DIOXIN CONTENT IN BLOOD OF THOSE WHO WERE BORN AFTER 1990 IN VIETNAM

Vu Chien Thang; Pham Lan Anh[1]; Vu Tung Sơn[2]; Doan Trong Tuyen[3]
1: Office 33, Vietnam Ministry of Natural Resources and Environment; 83 Nguyen Chi Thanh Ha Noi Vietnam; 2: Military Medical Academy; Ha Noi Vietnam; 3: Medical Military Department, Ministry of Defence; Nghi Tam Ha Noi Vietnam.

1. Introduction

Vietnam is known to have been seriously impacted with contamination due to dioxins from defoliant used by the US army during the Vietnam War. From 1961 to 1971, the US army sprayed around 77 million liters of defoliant with several herbicide mixtures such as Agent Orange, Agent Purple, Agent Blue, etc containing dioxins. The main congener was 2,3,7,8 – TCDD. Dioxins are very persistent in the environment. Since the late 1980s, Vietnam's economy developed at high speed together with the establishment of industrial parks which also have possibility to emit dioxins and contaminate the environment.

In the 1990-1992 period, some researches in various provinces and regions were conducted to identify dioxin concentration in blood of Vietnamese people, most of them were born befor 1971[1,4,6]. The synthesis[4] of findings of those researches shows that dioxin concentration in blood of those who live in the sprayed area is 12.6 ppt on average (min: 3.4, max: 32 ppt). It differs regarding dioxin concentration in blood of those who live in the sprayed areas and those who do not live in the sprayed areas with untypical differences among surveyed provinces. Yet, these have not been fully explained. In recent years, some studies identifying dioxin concentration in human blood to assess dioxin exposure risks have been implemented which are mainly in those dioxin heavily contaminated areas known as "hotspots" such as Bien Hoa air base, Da Nang air base[2,3].

This report is extracted as part of "*the dioxin concentration identification originated from Agent Orange and other sources in human blood and some of the regular foods in different regions in Vietnam*" study to assess dioxin exposure status after 1990, over 40 years after the US army terminated their spraying mission in the South of Vietnam. This also initially finds out about the dioxin exposure risks from other emission sources.

2. Materials and methods

2.1. Study location

The study locations are administrative units at ward and communal levels which are classified into areas as follows: 1) Areas with no risks of dioxin contamination from any emission sources (referred to as KV0); 2) areas with dioxin contamination risk from defoliant used by the US army during the Vietnam War (referred to as KVI) and 3) areas with dioxin contamination risk from industrial emission sources (referred to as KVII). The classification of areas subject to the dioxin contamination risks from different sources is implemented basing on the data on defoliant spraying records[7], site surveys of production units with high risk of dioxin emission. However, this classification only has its oriental function for the study and does not confirm the level and sources of dioxin contamination of those locations as it does not quantify dioxins in the environment.

2.2. Standards for blood sampling

Those who were sampled with blood for dioxin analysis were born in 1990 – 1995, and continuously live in the study area for at least 10 recent years. They also do not work in environment with high risk of dioxin exposure such as metallurgy, chemical manufacturing or cement production, etc.

2.3.Dioxin quantification in blood serum

The quantification of 17 congeners of dioxins/furans (PCDD/Fs) introduced by the World Health Organization (WHO) was analyzed at the Dioxin and Toxic Substance Laboratory belonging to Center for Environment Monitoring, Vietnam Environment Administration. The analysis method used was subject to PCDD/Fs analysis procedure in blood and blood serum which is U.S. EPA 1613 B.

Blood serum samples of the same age group, sex and location were combined into one sample for analysis.

3. Results and discussion

The results for quantification of the content of 2,3,7,8 – TCDD, $TEQ_{WHO2005}$ (calculated per the lipid content) and % 2,3,7,8 – TCDD/TEQ are shown details in Table 1,2.

Table 1: 2,3,7,8–TCDD level and $TEQ_{WHO2005}$ upper bound in pooled serum samples of male group (pg/g lipit)

