Abstracts

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Paternal Occupational Exposure to 2, 3, 7, 8 – Tetrachlorodibenzo-p-dioxin Birthweight and Birth Defects

C.C. Lawson, T.M. Schnorr, E.A. Whelan, J.A. Deddens, D.A. Dankovic, L.A. Piacitelli, M.H. Sweeney, L.B. Connally

National Institute for Occupational Safety and Health

Background: Agent Orange, a phenoxy herbicide, is a mixture of the herbicides 2,4-D [(2,4-dichlorophenoxy) acetic acid] and 2,4,5-T [(2,4,5-trichlorophenoxy)acetic acid]. Agent Orange was widely used as a defoliant in Vietnam and was contaminated with 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). Most of the general population is exposed to low levels of dioxins, primarily through dietary intake of animal fats.

Methods: Studies of occupational exposure to TCDD present the opportunity to assess the health effects of levels much higher than would be expected in the general population. We studied pregnancy outcomes among wives of male chemical workers who were highly exposed to chemicals contaminated with TCDD and among non-exposed neighborhood referents. Detailed information on reproductive, medical, lifestyle, and occupational factors was collected from current and former wives/partners via telephone interview. We estimated serum TCDD level at the time of conception using a pharmacokinetic model. The mean worker TCDD concentration was 254 parts per trillion (ppt), with a range of 3-16, 340 ppt. The mean referent concentration of 6 ppt was assigned to referent births and worker births conceived before exposure (pre-exposure births). A repeated measures analysis was used to assess the effect of TCDD on the birthweight of live, singleton, term births (> 37 weeks gestation).

Results: The mean birthweight was similar among referent births (n=604), pre-exposure worker births (n=259), and offspring born during or after exposure (n=292): 7.5 pounds (lbs), 7.4 lbs, and 7.6 lbs, respectively. There was no effect of continuous or categorical TCDD on birthweight when adjusted for infant sex, mother's education, parity, prenatal smoking, and gestational age. An analysis to estimate the potential direct exposure of wives during the time period of workers'
exposure yielded a non-significant increase of 0.29 lbs in the highest exposure group (TCDD >254 ppt) compared to referents (p=0.09), when adjusted for confounding variables. Mothers' reports of birth defects showed no evidence of an exposure relationship, though numbers were small.

Conclusion: These results do not support a causal relationship between paternal TCDD exposure and lowered birthweight. Because the estimated TCDD levels in this population were much higher than in other studies, the results provide evidence that paternal TCDD exposure does not increase the risk of low birthweight at levels above those observed in the general population. The study also offers reassurance to former workers and Vietnam veterans who may have been exposed to high levels of TCDD.

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