PERGAMON

Chemosphere 47 (2002) 117–137

CHEMOSPHERE

www.elsevier.com/locate/chemosphere

Dioxin reservoirs in southern Viet Nam—A legacy of Agent Orange

L. Wayne Dwernychuk [a,*], Hoang Dinh Cau [b], Christopher T. Hatfield [a], Thomas G. Boivin [a], Tran Manh Hung [b], Phung Tri Dung [b], Nguyen Dinh Thai [b]

[a] *Hatfield Consultants Ltd., Suite 201-1571 Bellevue Avenue, West Vancouver, BC, Canada V7V 1A6*
[b] *10-80 Committee, Ministry of Health, 35 Nguyen Huy Tuong Street, Thanh Xuan District, Ha Noi, Viet Nam*

PERGAMON

Chemosphere 47 (2002) 117–137

CHEMOSPHERE

www.elsevier.com/locate/chemosphere

Dioxin reservoirs in southern Viet Nam—A legacy of Agent Orange

L. Wayne Dwernychuk [a,*], Hoang Dinh Cau [b], Christopher T. Hatfield [a], Thomas G. Boivin [a], Tran Manh Hung [b], Phung Tri Dung [b], Nguyen Dinh Thai [b]

[a] *Hatfield Consultants Ltd., Suite 201-1571 Bellevue Avenue, West Vancouver, BC, Canada V7V 1A6*
[b] *10-80 Committee, Ministry of Health, 35 Nguyen Huy Tuong Street, Thanh Xuan District, Ha Noi, Viet Nam*

Received 19 June 2001; received in revised form 16 October 2001; accepted 26 October 2001

Abstract

In the isolated Aluoi Valley of central Viet Nam, very high levels of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) were measured in soil, fish fat, duck fat, pooled human blood and breast milk samples collected from A So village between 1996 and 1999. The village was situated on a former military base occupied by US Special Forces between 1963 and 1966. TCDD was a contaminant of the herbicide "Agent Orange", aerially sprayed in the valley between 1965 and 1970, and stored at the A So base. Measured levels were lower near the sites of two other former US bases in the valley which had been occupied for shorter periods of time. In areas where Agent Orange had been applied by low-flying aircraft, levels of TCDD in soil, food and human samples were elevated, but lower than those near the three former US bases.

We confirm the apparent food chain transfer of TCDD from contaminated soil to cultured fish pond sediments to fish and duck tissues, then to humans as measured in whole blood and breast milk. We theorize that the Aluoi Valley is a microcosm of southern Viet Nam, where numerous reservoirs of TCDD exist in the soil of former military installations south of the former demilitarized zone. Large quantities of Agent Orange were stored at many sites, used in ground and aerial applications, and spilled. TCDD, through various forms of soil disturbance, can be mobilized from these reservoirs after decades below the surface, and subsequently, introduced into the human food chain.

Keywords: Herbicide; 2,3,7,8-tetrachlorodibenzo-*p*-dioxin; Ranch Hand; Soil; Food; Human blood; Human milk

1. Introduction

The US armed forces started using herbicides in Viet Nam in 1962; the operation, code-named Ranch Hand, expanded in 1965 and 1966, and was terminated in 1971 (IOM, 2001). It has been estimated that over 72 million litres of herbicide were applied over Viet Nam, south of the former demilitarized zone on the 17th parallel (Westing, 1984; Cecil, 1986).

Herbicide was used for general defoliation, to enhance the detection of camps, trails and infiltration routes of opposing forces. Crop destruction was another use for herbicides in order to weaken opposing troops and force a diversion of manpower to food cultivation, procurement, and transportation. Ground applications of herbicide occurred from trucks or backpack sprayers

* Corresponding author. Tel.: +1-604-926-3261; fax: +1-604-492-65389.
E-mail address: wdwernychuk@hatfieldgroup.com (L.W. Dwernychuk).

