# DIOXIN ARCHAEOLOGY - REVEALIG OF POTENTIAL HUMAN EXPOSURES TO POLYCHOLORINATED DIOXINS AND FURANS IN THE EARLY 40's

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## Introduction

What is the explanation for the occurrence of polychlorinated dibenzo-p-dioxins and dibenzo-furans found in human blood plasma from the 40's? We have recently analysed an archived sample of freeze dried pooled normal human plasma prepared by the Lederle Laboratories, New York, in 1944. This unique sample contained 55 ppt of toxic dioxin equivalents, expressed in (i)-TEQs, on lipid basis. This is significantly more than the background levels in human blood plasma, both in the U.S. and Europe, today.

The occurrence of polychlorinated dioxins and furans, PCDDs and PCDFs, in human tissues was first reported in the literature in samples collected in the early 1980[1]. Isomer specific analyses of the notorious 2,3,7,8-substituted dioxins and furans in human blood, adipose tissue and milk have been carried out since the mid 80's in several laboratories world wide. The WHO has played an important role in bringing consensus to the analytical performance and high reliability[2] of the analytical results, making comparisons possible, by co-ordinating interlaboratory calibrations[3] and field studies. The typical current 'human dioxin profile' as well as representative general background levels are therefore well documented today.

Population background levels of dioxins in human tissues show declining trends in several European countries since the monitoring started in the early 80's, and are now down at their lowest levels ever seen in Europe[4,5].

The documented decline - by up to 30-50% - in human body burden of dioxins is attributed to restrictions in the use of chemicals that are potential sources for dioxins, such as chlorophenols and PCB. Also the improvements or ban of industrial and other technical processes known to generate dioxins, such as uncontrolled combustion and chlorination, have had effects on the reduction of emissions.

The sum of the concentrations of the highly toxic 2,3,7,8-substituted PCDDs and PCDFs, weighted by their toxicity, is commonly used to express the dioxin levels as toxic equivalents (TEQs)[6]. In ten different studies, found in the literature in 1996 and 1997 reporting on levels of dioxins in Europe and the U.S., the mean levels in the general populations were in the range of 8.4-41 ppt. Expressed in pico grams of (i)-TEQs per gram of blood lipids.
The individual variations - for the total toxic equivalents as well as for the different congeners - in the general populations are mainly correlated with factors such as age, dietary habits and lactation.

**Materials and Methods**

The archived human plasma product we have analysed here was produced under U.S. Government Licence No 17, by Lederle Laboratory, NY, in 1944 for use in Europe at the end of WW II. The plasma was freeze-dried and had been stored under vacuum, which was confirmed when we opened the glass bottle. The freeze-dried pooled plasma - which originated from a lot infected by hematite B and therefore was never used - was equivalent to 600 cc of human plasma. After reconstitution of the plasma the sample was cleaned up and the planar fraction was analysed in duplicate (CV<5%) by SIR high resolution mass spectrometry (R=10.000) according to the validated WHO protocol in our laboratory. A laboratory blank sample and a reference background plasma sample from 1997 were analysed at the same time. The blank sample had no native signals over the DL (0.1 - 1 ppt).

**Results and Discussion**

Our unique sample reveals, with its (i)-TEQ content of 55 ppt, that human exposure to dioxin could have been significant in the 40's (Table 1). It also indicates, through its PCDD dominated profile, that the possible exposure was different than it is today. The pattern of the PCDDs in the plasma sample from 1944 bares great resemblance - in the high levels of the two the two HpCDDs and OCDD - with an other archived sample, a municipal sewage sludge fertiliser from 1933[7]. It was suggested that various chlorination processes, rather than commercial pentachlorphenol, was the source of PCDDs in that sample.
Generally it has been assumed, even if it has never been scientifically confirmed, that human exposure to dioxins is a post WW II problem. Temporal trend studies, both in the U.S. and Europe, of deposition levels in sediments show that the concentrations peak in the early 70's and that they are now down at the 1940-level. Further, there is also a change in the homologue profile at 1945, from a PCDD dominated to a PCDF dominated one. A PCDD dominated profile is explained by 'chlorine' sources and a PCDF profile by 'thermal' sources[8].
Depositions of dioxins initially started to increase already in the 20's. This parallels the growing production and use of chlorine[9]. We may have underestimated certain human exposures to chlorinated compounds due to the unrestricted use of chlorine and chlorinated products in the beginning of the century.

Table 1 **Human plasma dioxin levels[a] in 1944 and 1997**

| **PCDD** | **1944[b]** | **1997[c]** |
|---|---|---|
| 2378-TCDD | *3.6* | *3.2* |
| 12378-PeCDD | 16 | 17 |
| 123478-HxCDD | 4.7 | 8.3 |
| 123678-HxCDD | 99 | 45 |
| 123789-HxCDD | 32 | 4.6 |
| 1234679-HpCDD | 410[d] | <4 |
| 1234678-HpCDD | 310 | 15 |
| OCDD | 7.500 | 230 |

The findings presented here are of general environmental interest as well as of great importance from various specific scientific aspects. They may add another piece to the dioxin puzzle. It should be bared in mind that isomer specific determination and evaluation of PCDDs and PCDFs in environmental samples reveals much more of the origin of the exposure than using toxic equivalents as the measure. Therefore reports on human exposure should present the data in a congener specific way.

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