Dioxin poisonings in Germany, the USA, and Russia

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Introduction

Four case reports of inadvertent dioxin poisonings are described. The first involves German chemical workers whose adipose tissue was found to have elevated levels of TCDD over 3 decades following exposure. The second involves a chemist in the USA who synthesized 2,3,7,8-TCDD and also 2,3,7,8-tetrabromodibenzo-p-dioxin (TBDD). This person was briefly hospitalized after exposure to TBDD with chloracne, a high dose and rare pathology, muscle aches and pains, fatigue, and other signs and symptoms of ill health. The third consists of Russian workers and their children who had elevated TCDD and PnCDD decades after work and potentially also home exposure. The fourth involves a pentachlorphenol (PCP) poisoning which led to the death of a US (Mississippi) worker. Differing patterns of tissue levels were found in each incident. Except the acute PCP poisoning, the elevated congeners were found decades following exposure, a characteristic finding of dioxins which are among the persistent organic pollutants (POPs) which have long half lives of elimination.

First case report, Germany, BASF workers

The first case report is from German workers who manufactured chlorophenols and phenoxy herbicides. Their adipose tissues were sampled in 1988, 32 years after known special factory exposure and analyzed for TCDD.[1] All 6 workers studied had chloracne from high exposure as well as genetic sensitivity to dioxins. The average concentration was 49 ppt TCDD on a lipid basis, about 10 times higher than the then mean level of TCDD in humans from industrial countries, and the range was 11-141 ppt. Table 1 shows their dioxin levels in detail.

These six patients were involved in direct contact with dioxins during a dioxin cleanup in 1953. They all developed persistent chloracne. Other medical signs and symptoms were noted after exposure including fatigue, headache, memory impairment, severe pain in the abdomen and extremities, liver pathology, elevated serum lipids, conjunctivitis, insomnia and gastrointestinal system pathology. These symptoms were documented in the patients' medical records. The medical problems listed above can be caused by dioxins although other causes are also possible.

Table 1. 1985 findings in German human fat tissue from a 1953 dioxin incident

Patient	Age	Gender	2,3,7,8-TCDD whole pg/g	PPT lipid
1	52	M	11	16
2	53	M	21	30
3	57	M	28	41
4	60	M	113	141
5	62	M	7.8	11
6	NA	M	41	57

In light of the history of chloracne following exposure plus the other reported or observed medical problems, it seemed to us reasonable to conclude that there were some probable causal linkages between the ingestion of the 2,3,7,8-TCDD, which was documented at the work site, and at least some of the subsequent illnesses, including the severe characteristic skin lesion, chloracne.

The patient with the 141 ppt TCDD adipose tissue level initially had difficulty documenting dioxin exposure for workers' compensation purposes but was assisted by the findings of 141 ppt which extrapolated back to

approximately 1,130 ppt at time of initial exposure some 32 years earlier.

Second case report, a poisoned American chemist

The second case report involves a US chemist who synthesized both 2,3,7,8-TBDD and about 16 g pure 2,3,7,8-TCDD[2]. He became sick both times but was hospitalized only for the TBDD instance in 1956. Choracne, headaches, back and severe leg pain on exertion were the most prominent symptoms. His measured TBDD in 1991 was 625 ppt, and his TCDD was 18 ppt 35 years after initial exposure. TBDD is usually not detected in the general population and general population TCDD at that time was approximately 5 ppt. With a 7-11 year half-life for TCDD and presumably a similar half life for TBDD, the original levels would have been approximately 8,000 ppt for TBDD and 200 ppt for TCDD. With this chemist only TCDD and TBDD were elevated, none of the other PCDD or PCDF congeners. We estimated a body burden of approximately 115-1300 micrograms or 2.0-21 micrograms per kg of body weight for TBDD and between 3.2-37 micrograms or 0.05-0.61 microgram/kg body weight for TCDD.

This case documents a workplace hazard outside of the more usual industrial settings. A similar poisoning of British chemists has also been described [3].

Third case report, Russian workers

Ufa, Russia, at the western base of the Ural Mountains, was the site of one very large chemical manufacturing plant, the Ufa, Khimprom Company which shipped chemicals worldwide. One small brick structure housed manufacturing equipment for phenoxy herbicides including 2,4-D, MCPA, 2,4,5-TrCP, and 2,4,5-T, the latter two contaminated with TCDD and PnCDD.