0045-6535/02/$ - see front matter
PII: S0045-6535(01)00300-9

to defoliate perimeters of military base camps and artillery bases. Agent Orange was the principal phenoxy herbicide mixture that was employed for these tasks (IOM, 1994), making up approximately 61% of recorded herbicides used (IOM, 2001). It was a 50/50 mixture of 2,4-dichlorophenoxyacetic acid (2,4-D) and 2,4,5-trichlorophenoxyacetic acid (2,4,5-T). 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) was a contaminant of the 2,4,5-T portion of the Agent Orange mixture (Dwyer and Flesch-Janys, 1995; IOM, 2001). According to Dow Chemical, one of the manufacturers of Agent Orange, levels of TCDD varied from <0.05 mg/l to ≈50 mg/l. Those values were determined in stocks of Agent Orange either returned from Viet Nam or purchased by the US military but not transported to Viet Nam (IOM, 2001). The estimated average TCDD concentration in Agent Orange, which would translate to the often quoted 170 kg of TCDD applied over southern Viet Nam, was 4.0 mg/l (Westing, 1984; Gough, 1986; IOM, 2001). Bengtsson (1976) reported a higher estimate of the average TCDD concentration in Agent Orange, ≈16 mg/l which would, therefore, increase the loading of pure TCDD to southern Viet Nam to ≈680 kg. Deriving an accurate average content is not possible.

During the late 1960s, and continuing to the present, there was concern regarding human health impacts caused by Agent Orange, or more specifically TCDD. Initially, attention had focussed on direct ecological effects of herbicides (Tschirley, 1969; Orians and Pfeiffer, 1970; Boffey, 1971; Westing, 1984). Interest widened to include toxic effects on humans following the report that 2,4,5-T was teratogenic in rodents (Courtney et al., 1970). Given that TCDD is viewed as one of the most toxic congeners in the dioxin family (Dwyer and Flesch-Janys, 1995; Hu and Bunce, 1999), it has been the primary focus of numerous investigations regarding Agent Orange and contamination of human tissues (Schecter et al., 1985, 1987, 1989b, 1990b, 1992b, 1995, 2001; Dai et al., 1994a,b, 1995; Phiet et al., 1994; Schecter, 1994a; Roumak et al., 1995). Numerous toxic effects in humans have been associated with TCDD exposure (IARC, 1997; ATSDR, 1998).

Residual TCDD concentrations in Viet Nam are a function of spray frequency and distribution, partitioning, bioavailability, recycling in the ecosystem, decomposition rate, and the presence or absence of historical storage sites for Agent Orange. Aerial applications of Agent Orange covered an estimated 10% of southern Viet Nam (IOM, 1994). Large C-123 cargo planes, carrying 1000 US gallons of Agent Orange, dispensed their payload at low altitudes, in a single continuous spray pattern 14 km in length for a run duration of 4.5 min (Cecil, 1986). Bulk storage facilities at the Bien Hoa base, near Ho Chi Minh City, housed up to 90 000 US gallons of herbicide, in addition to large storage areas for 55 US gallon drums (Cecil, 1986). Herbicide supply operations were also carried out at Da Nang, Nha Trang, Phu Cat and Saigon (US Army documents, 1969).

Accordingly, there are two major sources of potential TCDD contamination in the Vietnamese environment; the first, from spray missions by aircraft, the second at former US and allied military installations where Agent Orange was stored, dispensed, and used for perimeter spraying.

Storage sites on military installations experienced spills. At the former Bien Hoa base, a 7500 US gallons of Agent Orange were spilled on 1 March, 1970. Three smaller spills of several hundred gallons each also occurred at this base between January and March, 1970 (US Army documents, 1970).

During the Viet Nam conflict, numerous military bases were established throughout southern Viet Nam. The Aluoi Valley (Thua Thien Hue Province), in particular, was subjected to heavy military activity. It was an integral, strategic portion of the Ho Chi Minh Trail, a conduit for men and equipment moving from the north into southern Viet Nam. Early in the war, the valley was the site of three US Special Forces bases (Fig. 1), and was sprayed repeatedly with Agent Orange, up to 11 times in some areas (Cecil, 1986; Zaffiri, 1988; US Army documents, 1995). Accordingly, the Aluoi Valley was ideal for our study of residual dioxin contamination in the environment and humans.

In December 1965, the Special Forces bases at Aluoi and Ta Bat were deemed untenable by the US military and abandoned after less than a year of operation. The base at A So (formerly named A Shau) was opened in April 1963 (Brown, 1999), but following an attack by the North Vietnamese in March 1966, this Special Forces base was also abandoned (Stanton, 1985). During US occupation of the A So base, Agent Orange was stored and used on site dating back to 1963 (Declassified US military documents, authority NND 931713/903562, National Archives, Washington, DC, 2001).

