A REVIEW OF PUBLIC HEALTH LESSONS LEARNED IN TWO DECADES OF DIOXIN RESEARCH

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INTRODUCTION

My colleagues and I have learned many lessons since my introduction to dioxin research, which began February, 1981 with the PCB transformer fire of the Binghamton (New York) State Office Building. These encompass both science and policy aspects of dioxin research.

MATERIALS AND METHODS

This consists of a historical review of major findings beginning with the Binghamton incident. Dioxin analyses were performed in a congener-specific manner by high-resolution gas-chromatography mass-spectrometry[1].

RESULTS AND DISCUSSION

The Binghamton State Office Building fire showed that the original Buser-Rappe laboratory work with PCBs and chlorinated benzenes could occur in the real world. In the presence of heat and oxygen, dioxins and dibenzofurans would be generated, primarily from chlorinated benzenes and PCBs, respectively.

The original building cost $19,000,000 to construct, and had a life span of approximately a single decade prior to contamination from the fire. A 13-year cleanup, which met the New York State Department of Health Cleanup Criteria, was estimated to cost $52,000,000. This was the first PCB fire generating dioxins and dibenzofurans to be recognized and led EPA to ban PCB transformers from public buildings.

This fire led to the first congener-specific dioxin and dibenzofuran determinations in blood and adipose tissue, or fat, of the US general population and of US workers. Congener-specific analysis, as suggested by Rappe, indicated elevation of the same congeners in human samples as found in the contaminated Binghamton soot [2,3,4]. This analysis showed that every person tested in the USA had surprisingly high measured levels of PCDD/Fs, about 1,400 ppt lipid, meaning that there were no non-exposed persons with respect to dioxins. Because of this finding, changes in basic epidemiologic exposure criteria were necessry, and were later employed by Fingerhut, others, and in Dutch children studies [5,6,7].

We have found some US Vietnam veterans from Massachusetts and Michigan with elevated TCDD levels from Agent Orange. In comparison to these sample populations, a markedly higher percentage of Vietnamese living in the south of Vietnam had elevated levels of TCDD from Agent Orange-as high as 1,850 ppt TCDD in one nursing woman's milk [8,9,10]. In addition, we are

currently finding elevated TCDD levels in children born after the spraying ended in 1970, and levels as high as 271 ppt in some southern Vietnamese (1999)[11]. These new measurements are attributed to recent contamination, presumably from Agent Orange spills and current contamination of fish.

It has been established that the general population is contaminated with dioxins worldwide, with lower levels (i.e. TEQs usually under 10 ppt lipid adjusted) found in less industrial countries such as Thailand, China, north of Vietnam, Russian Siberia, Cambodia and others. In industrial countries, levels of dioxins in human tissues are much higher, with TEQs about 20-60 ppt lipid in blood samples[12,13].

In Russia, we have found men and women chemical manufacturing workers with elevated TCDD and PnCDD levels decades after cessation of their workplace exposure. Also, some of the children of these workers had elevated TCDD levels, either from nursing or household exposure to contaminated work clothing [14,15].

Chinese agricultural workers and their children were contaminated with higher chlorinated dioxins and dibenzofurans from the use of PCP, which is used to kill snails, known to spread schistosomiasis[16].

Levels of dioxins in food, human milk and blood seem to be decreasing in Europe, but we are not certain of whether US population levels are following the same trend. In a small study of strict vegetarians, vegans, we did find that their blood dioxin levels were considerably low compared to the general US populations[17].

The US food supply provides 1-6 ppt TEQ/kg BW per day for adults, but 35-65 ppt for nursing infants in the first year of life[18]. Transplacental transfer of dioxins and dibenzofurans has been documented by measurement of human aborted fetuses as young as 12 weeks gestational age[19].

Cooking can increase or decrease dioxin content in food. Thus, estimates of food dioxin levels based on raw food samples may be incorrect[20,21].

Presently, we do not know current levels of dioxin in humans from most countries or continents of the world, specifically from less developed countries. Without such data, it will be impossible to determine if contamination of people is increasing or decreasing worldwide. This ultimately will be the best test of the efficacy of governmental regulations and enforcement.

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