Dioxin and Dibenzofuran Levels in Blood and Adipose Tissue Following Occupational Exposures to Pentachlorophenol

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Abstract

Pentachlorophenol (PCP) wood preservative is characteristically contaminated with higher chlorinated congeners of dioxins (PCDDs) and dibenzofurans (PCDFs). We found elevated PCDD/PCDF blood levels in a worker following chronic exposure to PCP. We compare these levels to levels found in adipose tissue of a worker who died following heavy, acute PCP exposure. An analysis of commercial PCP is also presented for comparison.

Introduction

One of us (JJR) previously reported dioxin levels of adipose tissues from an adult male acutely poisoned with pentachlorophenol (PCP) who died shortly after exposure[1,2]. In this paper we present the analysis of a recently obtained commercial PCP sample and also the blood of a 56 year old male worker from Oregon who was chronically exposed to PCP used as a wood preservative. The Oregon worker was exposed to PCP between 1978 and 1987 while applying PCP to lumber; the blood sample was obtained in 1996. In addition to the time difference between exposure and sampling for the two workers the Oregon worker suffered from pancreatic cancer and died shortly after the blood sample was obtained.

Methods

For the chronically exposed worker, 100 mL of whole blood was collected shortly before the patient's death. It was frozen and shipped on dry ice to Health Canada for analysis. Analytic methods have been presented previously[3]. The pentachlorophenol sample was stored in a barn in New England during the 1990s. An aliquot was sent to ERGO Forschungsgesellschaft for analysis. Analytic methods have been previously presented[4,5]. A pooled United States (U.S.) blood sample (N=100) for comparison was collected in 1996, frozen and shipped to ERGO, for analysis. Analytical methods have been described[6].

Results

Figure 1 shows the polychlorinated dibenzo-p-dioxin (PCDD) and polychlorinated dibenzofuran (PCDF) congener pattern and measured levels for the PCP analysis. Although higher chlorinated congeners predominate, low levels of 2,3,7,8-TCDD and 2,3,7,8-TCDF were present. Figure 2 presents the pattern and measured levels of PCDDs/PCDFs in adipose tissue of the acutely poisoned worker. Higher chlorinated

dioxin and dibenzofuran congeners predominate. Figures 3 and 4 show patterns and levels of PCDDs and PCDFs, respectively, in the blood of the worker chronically exposed to PCP. These are compared with American general population levels in blood[7]. In general higher chlorinated congeners predominate, but OCDF was found in lower levels than 1,2,3,4,6,7,8-HpCDF.

Discussion and Conclusions

Four analyses are presented: one of a sample of PCP itself, one pooled blood specimen from the U.S. general population, and two of persons exposed to PCP, one acutely just prior to tissue (adipose) sampling and one in a more chronic fashion years before tissue (blood) sampling. In all instances the higher chlorinated dioxins and dibenzofurans predominate. The finding of somewhat lower OCDF levels in the chronically exposed worker may have any of a number of causes. The time after exposure was long and OCDF may have been eliminated. Cachexia frequently accompanying cancer may have altered metabolism in a fashion which produced the lower OCDF level. Partitioning differences between blood and adipose tissue might contribute to the lower OCDF levels. The increase in PCDD/F levels might be explained by aging rather than PCP exposure. Loss of weight from cancer might also be responsible for elevation of some congeners. The PCP to which the worker was exposed might have been lower in OCDF than the sample which we analyzed. Despite the cancer, the levels and patterns of PCDD/Fs in the Oregon worker are consistent with expectations following exposure to PCP. The larger issue of changes in tissue dioxin and dibenzofuran levels with wasting diseases including but not limited to cancer remains to be more fully explored.

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References

1. Ryan, J.J.; Lizotte, R.; Lewis, D.A. Human tissue levels of PCDDs and PCDFs from a fatal pentachlorophenol poisoning. *Chemosphere* **1987**, 16(8/9), 1989-1996.

2. Gray, R.E.; Gilliland, R.D.; Smith, E.E.; Lockard, V.G.; Hume, A.S. Pentachlorophenol Intoxication: Report of a Fatal Case, with Comments on the Clinical Course and Pathologic Anatomy. *Arch Environ Health* **1985**, 40(3), 161-164.

3. Ryan, J.J.; Lau, B.P.-Y.; Boyle, M.J. *Biological Mass Spectrometry*, eds. Matsuo, T.; Capriolo, R.M.; Gross, M.L.; Seyama, Y. **1994**, Chapter 3.16, 583-602.

