conflicting results. Baxter found no abnormalities in the urinary excretion of coproporphyrin, uroporphyrin, and alpha levulonic acid (ALA) among workers engaged in the production of PCP at a plant in the United Kingdom. Pines et al found higher mean urinary excretion of coproporphyrin and ALA in workers employed in wood processing and furniture manufacture compared to controls[Pines]. Workers in this pilot study, however, were exposed not only to technical PCP but also to solvents used in wood finishing. Coproporphyrinuria has also been observed in workers with high serum concentrations of PCBs[Maroni].

Animal studies are consistent with the induction of porphyrinuria by exposure to contaminants present in technical PCP. Goldstein et al compared the porphyrinogenic effect of technical (contaminanted with CDDs and CDFs) vs pure PCP in female rats[Goldstein]. Rats fed 500 ppm of technical PCP for 8 months had significant elevations in urinary excretion of coproporphyrin, uroporphyrin, and ALA compared to controls, while pure PCP was not porphyrinogenic. Technical PCP, however, did not increase the activity of ALA synthetase in this study, although dioxins such as TCDD are believed to exert their porphyrinogenic effect through induction of ALA synthetase as well as inhibition of UROD[Doss] .

In our study urinary porphyrin excretion was highest among the subgroups of workers with chloracne who had worked with both PCP and PCBs. Results of recent animal studies suggest a possible synergistic interaction between certain PCB congeners and TCDD in producing hepatic porphyrin accumulation. Van Birgelin et al observed an 800 fold increase in hepatic porphyrin accumulation in Sprague-Dawley rats that were co-administered PCB 153 and TCDD in a 13-week feeding study[Van Birgelin]. Extreme individual variation in hepatic porphyrin accumulation was observed in these animals. Van Birgelin et al hypothesize that the synergistic interaction observed in their animal studies may be due to the combined effects of Ah receptor mediated induction of CYP1A2, which may play a role in the oxidation of uroporphyrinogen III to URO III; together with possible induction of ALA synthetase. These animal data are consistent with our observation