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RESEARCH ARTICLE

Estimation of Dioxins in Workers Collecting and Burning of Waste

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Abstract

The study included measuring the concentration 17 congeners from PCDD/Fs (7 PCDDs and 10 PCDFs) in the blood lipids of workers collecting and burning of waste for 56 candidates, the range of age was 23–42 years, classified into two categories depending on the period of exposure. The first category (category 2) includes 27 persons, the exposure period was <10 years, the mean age was 30.3±3.5 years, the mean PCDDs, PCDFs and PCDD/Fs were 8.61, 5.34 and 13.95 (pg WHO–TEQ/g lipid), respectively. The second category (category3) includes 29 persons, the exposure period was >10 years. The mean age was 35.6±2.8 years, the mean PCDDs, PCDFs and PCDD/Fs were 9.89, 5.65 and 15.54 (pg WHO–TEQ/g lipid), respectively. The data were compared with a control group (category1) (general (non-exposed) populations) including 50 candidates; the mean age was 32.2± 2.6 years, the mean PCDDs, PCDFs and PCDD/Fs were 6.87, 3.38 and 10.25 (pg WHO–TEQ/g lipid), respectively. A probability of 0.05.

Keywords: *PCDD/Fs, Burning of waste, Polychlorinated dibenzodioxins, Polychlorinated dibenzofurans.*

Introduction

A few municipalities of the Salah al-Din governorate were selected in Iraq. In places where workers collect and then burn waste, workers in waste collection and burning are exposed to fumes from solid waste, which they burn. Fumes are widely concentrated in areas where waste is burned. Makes the workers in this subject more susceptible to inhaling fumes, dioxin is the common name for a chemical compounds known as polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) [1], each numbered position can be substituted one or the other by a hydrogen or chlorine atoms, resulting in 75 different congeners in the PCDDs and 135 in the PCDFs whose structures are shown in Fig.1. (A, B) [2]. Have been included seventeen 2,3,7,8-substituted PCDD/Fs in the internationally agreed-upon Toxic Equivalency Factor (TEF), 2,3,7,8-tetrachlorodibenzo-p-dioxin TCDD that estimates the overall dioxin-like toxicity of compounds [3,4]. Physical and chemical properties of each congener vary according to the degree and position of chlorine substitution [5], is a common pollutant in industrialized areas [6]. With the economic development, more industrial pollutants are released. Among those PCDD/Fs are well-known for their health effects to the human body [7].

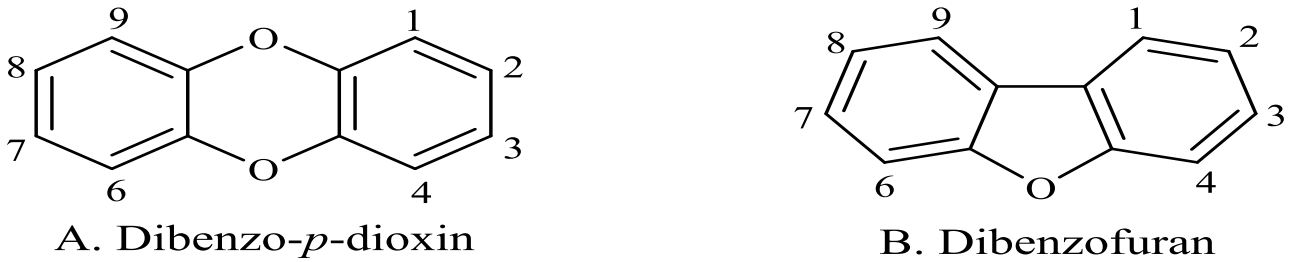

Fig.1: Chemical activators (A) PCDDs and (B) PCDFs [2]

The most dangerous emissions can be caused by burning plastics containing organochlor-based substances like Poly vinyl chloride (PVC). When those plastics are burned, quantities of dioxins, a group of highly toxic chemicals are emitted [8]. PCDD/Fs are highly toxic compounds that do not readily undergo biodegradation and its ubiquitous contaminants with long persistence (decades) in the environment and animal organisms [9].