4. Schecter, A.; Päpke, O.; Lis, A.; Ball, M. Chlorinated dioxin and dibenzofuran content in 2,4-D amine salt from Ufa, Russia. *Organohalogen Compounds* **1993**, 11, 325-328.

5. Päpke, O.; Ball, M.; Lis, Z.A.; Scheunert, K. PCDD and PCDF in indoor air of kindergartens in northern W. Germany. *Chemosphere* **1989**, 18(1-6), 617-626.

6. Päpke, O.; Ball, M.; Lis, Z.A.; Scheunert, K. PCDD/PCDF in whole blood samples of unexposed persons. *Chemosphere* **1989**, 19, 941-948.

7. Schecter, A.J.; Päpke, O.; Fürst, P. Is there a decrease in general population dioxin body burden? A review of German and American data. *Organohalogen Compounds* **1996**, 30, 57-60.

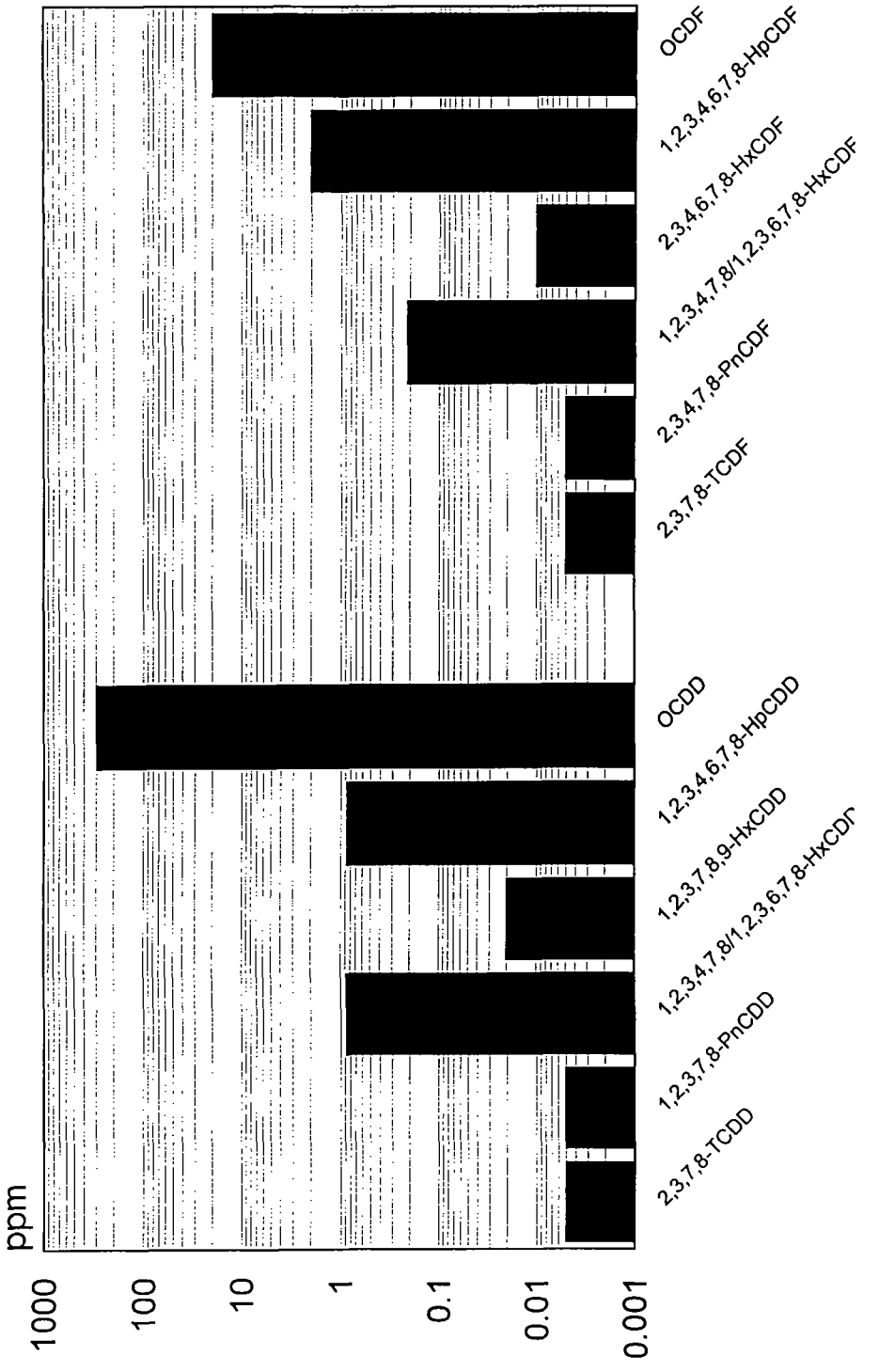

Figure 1. Measured Levels of PCDDs/PCDFs in Pentachlorophenol (mg/kg, ppm)