Ward	Area	Sex	n	average age	2,3,7,8 TCDD	TEQ	% TCDD /TEQ
H. Ly	KV0	m	8	20,62	<0,9	8,71	10,34
Tu Ly	KV0	m	13	19,15	2,65	6,11	43,44
T.Dau	KV0	m	20	19,55	<0,9	5,16	17,45
S.Trai	KV0	m	29	20,65	<0,9	4,89	18,41
D.Minh	KV0	m	24	20,39	1,95	6,59	29,64
Y.Cg	KV0	m	52	19,96	<0,9	3,26	27,57
H.Dao	KV0	m	20	19,90	1,93	5,61	34,34
H.Tan	KV0	m	18	18,88	<0,9	7,74	11,63
P.Thng	KV0	m	24	20,91	3,84	6,73	57,11
T.Son	KV0	m	25	21,00	<0,9	8,6	10,46
N.Ha	KV0	m	21	19,90	5,98	10,3	58,05
H.Lam	KVI	m	26	19,50	5,13	12,19	42,07
G.Son	KVI	m	14	20,21	2,48	11,7	21,24
G.Hoa	KVI	m	10	19,00	2,09	12,14	17,21
A.T.Dg	KVI	m	25	20,64	2,37	11,01	21,5
T.D.Hp	KVI	m	20	20,15	<0,9	13,84	6,5
T.Than	KVI	m	25	19,64	5,27	15,35	34,34
V.An	KVI	m	25	20,80	<0,9	7,25	12,42
A.Ngo	KVI	m	15	20,53	<0,9	10,39	8,66
H.Lam	KVI	m	11	20,09	<0,9	5	18,02
B.Than	KVI	m	22	19,09	1,7	11,05	15,39
N.Ninh	KVI	m	22	21,09	5,03	8,42	59,71
Bo Y	KVI	m	25	20,36	<0,9	4,11	21,9
Ro Koi	KVI	m	23	20,91	3,69	5,74	64,3
D.Phog	KVII	m	10	21,0	1,75	8,5	20,59
K.An	KVII	m	10	21,30	<0,9	5,97	15,07
K.Cu	KVII	m	12	20,50	<0,9	4,64	19,41
L.Hien	KVII	m	13	22,15	2,69	6,55	41,13
G.Sang	KVII	m	29	19,82	1,45	5,03	28,74
C.Gia	KVII	m	37	20,62	<0,9	4,9	18,38
B.Son	KVII	m	30	19,73	<0,9	3,98	22,6
L.Hiep	KVII	m	30	20,26	<0,9	9,73	9,25
T.Tho	KVII	m	26	20,06	<0,9	23,71	3,8

LOQ (Limit of quantification) = 0,9 pg/g lipit; TEQ upper bound = $\sum (C_i \times TEF_i)$, i: 17 congeners PCDD/Fs, level of congeners smaller then LOQ by calculation of TEQ using the value of LOQ.

3.1. $TEQ_{WHO2005}$ in blood serum

In the areas with no risks of dioxin contamination from any emission sources (KV0), there were total 11 serum samples each sex group collected and analyzed: average TEQ in male group was 6.70 pg/g lipid (3.26-10.30); and respectively on female sample at 9.95 pg/g lipid (3.63 - 26.96). The difference of TEQ between male and female is no statistically significant with $p > 0.05$ ($p = 0:18$).

In areas with dioxin contamination risk from defoliant (KVI total 13 serum samples each sex group collected and analyzed: average TEQ in male group was 9.86 pg/g lipid (4.11-9.86); in female samples average TEQ was of 7.53 pg/g lipid (3.92-11.53). The difference of TEQ between male and female is statistically significant with $p<0.1$ ($p=0.059$).

In areas with dioxin contamination risk from industrial emission sources (KVII): there were total 9 blood serum samples each sex group collected and analyzed: average TEQ in male group was was 8.11pg/g lipid (3.98-23.71); in female samples with average TEQ of 8.55 pg/g lipid (4.40- 17.37). The difference of TEQ between male and female is no statistically significant with $p>0.05$ ($p=0.86$).

3.2. The percentage of 2,3,7,8 – TCDD in total TEQ

The proportion (percentage) of congener 2,3,7,8 – TCDD in total TEQ is an indicator for the dioxin emission sources, if dioxins are from defoliants, the percentage of 2,3,7,8 – TCDD/TEQ is usually higher than dioxins from other emission sources[2,3,5]. The results show that even the highest percentage of % 2,3,7,8 – TCDD/TEQ was at only 64.3% and found in Ro Koi (belonging to KVI). The differences of proportion of congener 2,3,7,8 – TCDD in total TEQ among studied areas however do not show statistical significance (Table3).

The results found in this report is only part of "*the dioxin concentration identification originated from Agent Orange and other sources in human blood and some of the regular foods in different regions in Vietnam*" study. The data regarding other age groups and foods in areas KV0, KVI and KVII are being analyzed and will provide a more comprehensive assessment. Nevertheless, from this initial result, it does not show the difference of dioxin concentration in blood of those who were born after 1990 in areas with different exposure risks. Previous studies[1,4,6] show that dioxin concentration found in blood of those who lived in KVI area higher than other areas. Yet this does not reflect in our study which means there is a possibility that dioxin residue in this area has been much reduced than before.

Table 2: 2,3,7,8 – TCDD level (pg/g lipit) and $TEQ_{WHO2005}$ (upper bound) in serum pooled samples

