

Chronic Hepatic Porphyrin and Exposure to Substances Contaminated with 2,3,7,8-Tetrachlorodibenzo-p-dioxin

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Background: 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), produced as an undesirable contaminant in the manufacture of 2,4,5-T and its derivatives, is known to cause hepatic porphyria in rodents. Several reports have described chronic hepatic porphyria (CHP) among TCDD-exposed individuals; however, other investigators question the association between CHP and TCDD exposure.

Study Objective: To evaluate the association between occupational exposure to TCDD-contaminated substances and CHP.

Design: A cross-sectional medical study.

Participants: The exposed participants were employed at two chemical plants more than 15 years earlier in the manufacture of sodium trichlorophenol and its derivatives. The referent group consisted of age, race, neighborhood and gender-matched individuals with no occupational exposure to phenoxy herbicides. A total of 281 workers and 260 unexposed referents participated in the medical study.

Main Results: The workers had substantial exposure to substances contaminated with TCDD, as evidenced by a mean serum TCDD level, lipid adjusted, of 220 ppt compared to a mean of 7 ppt in the unexposed referent group. No difference was found between workers and referents in the prevalence of CHP, nor were there differences in the urinary uroporphyrins or coproporphyrins.

Conclusions: This study found no evidence of an elevated risk for CHP among a group of workers with high exposure to TCDD. Furthermore, this study found no association between TCDD exposure and elevation in the uroporphyrin level or elevation in the coproporphyrin level. A reevaluation of the literature indicates that there is insufficient evidence available to convincingly support an association in humans between CHP and TCDD exposure.