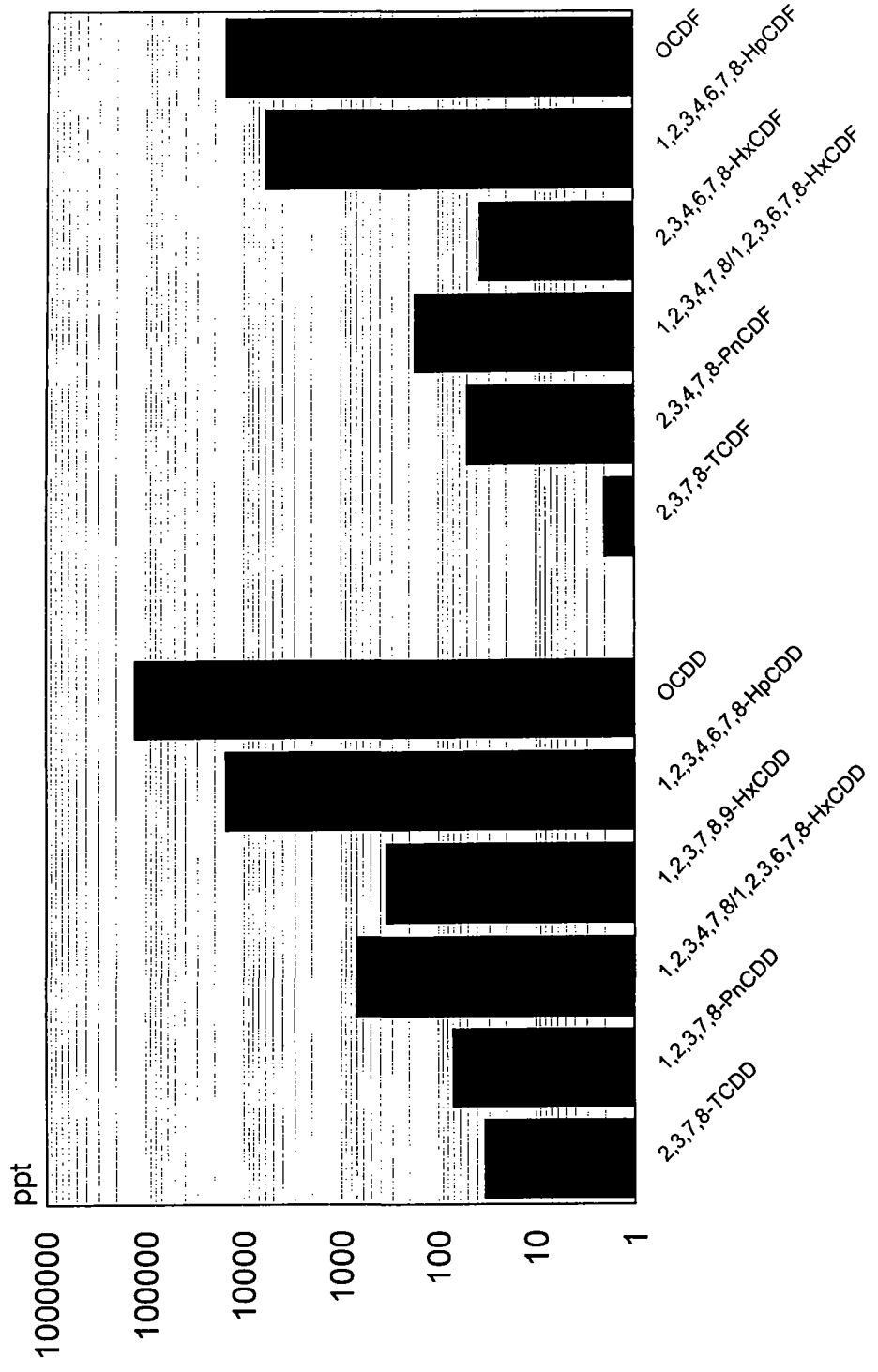

Figure 2. Measured Levels of PCDDs and PCDFs (pg/g) from the Fat Tissue of a Victim of PCP Poisoning

