Kadmium och hälsorisker

En rad rapporter visar eller misstänker samband mellan kadmium och följande hälsorisker:

- Skador på njurar
- Skador på ben
- Cancer
- Ökad dödlighet
- Stör endokrina körtlar
- Stör fortplantning
- Påverkar barns utveckling
- Kvinnor mer känsliga
- Skador på embryo
- Kan orsaka missbildningar
- Överförs genom moderkakan med navelsträngsblod till foster
- Minskad storlek hos nyfödda

Introduction*

Human exposure to the toxic metal, cadmium (Cd), occurs mainly through food, such as cereals, seafood and offal, and inhalation of tobacco smoke (Järup and Åkesson 2009)

Once absorbed, Cd has a long half-life in the body, especially in the kidneys. Chronic Cd exposure has been shown to adversely affect kidney and bone, and to increase the risk of cancer (Straif et al. 2009) and overall mortality (Järup and Åkesson 2009).

Cd also functions as an endocrine disruptor (Ali et al. 2010; Johnson et al. 2003), and may thus affect reproduction and child development (Henson and Chedrese 2004).

In general, women are more susceptible to Cd toxicity, mainly due to increased intestinal uptake of Cd given low iron stores (Berglund et al. 1994; Nishijo et al. 2004a; Vahter et al. 2007; Åkesson et al. 2002), which are more prevalent in women than in men.

There is, however, little information on effects of Cd exposure in early life. Cd has been shown to be both embryotoxic and teratogenic in a variety of animal species (Thompson and Bannigan 2008), but this has not yet been confirmed in humans.

Cadmium accumulates in human placenta (Kippler et al. 2010a; Osman et al. 2000), but still it should be noted that the placenta is not a complete barrier as cadmium concentrations in cord blood increase with maternal exposure (Kippler et al. 2010a).

There is increasing evidence of associations between maternal Cd exposure and adverse pregnancy outcomes, such as reduced size at birth (Galicia-Garcia et al. 1997; Llanos and

Ronco 2009; Nishijo et al. 2004b; Salpietro et al. 2002; Shirai et al. 2010; Zhang et al. 2004) and pre-term delivery (Nishijo et al. 2002).

However, studies that have reported associations included very few women (44-78 women)and associations with the same outcomes varied markedly among the studies. Residual confounding cannot be excluded since very few covariates were considered. In contrast, two larger studies (106-262 women) found no association between maternal Cd exposure and size at birth (Odland et al. 1999; Osman et al. 2000).

In the present study, we took advantage of our large, population-based, longitudinal mother-child cohort in Bangladesh to assess the effects of maternal Cd exposure on size at birth. We previously reported that arsenic (As) exposure is associated with reduced size at birth (Rahman et al. 2009).

Additionally, pregnant women in this rural area had elevated concentrations of Cd in their placentas, and placental Cd was inversely associated with zinc (Zn) in cord blood, suggesting a possible effect of Cd on the transfer of Zn to the fetus (Kippler et al. 2010a). Thus, it is possible that Cd has adverse effects on pregnancy outcomes as well.

* Från " *Maternal Cadmium Exposure During Pregnancy and Size at Birth: A Prospective Cohort Study", sid 4 och 5,*

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