

Effect of DMPS and DMSA on the Placental and Fetal Disposition of Methylmercury.

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Methylmercury (CH_3Hg^+) is a serious environmental toxicant. Exposure to this metal during pregnancy can cause serious neurological and developmental defects in a developing fetus. Surprisingly, little is known about the mechanisms by which mercuric ions are transported across the placenta. Although it has been shown that 2,3-dimercaptopropane-1-sulfonate (DMPS) and 2,3-dimercaptosuccinic acid (DMSA) are capable of extracting mercuric ions from various organs and cells, there is no evidence that they are able to extract mercury from placental or fetal tissues following maternal exposure to CH_3Hg^+ . Therefore, the purpose of the current study was to evaluate the ability of DMPS and DMSA to extract mercuric ions from placental and fetal tissues following maternal exposure to CH_3Hg^+ . Pregnant Wistar rats were exposed to CH_3HgCl , containing [^{203}Hg], on day 11 or day 17 of pregnancy and treated 24h later with saline, DMPS or DMSA. Maternal organs, fetuses, and placentas were harvested 48h after exposure to CH_3HgCl . The disposition of mercuric ions in maternal organs and tissues was similar to that reported previously by our laboratory.

The disposition of mercuric ions in placentas and fetuses appeared to be dependent upon the gestational age of the fetus. The fetal and placental burden of mercury increased as fetal age increased and was reduced by DMPS and DMSA, with DMPS being more effective.

The disposition of mercury was examined in liver, total renal mass, and brain of fetuses harvested on gestational day 19.

On a per gram tissue basis, the greatest amount of mercury was detected in the total renal mass of the fetus, followed by brain and liver. DMPS and DMSA reduced the burden of mercury in liver and brain while only DMPS was effective in the total renal mass.

The results of the current study are the first to show that DMPS and DMSA are capable of extracting mercuric ions, not only from maternal tissues, but also from placental and fetal tissues following maternal exposure to CH(3)Hg(+).