In the Ufa incident there was elevated 2,3,7,8-TCDD and to a lesser extent 1,2,3,7,8-PnCDD in the blood of workers working with the phenoxy herbicides in the 1960s and also their children.[4] To a lesser extent, others at the chemical plant also had increased exposure to dioxins, documented by elevated blood levels. Workers' (N=34) blood TCDD had a mean of 231, a median of 167, and a range of 35 to 1680 on a lipid basis -35 years after initial exposure. It is believed likely that the workers' children had elevated levels by contact with contaminated clothing, and that the other workers had elevated levels from dioxin movement through the air with respiratory or gastrointestinal intake. Even the general population of Ufa had elevated TCDD levels with a value of 19 ppt. In 1992 the TCDD level in the Russian State of Bashkortostan in which Ufa is situated, was 4.8 ppt (median of 16 pools) with an N of 264 donors.

The 2,4-D workers (N=6) had 48-83 ppt of TCDD, presumably from contamination with 2,4,5-T made in the same facility. The TrCP wokers (N=3) had TCDD of 302-1,240 ppt. The MPCA workers (N=2) had 57 and 121 ppt TCDD. The 8 children of 2,4,5-T workers had 11-80 ppt of TCDD. Factory administrative workers had 16-65 ppt TCDD and two Ufa city pools were 12 and 62 ppt.

There was overlap of TCDD blood levels in persons with and without chloracne. Persons with levels approaching 400 ppt blood TCDD from exposure 35 years previously when calculated levels would have been approximately 5,000 ppt were sometimes found to never have had chloracne. Persons with levels between 200 – 400 ppt in 1992 were found to have reported having chloracne only once. Levels below 50 ppt were found in persons who reported acne sometimes whereas persons with levels above 400 ppt were found to have all reported chloracne.

Fourth case study: A US worker fatally poisoned with pentachlorophenol (PCP)

The fourth case report is that of a worker who was fatally poisoned with PCP while handling large cakes of this chemical. This acute incident provided autopsy tissue which was analyzed for dioxins and dibenzofurans. Very elevated levels of higher chlorinated dioxins and dibenzofurans were found in a typical PCP pattern (Table 2) [5].

Discussion

These cases document differing accidental contaminations with dioxins. The congener patterns differ depending on the source of the dioxins or dibenzofurans and is characteristic of that contamination. Pure TCDD contamination is rare.

Table 2 Fat tissue analysis of PCP poisoned person

	Level
2.3.7.8-Tetra-CDD	33
1.2.3.7.8-Penta-CDD	70
1.2.3.4.7.8-Hexa-CDD 1.2.3.6.7.8-Hexa-CDD	700
1.2.3.7.8.9-Hexa-CDD	350
1.2.3.4.6.7.8-Hepta-CDD	15300
OCDD	129000
2.3.7.8-Tetra-CDF	nd(4.3)
1.2.3.7.8-Penta-CDF	na
2.3.4.7.8-Penta-CDF	50
1.2.3.4.7.8-Hexa-CDF 1.2.3.6.7.8-Hexa-CDF	174
1.2.3.7.8.9-Hexa-CDF	na
2.3.4.6.7.8-Hexa-CDF	37
1.2.3.4.6.7.8-Hepta-CDF	6020
1.2.3.4.7.8.9-Hepta-CDF	787
OCDF	15300
TEQ (pg/g or ppt, lipid)	
2,3,7,8-TCDD	33
PCDD	374
PCDF	202
Total TEQ	609

TCDD and TBDD poisoning was seen in the chemist who synthesized these chemicals. TCDD elevation alone has also been reported from the Vienna poisoning of five persons and from that seen from the Ukraine which are believed to be deliberate poisonings. Even Vietnamese with elevated 2,3,7,8-TCDD from the 2,4,5-T phenoxy herbicide in Agent Orange have elevation not only of TCDD but also, in some cases, of 1,2,3,7,8-PnCDD, similar to that seen in the Russian workers[6, 7]. The pattern of dioxins and dibenzofurans can be used to point towards the source of the contamination or poisoning. In the Binghamton State Office Building fire with PCBs and to a lesser extent polychlorinated benzenes used as transformer fluid, the pattern of mostly elevated blood (and environmental) PCDFs and PCDDs to a lesser extent are consistent with the pyrolysis of the transformer fluid[8]. To the best of our knowledge, there have only been two known incidents and six people involved in deliberate TCDD poisonings, one from the Ukraine [9, 10]and five from Vienna[11-13].

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