Dioxin from Ultrafine particles or nanoparticles, its size <0.1 μm, It is easily absorbed from the lungs when it inhales its fumes, Resulting in increased bioavailability [5], Where they accumulate in the adipose fraction of organs and tissues, being lipophilic [10]. In human whole blood, about 80% of PCDD/Fs are associated with the lipoprotein fraction, 15% associated with protein (primarily human serum albumin), and 5% associated with cellular components. PCDD/Fs a lipophilic compound with a half-life depends on human body tissue and congeners types [11]. The metabolic pathways concerned in dioxin excretion are unclear [12].

Health Effects

In assessing the risk of TCDD, the United States Environmental Protection Agency (USEPA) came up with a virtual safe dose of 6fg/kg body weight per day. The reactions of the various member states of the European Union to these risk evaluations have put an emission limit of 0.1 ng/m3 toxic equivalency (TEQ), and tolerable daily intake of 1–4 pg - TEQ/ day/kg [5]. The exposure damage a wide range of tissue and species-specific responses-such as hepatotoxicity, wasting syndrome, , immunotoxicity teratogenicity, endocrine disruption, reproductive disorders [13,14,15]. It also causes renal toxicity and vascular dysfunction, soft tissue, skin lesions (chloracne), lymphomas, stomach cancer, elevated blood lipids, biochemical liver-test abnormalities, fatal injury, neurotoxicity and immune system. Additionally, genetic, carcinogenic, reproductive, and developmental effects have been showed in many animal studies, although species vary dramatically insensitivity to the PCDD/Fs congeners [5, 16, 17].

Mechanism of Action

The Aryl Hydrocarbon Receptor (AhR) has been widely characterized for the essential role it plays in mediating the toxic responses caused by PCDD/Fs. May also it causes inflammatory responses of renal tubular cells, and inflammation plays role in the pathophysiology of Chronic Kidney Disease [18]. Clearly, many of the effects are mediated through an interaction with the AhR. Dioxins induce a broad spectrum of biological responses, including induction of gene expression for cytochrome CYP1A1 and p450 [19]. AhR as a ligand dependent transcription factor that binds to dioxins structurally, When it binding with a ligands, translocates the cytoplasmic AhR to the nucleus, formed heterodimerizes with aryl hydrocarbon receptor nuclear translocator (AhRNT), and mediates numerous biological and toxicological effects by inducing the transcription of various AhR-responsive genes. As well as induction of Reactive Oxygen Species (ROS) formation oxidative DNA damage, and disruption of normal hormone signaling pathways, reproductive and developmental defects, and AhR ligation controls oxidation/antioxidation [19,22].The variation in toxicity amount the dioxins and the effect at the AhR is about 10,000 fold, with TCDD is the most potent, Fig.2. Depicts a schematic model of the dioxin action in the cell [19].

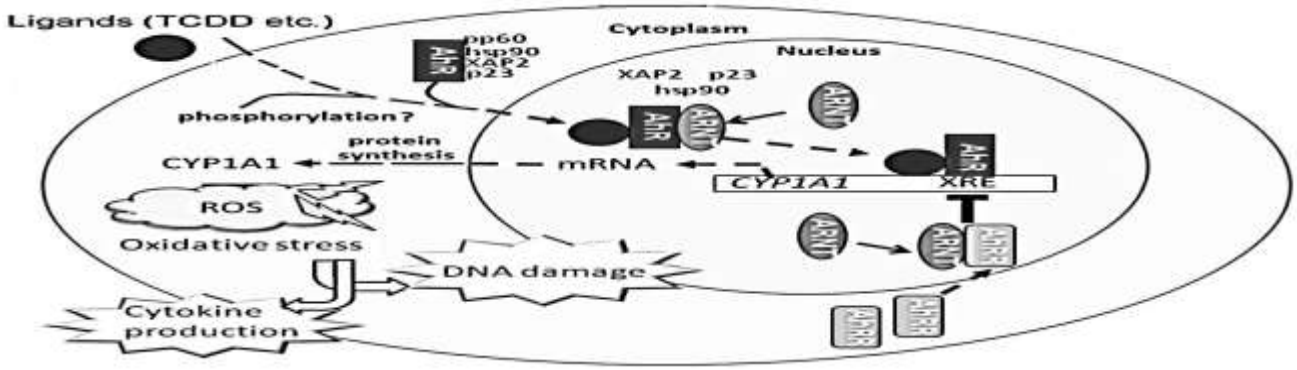

Fig.2: Simple mechanistic model for TCDD toxicity using the AhR pathway. AhR exist in the cytoplasm as a protein complex with hsp90, XAP2, and p23, ARNT: Ah receptor nuclear translocator. AhRR: Ah receptor repressor. CYP1A1, P450 and 1A1 cytochromes.XRE: xenobiotic-responsive element. ROS: reactive oxygen species [19, 22, 23]