Ward	Area	Sex	n	average age	2,3,7,8 TCDD	TEQ	% TCDD /TEQ
H. Ly	KV0	f	7	19,85	<0,9	26,96	3,34
Tu Ly	KV0	f	18	20,22	2,72	6,42	42,39
T.Dau	KV0	f	26	19,65	<0,9	17,83	5,05
S.Trai	KV0	f	21	20,09	<0,9	4,49	20,04
D.Min	KV0	f	26	19,46	1,68	10,85	15,49
Y.Cg	KV0	f	26	20,15	2,09	4,43	47,2
H.Dao	KV0	f	25	20,94	2,11	6,35	33,3
H.Tan	KV0	f	12	19,33	<0,9	3,63	24,78
P.Thng	KV0	f	25	21,60	<0,9	16,19	5,56
T.Son	KV0	f	24	21,41	<0,9	6,14	14,65
N.Ha	KV0	f	23	20,65	<0,9	6,21	14,48
H.Lam	KVI	f	22	19,53	<0,9	7,82	11,5
G.Son	KVI	f	13	19,53	1,63	5,96	27,33
G.Hoa	KVI	f	9	19,00	3,53	9,24	38,17
A.T.Dg	KVI	f	25	20,40	<0,9	11,53	7,81
T.D.Hp	KVI	f	31	20,15	<0,9	5,17	17,41
T.Than	KVI	f	32	20,76	2,71	5,9	45,96
V.An	KVI	f	25	21,28	<0,9	6,25	14,41
A.Ngo	KVI	f	12	19,75	2,35	6,86	34,24
H.Lam	KVI	f	11	20,09	3,99	7,44	53,69
B.Than	KVI	f	25	20,56	<0,9	10,71	8,4
N.Ninh	KVI	f	25	20,44	1,82	11,07	16,47
Bo Y	KVI	f	38	20,35	<0,9	3,92	22,97
Ro Koi	KVI	f	24	19,79	1,62	6,07	26,69
D.Phog	KVII	f	4	19,75	<0,9	13,38	6,72
K.An	KVII	f	20	21,10	1,69	4,63	36,43
K.Cu	KVII	f	8	20,12	<0,9	6,09	14,77
L.Hien	KVII	f	10	21,80	1,91	5,08	37,57
G.Sang	KVII	f	30	20,50	<0,9	4,4	20,46
C.Gia	KVII	f	26	21,03	1,74	5,91	29,38
B.Son	KVII	f	23	19,47	1,62	6,43	25,21
L.Hiep	KVII	f	19	20,50	<0,9	13,7	6,57
T.Tho	KVII	f	26	21,15	<0,9	17,37	5,18

LOQ (Limit of quantification) = 0,9 pg/g lipit; TEQ upper bound = □ (C_i x TEF_i), i: 17 congeners PCDD/Fs, level of congeners smaller then LOQ by calculation of TEQ using the value of LOQ.

4. Acknowledgements

We would like to express our sincere thanks to the Ministry of Natural Resources and Environment for creating favorable condition for us to conduct this study; our thanks also go to colleagues who have contributed great effort to this study.

Table 3: The percentage of 2,3,7,8 – TCDD in total TEQ

Area	Sex	n	Mean (min-max)	Standard Deviation
KV0	M	11	28.95 10.34-58.05	17.64
	F	11	20.57 3.34-47.20	14.92
KVI	M	13	26.40 6.50-64.30	18.50
	F	13	25.00 7.81-53.69	14.52
KVII	M	9	19.89 3.80-41.13	10.81
	F	9	20.25 5.18-37.57	12.73

References:

1. Arnold Schecter, MD, MPH, Le Cao Dai, MD, Le Thi Bich Thuy et al (1995): Agent Orange and the Vietnamese: The Persistence of Elevated Dioxin Levels in Human Tissues. American Journal of Public Health April 1995, Vol. 85. No. 4 p; 516 -22;
2. Hatfield Consultants; Office of the National Committee 33 (2009): Comprehensive Assessment of Dioxin Contamination in Da Nang Airbase, Viet Nam: Environmental Levels, Human Exposure and Options for Mitigating Impacts. Final Report; August 2009. Office of the National Committee 33, MONRE.
3. Hatfield Consultants; Office of the National Committee 33 (2011): Enviromental and Human health Assessment of Dioxin Contamination in Bien Hoa Airbase, Viet Nam: Final Report; August 2011. Office of the National Committee 33, MONRE.
4. Hoàng Đình Cầu, Trần Mạnh Hùng, Phùng Chí Dũng và CS (2000): Tiểu mục 2: Thống kê kết quả phân tích dioxin/furan tại các phòng thí nghiệm nước ngoài hợp tác với Ủy ban 10 – 80. Hậu quả các chất hóa học đã sử dụng trong chiến tranh Việt nam 1961 – 1971. Kỷ yếu công trình, Quyển II, Phần một. Ủy ban quốc gia điều tra hậu quả chất hóa dùng trong chiến tranh Việt Nam, Hà Nội 2000; trang 205 – 209.

5. Nguyễn Hùng Minh (2008): Xây dựng mô hình nhận dạng dioxin từ chất độc hóa học do Mỹ sử dụng và các nguồn phát thải tiềm tàng khác ở Việt Nam. Bộ Tài nguyên và Môi trường, 6/2008.
6. Schecter, A., Dai, L.C., et al (2001): Recent dioxin contamination from Agent Orange in residents of a southern Vietnam city. J. Occup. Environ. Med. 43; p 435-443.
7. Văn phòng Ban chỉ đạo 33 (2011): Bản đồ băng rải và mật độ chất diệt cỏ do quân đội Mỹ sử dụng trong chiến tranh ở Việt Nam. Văn phòng Ban chỉ đạo 33, 2011.