pregnancy, there was a significant positive association between maternal DDE levels and rapid growth among those born to mothers of normal weight (interaction $P < 0.05$ for overweight $\times$ DDE). Other contaminants were not meaningfully associated with rapid early growth.

Conclusions: This analysis suggests elevated maternal DDE levels may be a risk factor for rapid growth in infancy. This finding is consistent with two recent studies (Verhulst *et al*, 2009; Karmaus *et al*, 2009) which found intrauterine exposure to DDE, but not several other chemicals, to be associated with obesity later in life. Future studies are needed to better understand the potential role of environmental obesogens in the obesity epidemic, including their possible role in rapid early weight gain. Support: Spanish Ministry of Health, Instituto de Salud Carlos III, the Generalitat de Catalunya-CIRIT, and the European Union projects EARNEST FOOD-CT-2005-007036 and NewGeneris FOOD-CT-2005-01632.

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Long-term effects of maternal nicotine exposure during lactation on body adiposity, lipid profile and hypothalamic leptin signaling pathway

E. Oliveira[1], C.R. Pinheiro[1], A.P. Santos-Silva[1], Lima NS, G. Castro, J.F. Nogueira Neto, M.C.F. Passos[2], E.G. Moura[1], P.C. Lisboa[1]