The hill tribe people (primarily Pa Co, Ca Tu, and Ta Oi) that presently inhabit the Aluoi Valley, represent an isolated population which experiences little in- or out-migration. The valley has no industry and, therefore, has no industrial emissions which might have confounding results. The people are poor and subsist as an agrarian society raising vegetables, rice, manioc, poultry, some livestock and cultured fish, primarily carp. For economic reasons, pesticide use in the valley is minimal.

2. Materials and methods

Between 1996 and 1999, samples of soil, fish pond sediment, foods, whole human blood and human breast milk were collected from the valley and its residents.

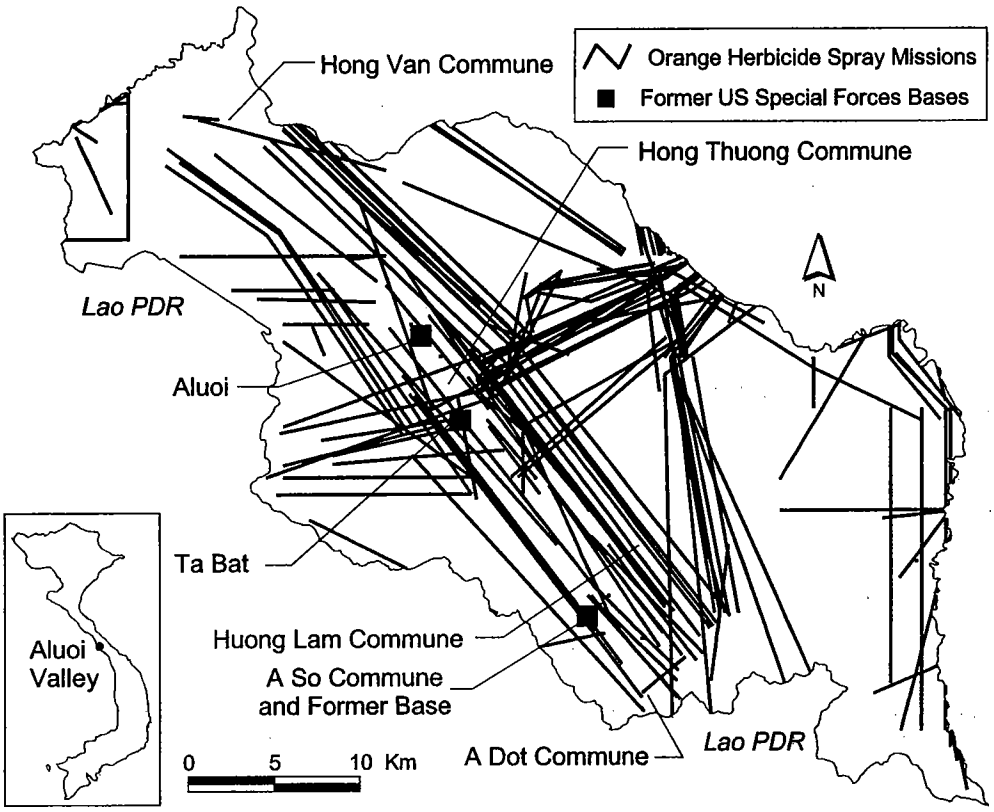

Fig. 1. Location of communes studied and Agent Orange aerial spray missions, Aluoi Valley, Viet Nam, 1965–1970 (source: US Department of the Army).

Field supplies for sample collection, storage and shipment originated in Canada.

2.1. Soil and fish pond sediment

A stainless steel soil corer was used to collect samples throughout the valley. A mine detector was used first, to ascertain that a sampling location was clear of buried unexploded ordnance (UXO). UXO, in many areas, littered the landscape, particularly on the Ta Bat base.

In 1996 and 1997, soil was collected from the A So Special Forces base, and at select locations throughout the valley, from 0 to 10 cm depth stratum. In 1999, the two principal sampling locations were soil from areas sprayed with Agent Orange by C-123 aircraft, and soil from the three former US Special Forces bases (Fig. 1). Samples from spray zones were collected at least 40 m from the main road which runs through the valley from A Dot commune in the south, to Hong Van commune in the northwest.

