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SUMMARY OF TOXICITY, DISTRIBUTION, AND EXCRETION OF DIOXIN

(Information from A. L. Young et al, The Toxicology, Environmental Fate, and Human Risk of Herbicide Orange and its Associated Dioxin, Report OEHL TR-78-92, USAF)

Of the compounds in the herbicide or defoliant agent Orange, the most toxic is dioxin, really a group of closely related chlorinated biphenyl compounds whose principal member is TCDD. Dioxin was a contaminant of varying concentration, usually in a few (0.02-15) parts per million, of Agent Orange, although some lots may have contained somewhat greater concentrations.

TCDD has the same range of acute toxicity as strychnine. It kills animals after weeks of ingesting weekly oral doses of one or more micrograms per kilogram of body weight, the exact amount depending on the species. Apparently delayed toxic effects have not been studied in animals.

Birth defects and fetal death occurred in animals during dioxin feeding. Rodents chronically fed TCDD showed an increase in tumors, specifically liver and lung carcinomas.

Animals excrete dioxin primarily in the feces whether they ingest TCDD or have it injected. It is not metabolized and persists undegraded in the liver for 11 to 20 days in mice. Radioactive compound in guinea pigs, injected the day before, was located in fat (2.36%), adrenals (1.36%), and liver (1.13%). No other organ accounted for as much as one per cent. In rats the half-life of TCDD is 12 to 24 days but it is excreted slowly and remains unmetabolized over an extended period.

The only clearly related symptom to dioxin exposure in man is chloracne, a condition which may persist for some months after exposure.

There has been no good evidence of the delayed appearance of toxic effects in humans and no one has demonstrated prolonged retention or persistent excretion of dioxin in persons exposed years earlier.

Despite this, lay claims have been made that toxic effects are still being manifest as a result of possible exposure to Agent Orange prior to 1970. Conjecture that dioxin may persist in human fat for years after exposure has further excited anxiety. It has become important, therefore, to determine whether the compounds can be detected in body fat after exposure to herbicides and whether there are more in the fat of Viet Nam veterans who had contact with Agent Orange than in American contemporaries who did not. To do so, fat will be assayed for dioxin by combined gas-liquid chromatography and mass spectrophotometry, a method that can detect the compounds in parts per trillion.

If a small number of Viet Nam veterans with exposure and matching controls without give no evidence of dioxin or have an equal content, no further assays will be needed. If the exposed veterans' fat contains a larger amount it may prove possible to use the determinations to demonstrate prior contact with Agent Orange.

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