Methods

Study Subjects

We had selected 56 candidates from a total 62 participant (We excluded 6 persons because for non-compliance with the questionnaire requirements), we collected the blood samples from workers collecting and burning of waste. All candidates chosen had been divided into two categories depending on the period of exposure in addition to the control group (category 1). The range of age was 23-42 years. The first category (category 2) includes 27 samples, the mean age was 30.3±3.1 years (range, 23-36 years), and the exposure period was <10 years. The second category (category 3) includes 29 samples, the mean age was 35.6±2.8 years (range, 32–41 years), and the exposure period was >10 years. The control group included 50 candidates; the mean age was 32.2± 2.6 years.

Questionnaires

All candidates provided the questionnaire information and showed them Informed consent orally. Information obtained from the questionnaire included many characteristics (age, start working date, interruption or departure Date (any candidate must be in his profession for at least 4 years), the nature of the local area, alcohol intake, tobacco use, dietary patterns, diseases and drugs used. Ethical approval for the study was granted by the Regional Hospital Ethics Committee.

Preparation of Blood Samples

Drawn about 30(ml) of fasting venous blood were obtained from each individual at clinics or hospitals in the study area with anticoagulant and stored at -40(C°) until shipment to the Laboratory for PCDD\Fs analyzes.

Measurements of PCDD/Fs

The GC/MS program, contain a variety of public libraries such as *NIST*, Wiley and original private libraries are also available can be used, previously calibrated on accordance with European guideline, uses *(NIST)* best efforts to deliver a high-quality copy of the database and to verify that the data contained therein have been selected on the basis of sound scientific judgment [24, 25]. Gravimetry was used to analyze lipid content for of each sample, which was extracted using a 2:1 acetone/hexane, the resultant organic layer was filtered and evaporated to dryness (the samples was evaporated under reduced pressure) to evaluate the lipid content in the samples. The cleaned extract was then analyzed by Standard Gas Chromatograph-Mass Spectrometer-Advanced Standard Model with Benefit of Economy, *Shimadzu (GCMS-QP2010 SE)* in the graduate research laboratory at the University of Samarra. Seventeen different congeners were measured from PCDD/Fs, included 7 PCDDs and 10 PCDFs. The results were compared with a control group for blood concentrations of PCDD/Fs congeners in general (non-exposed) populations in 50 Participants shown in Table 3 and Fig.4.

Data Analysis and Statistical Methods

PCDD/Fs concentration is expressed in picogram (pg WHO2005–TEQ DF/g) of lipid which is calculated by multiplying the levels of congeners PCDD/Fs individual in blood lipids by the TEF values shown in Table 2, as recommended by the World Health Organization (WHO) in 2005 [26]. When levels of individual congeners were below the detection of limit, a value equal to half the limit of detection was assumed for the estimation of the toxic equivalency PCDD/Fs (TEQ DF) concentration. The relationship between all categories was evaluated by analysis of variance (ANOVA), as well as measures of central tendency and measures of dispersion as a mean, median and standard deviation (SD) using SPSS Version 19.0. A probability of 0.05 or less was considered as significant.

Results and Discussion

Fifty-six candidates were selected, divided into two categories depending on the exposure periods. The range, mean and SD were calculated at a probability level <0.05 (2-tailed), for all variables. The first category (category 2) includes 27 persons, the exposure period was <10 years, the mean PCDDs, PCDFs and PCDD/Fs were 8.61, 5.34 and 13.95 (pg WHO–TEQ/g lipid), respectively. The second category (category 3) includes 29 persons, the exposure period was >10 years, the mean PCDDs, PCDFs and PCDD/Fs were 9.89, 5.65 and 15.54 (pg WHO-TEQ/g lipid), respectively, shown in Table 1. The data were compared with a control group (category1) (general (non-exposed) populations) including 50

candidates, the mean PCDDs, PCDFs and PCDD/Fs were 6.87, 3.38 and 10.25 (pg WHO–TEQ/g lipid), respectively shown in Table 3 and Fig.4.