Figure 3. Measured Levels of PCDDs in Whole Blood from a Worker and a Binghamton Pooled Sample (N=100)

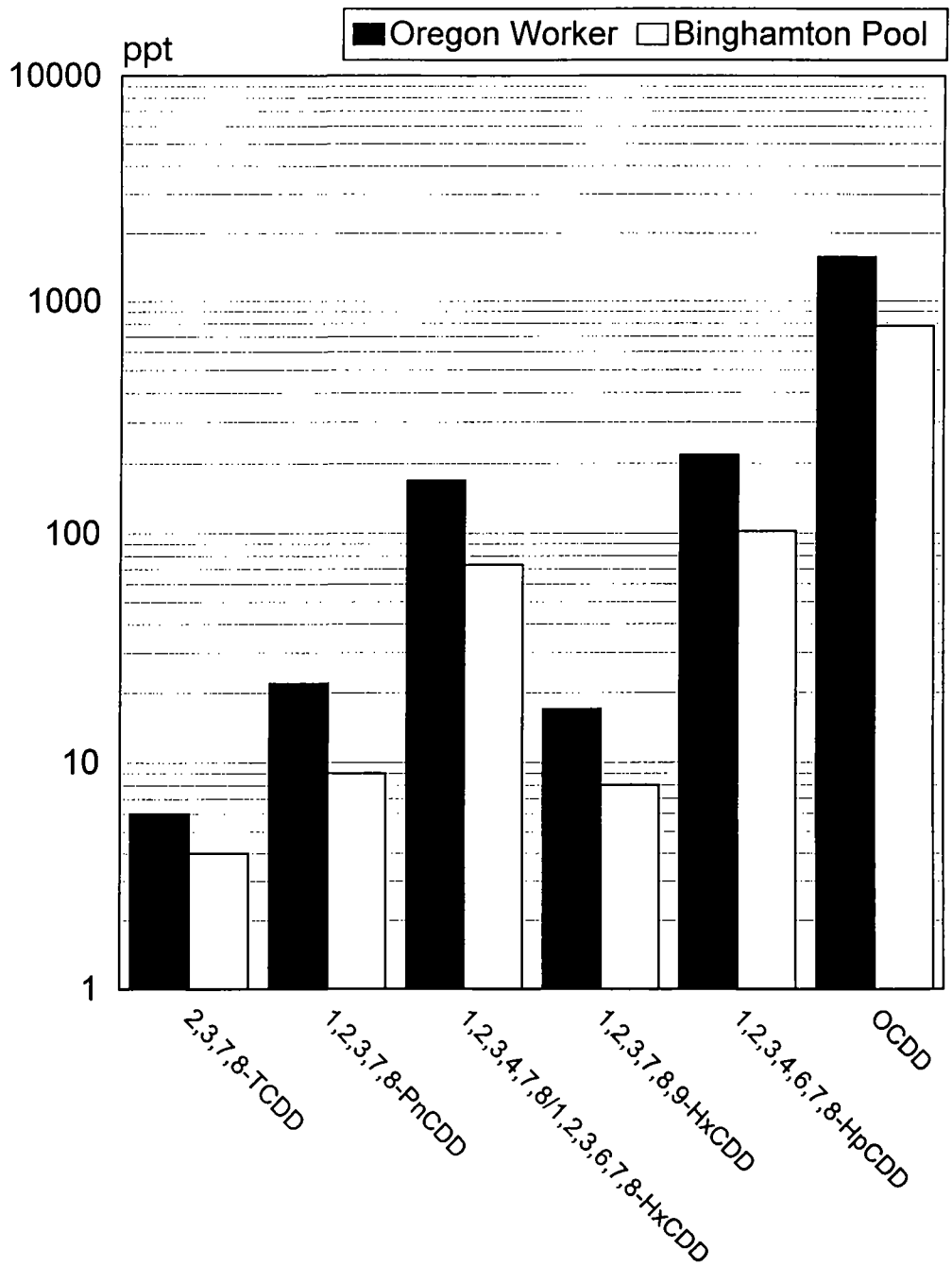

Figure 4. Measured Levels of PCDFs in Whole Blood from a Worker and a Binghamton Pooled Sample (N=100)