The sampling design for soil collections from the former bases used the visible traces of the former airstrips as north/south axes. The length of an airstrip was partitioned into three segments, north, centre, and south. In each of these segments, three sites were established (west, centre (in the middle of the strip), and east); therefore, a total of nine primary sites were established for each of the three bases, for a total of 27 former military base sites.

Each primary soil sample from the military bases or from aerially sprayed locations consisted of 10 subsamples composited to form a single test medium. Subsamples were collected within a 30 m radius of the primary site. All samples were placed in pre-cleaned glass jars with teflon lids and frozen. Following collection of each core, equipment was washed with acetone and hexane to prevent cross contamination.

Fish pond sediments were collected from water depths of approximately 60 cm. Bottom sediments were scooped into pre-cleaned glass jars with teflon lids and frozen.

2.2. Vegetable and animal tissues

Samples of rice, manioc and vegetable cooking oil were obtained from commune residents throughout the valley between 1996 and 1999. Ducks, chicken eggs, cultured fish and wild fish were also obtained from

commune residents, and dissected at our field premises. Latex gloves and stainless steel dissecting equipment were used during sample processing in the field, and rinsed with acetone and hexane between dissections. All samples were placed in pre-cleaned glass jars with teflon lids and frozen.

Beef and pork samples were obtained from the local market. All animal and vegetable matter was produced in the Aluoi Valley.

2.3. Whole human blood and breast milk

In 1999, four communes were selected for collection of whole human blood, A So (on the site of the former US Special Forces base), Houng Lam (adjoining A So), Hong Thuong, and Hong Van (Fig. 1). Hong Van received the fewest aerial applications of Agent Orange.

Volunteers from each commune were divided into four groups: males $\geqslant$ 25 years of age (born before or during the war), males $<$ 25 years of age (born after the war), females $\geqslant$ 25 years of age, and females $<$ 25 years of age.

Whole blood was collected by health practitioners from Thue Thien Hue Provincial Health Department under our supervision. Samples were collected in vacutainer hemoguard tubes containing sodium heparin. All samples were frozen within 1 h of collection. Because of health issues and cultural sensitivities, there was permission for a maximum of 3.5 ml of blood to be extracted from each person. Therefore, pooling of blood samples was necessary, and was done in the laboratory in Canada.

Four lactating primaparous females from each of the four communes listed above were selected for collection of human milk samples. Volunteers provided 15–50 ml of milk. A single multiparous female with 10 children also donated milk at our request; this female had given birth to a child with spina bifida who was 2 years of age and being breast fed at the time. All samples were placed in pre-cleaned glass jars with teflon lids and frozen within 1 h of collection.

2.4. Laboratory analysis

All samples from Viet Nam were forwarded in a frozen state to AXYS Analytical Services, Sydney, British Columbia (BC), Canada. AXYS has been certified by the World Health Organization (WHO) for dioxin analysis in human blood and breast milk (WHO/EURO, 2001).

All soil and biological samples being tested for polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) were spiked with ^{13}C-labelled surrogate standards (tetrachlorodioxin, tetrachlorofuran, pentachlorodioxin, pentachlorofuran, hexachlorodioxin, hexachlorofuran, heptachlorodioxin, heptachlorofuran, and octachlorodioxin) prior to analysis. Soil samples were soxhlet extracted. Tissue samples were ground with sodium sulphate, loaded into a glass chromatographic column and eluted with solvent. All whole blood and breast milk samples were spiked with ^{13}C-labelled surrogate standards (as listed above) prior to analysis. Samples were liquid/liquid extracted by shaking with solvent. All extracts were subject to a series of chromatographic cleanup steps prior to analysis for PCDDs and PCDFs by high resolution gas chromatography with high resolution mass spectrometric detection (HRGC/HRMS).

The laboratory quality assurance/quality control (QC) program includes matrix specific method recovery studies, verification of standard solution accuracy against recognized standard reference solutions, analysis of certified reference materials, and participation in interlaboratory comparison programs.

The accuracy of 2,3,7,8-TCDD in the standard solutions used for analysis was verified against NIST SRM 1614; the accuracy of other 2,3,7,8-substituted PCDD and PCDF congeners was verified against a standard reference solution characterized by interlaboratory testing (12 independent labs). Sediment and tissue certified reference materials supplied by Environment Canada and NIST were analyzed to demonstrate the method accuracy for real samples. The program of interlaboratory testing includes participation in studies organized by the University of Umeå, the WHO, QUASIMEME, and Environment Canada encompassing sediment, tissue, milk, and blood samples.