Table 1: PCDD/Fs concentration TEQ (pg/g lipid) in workers collecting and burning of waste, and Control group

groups	age	TEQ concentration (pg/g lipid)					N [a]	P [b]
		mean	median	Min	Max	SD		
Category2 (<10 years)	30.3±3.5	13.95	13.86	9.82	16.92	1.4	27	0.05
Category3(> 10 years)	35.6±2.8	15.54	15.31	12.1	21.4	1.7	29	0.05
Category 1 (Control group)	32.2±2.6	10.25	10.14	8.45	13.11	0.8	50	0.05

A. Number of participants in the group.
B. Probability level.

Table 2: Individual congeners Levels of PCDD/Fs (WHO2005–TEQ DF/g of lipid) in workers collecting and burning of waste for two categories exposed (category 2 and category 3)

congeners			TEQ concentration (pg/g lipid) category 2 (<10 years)							TEQ concentration (pg/g lipid) category 3 (>10 years)				
S[a]	dioxins	(TEF)[b]	(%)[c]	mean	Median	min	max	SD	(%)[c]	mean	Median	min	max	SD
	2,3,7,8-TCDD													
	1,2,3,7,8-PeCDD													
	1,2,3,4,7,8-HxCDD													
	1,2,3,6,7,8-HxCDD													
1	1,2,3,7,8,9-HxCDD	1	67	1.30	1.54	0.80	1.28	0.4	79	1.72	1.16	1.13	1.62	0.6
2	1,2,3,4,6,7,8-HpCDD	1	100	4.20	4.03	3.08	4.64	0.7	100	4.52	4.20	3.64	5.87	0.8
3	OCDD	0.1	74	0.29	0.25	0.22	0.31	0.05	88	0.28	0.28	0.25	0.39	0.05
4	Total PCDDs TEQ	0.1	100	1.96	2.11	1.35	2.22	0.5	100	2.36	2.01	1.70	2.81	0.5
5		0.1	96	0.45	0.50	0.30	0.74	0.17	100	0.56	0.67	0.39	0.94	0.23
6		0.01	100	0.38	0.35	0.18	0.31	0.10	100	0.39	0.28	0.33	0.40	0.14
7	furans	0.0003	100	0.04	0.04	0.02	0.06	0.01	100	0.05	0.05	0.03	0.08	0.01
	2,3,7,8-TCDF	-	-	8.61	8.82	5.95	9.56	1.93	-	9.89	8.66	7.47	12.10	2.33
8	1,2,3,7,8-PeCDF	0.1	41	0.11	0.15	0.08	0.20	0.06	50	0.17	0.18	0.09	0.26	0.08
9	2,3,4,7,8-PeCDF	0.03	30	0.05	0.06	0.03	0.05	0.01	46	0.07	0.04	0.04	0.06	0.01
10		0.3	100	3.70	3.35	2.78	5.31	0.8	100	3.76	4.81	3.21	6.72	0.8
11	1,2,3,4,7,8-HxCDF	0.1	10	0.60	0.64	0.41	0.75	0.14	100	0.71	0.68	0.52	0.94	0.16
12	1,2,3,6,7,8-HxCDF	0.1	96	0.44	0.42	0.27	0.53	0.11	100	0.47	0.48	0.38	0.67	0.1
13		0.1	7	0.07	0.08	0.04	0.10	0.08	17	0.09	0.09	0.06	0.13	0.04
14	1,2,3,7,8,9-HxCDF	0.1	7	0.28	0.25	0.21	0.29	0.08	13	0.28	0.26	0.25	0.36	0.1
15	2,3,4,6,7,8-HxCDF	0.01	56	0.09	0.08	0.04	0.11	0.02	71	0.09	0.10	0.07	0.14	0.02
16		0.01	8	0.01	0.01	0.01	0.01	0.0	13	0.01	0.01	0.01	0.01	0.00
17	1,2,3,4,6,7,8-HpCDF	0.0003	0	0.00	0.00	0.00	0.00	0.0	5	0.00	0.00	0.00	0.00	0.0
	1,2,3,4,7,8,9-HpCDF	-	-	5.34	5.04	3.87	7.35	1.3	-	5.65	6.66	4.63	9.30	1.32
	OCDF	-	-	13.95	13.86	9.82	16.92	-	-	15.54	15.32	12.10	21.40	-
	Total PCDFs TEQ													
	Total PCDD/Fs TEQ													

Sequence of congeners
By using the 2005 WHO toxic equivalency factor [26]
Percentage to number of detection for each Individual congener in all samples

Individual congeners Levels concentration of PCDD/Fs (WHO2005–TEQ DF/g of lipid) and percentage to number of detection for each Individual congener, each congener individual was given Sequence of congeners, the results shown in Table 2 and Fig.3. Also contains Table 3 the levels of concentration of

each Individual congener for category 1 (control group).

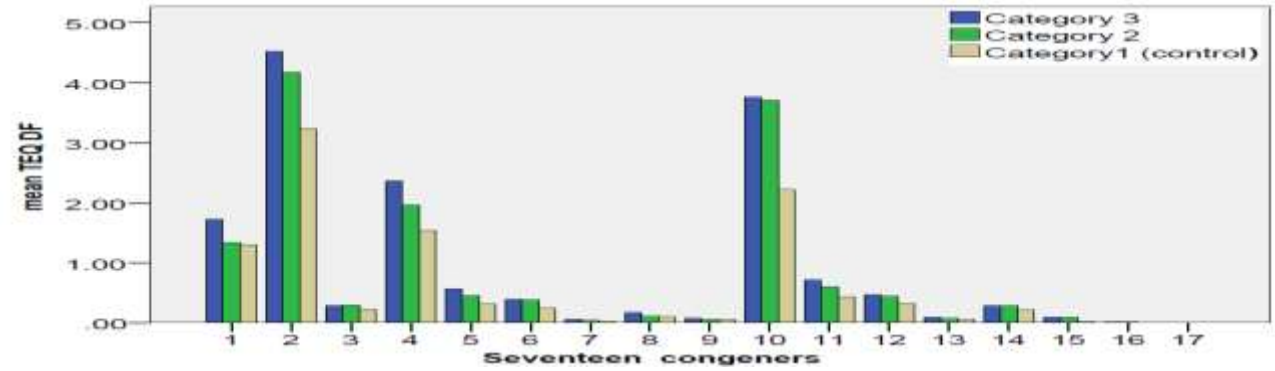

Fig.3: TEQ DF concentrations and Individual congener's profiles in serum samples from all subjects

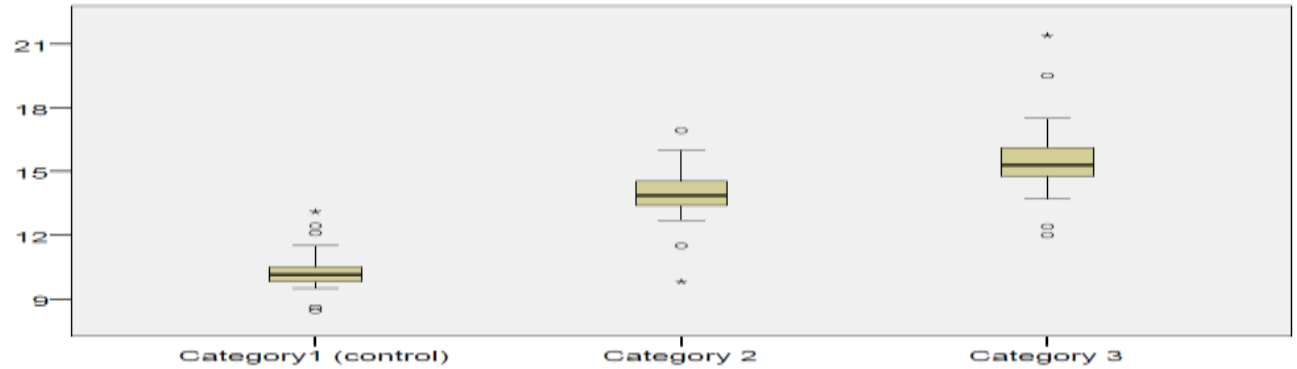

Fig.4: Levels of TEQ DF in both categories and Category 1(control group), the dark line in the middle of the boxes is the median. Indicates the bottom of the boxes to the percentile cases, lie within the boxes. The T-bars that extend from outside the boxes is the minimum or maximum values. The points are outliers. The asterisks or stars are extreme outliers

PCDD/Fs exposure is an emerging health problem in the world. While there are some studies in laboratory animals supporting a possible association between dioxin exposure and chronic kidney disease.

Table 3: Concentration levels each Individual congener for category 1 (control group) (pg WHO–TEQ DF /g lipid) in general (non-exposed) populations

Congeners	TEQ concentration (pg/g lipid) category 1 (control group)			
	min	max	median	mean
Chlorinated dibenzo-p-dioxins 2,3,7,8-TCDD	0.69	1.28	1.1	1.35
1,2,3,7,8-PeCDD	2.66	3.41	2.92	3.21
1,2,3,4,7,8-HxCDD	1.61	2.86	2.27	2.06
1,2,3,6,7,8-HxCDD				
1,2,3,7,8,9-HxCDD				
1,2,3,4,6,7,8-HpCDD	0.15	0.45	0.26	0.24
OCDD	0.02	0.04	0.027	0.02
Total PCDDs TEQ	5.14	8.04	6.55	6.87
Chlorinated dibenzofurans 2,3,7,8-TCDF	0.07	0.25	0.08	0.10
1,2,3,7,8-PeCDF	0.03	0.06	0.046	0.05
2,3,4,7,8-PeCDF	2.41	3.35	2.27	2.42
1,2,3,4,7,8-HxCDF	0.8	1.32	1.08	0.76
1,2,3,6,7,8-HxCDF				
1,2,3,7,8,9-HxCDF				
2,3,4,6,7,8-HxCDF				
1,2,3,4,6,7,8-HpCDF	0.01	0.09	0.09	0.05
1,2,3,4,7,8,9-HpCDF				
OCDF	0.0	0.0	0.0	0.0
Total PCDFs TEQ	3.32	5.04	3.56	3.38
Total PCDD/Fs TEQ	8.45	13.11	10.14	10.25

PCDD/Fs were associated with decreased renal function, and serum TEQ DF was an important determinant of creatinine and uric acid serum, thus affecting the Glomerular Filtration Rate (GFR). A decrease in the GFR accounts for the hyperuricemia associated

with failure and insufficiency renal [27].It may be possible that a lower GFR will decrease the amount of PCDD/Fs eliminated from the body, therefore, cause an increase in serum PCDD/Fs levels, PCDD/Fs Become slowly excreted by the kidney because these compounds are lipophilic and poorly degradable by the enzymatic machinery [28].

I.e. that TEQ DF works as a double-edged sword. Other data demonstrated that TCDD may lead to an increase in blood pressure via increased renal oxidative stress and vascular reactivity. However, melatonin might ameliorate the blood pressure disturbed by TCDD in part by decreasing the oxidant activity induced by TCDD [29].A recent data found that a high dioxin level remained an independent predictor of Metabolic Syndrome (as fasting glucose, triglycerides, high-density lipoprotein (HDL) and blood pressures) in men, but not in women, regardless of the age at starting exposure [30].

In New Zealand, historical exposure: the highest TCDD lipid concentration 11 times higher than the comparative 1997 national average with exposure in 1968. Elevated TCDD concentrations were observed primarily, in the older study participants [31].In Japan in 1968, an accidental human exposure to rice oil contaminated with PCDDs, led to the development of Yusho disease. Yusho mothers delivered descendants with low birth weights and hyperpigmented skin and mucosa, which are characteristic of fetal Yusho disease [32]. Contradicting with data that preceded it suggest that PCDDs has a negative effect on bone mineral density in women; the findings should be interpreted carefully, because no increase in the serum level of this congener was observed in patients with Yusho disease [33].

In conclusion, we inference that workers collecting and burning of waste have levels of dioxins and furans cannot be underestimated when compared with the control group. The concentration of dioxins increasing constantly with increasing of exposure period. Causing many health risks supported by some laboratory animal studies.

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