Samples were analyzed in batches alongside QC samples. Each analysis batch included a laboratory blank to demonstrate acceptable laboratory background levels, a spiked matrix reference sample to demonstrate analyte recoveries, and a duplicate sample (sample size permitting) to demonstrate the analytical precision achieved. The results for the batch QC samples must fall within predefined acceptance limits for the sample data to be accepted. On-going evaluation of QC sample data was conducted to ensure the analytical system was operating in a state of control.

Total toxic equivalents (TEQ) for each sample analyzed were calculated in the laboratory using the "international" dioxin TEQ (I-TEQ; NATO, 1998). For non-detectable (ND) and NDR (chromatographic peak was detected, but did not meet quantification criteria) designations, half the detection limit of the sample was used in the total I-TEQ calculation.

3. Results and discussion

3.1. Soil

We found high levels of dioxins at the former military bases, and lower but elevated concentrations in soils from areas aerially sprayed with Agent Orange. Some

concentrations corresponded to highly polluted industrial areas elsewhere, and many exceeded health or safety guidelines in place in other jurisdictions.

3.1.1. Soil from former military bases

Soil from the former Special Forces base at A So was elevated in TCDD both in 1996 (110 pg/g) and in 1997 (897.85 pg/g, Table 1). These high concentrations led to the sampling design applied in 1999 to bases and aerially sprayed regions of the valley.

In 1999, TCDD levels on the former base at A So ranged from 4.2 pg/g at the southeast sector of the base, to 360 pg/g at the north-center site (Table 1). Soil from the former bases at Aluoi and Ta Bat also had elevated concentrations of TCDD (maximum of 35 pg/g at Ta Bat, Table 1), but lower than at A So. These two bases were not in operation for an extended period, compared to A So, and therefore, Agent Orange use on these bases was probably much less. However, all three bases had generally higher TCDD levels in soil than those at distant locations which had only received aerial spray (see below), indicating higher usage rates of Agent Orange on the bases.

TCDD was responsible for a high proportion of the total I-TEQ in all of the soil samples from former bases. In 1996 and 1997 samples from the former A So base, showed that TCDD represented 97.7% and 99.6%, respectively, of the total I-TEQ (Table 1). Other dioxin and furan congeners were low, and might have resulted from the use of other herbicides, wood preservatives, pesticides or incineration. In 1999 at A So, the contribution of TCDD to total I-TEQ exceeded 85.7% in all cases, and the northern sites all approached 100%. There is little doubt that Agent Orange was the source of TCDD contamination in the soil where the present-day A So commune is established. All soil sampled in 1999 from the former Aluoi and Ta Bat bases also showed that TCDD contributed significantly to the total I-TEQs. The lowest proportion was 78.2% (centre east, Ta Bat base), with all others in the 80–90% range (Table 1). Other congeners of PCDD and PCDF were detected in all soil samples from the former Special Forces bases, but their contribution to overall toxicity was small (Table 1).

3.1.2. Soil from aerially sprayed areas

PCDDs and PCDFs were lower in locations away from the former military bases, locations that had received only aerial spraying (Table 2). These locations extend from A Dot commune at the southern end of the valley, northwest through the other communes to Hong Van (Fig. 1). Total I-TEQs ranged from less than 1 pg/g to less than 16 pg/g (Table 2), compared to TEQs from about 5 pg/g to several hundred pg/g in soils of the former bases. TCDD levels in the spray zone ranged from ND to 15.0 pg/g, but mostly below 5 pg/g, compared to the 4 pg/g to several hundred pg/g at the bases. The less extreme levels in soils of sprayed areas would be expected, since they did not receive anything comparable to the protracted use of Agent Orange for perimeter clearance at the bases, or spills of stored material.

TCDD was again a major contributor to total I-TEQ at the sprayed-only sites, but somewhat less important than in soils at the bases. The percentages of total I-TEQ were principally in the 50–80% range. Only four of the 18 soil samples from aerially sprayed communes exceeded the minimum proportion of 85.7% found at military bases (Huong Phong, Hong Quang, Aluoi Market, and Hong Kim; Table 2). The highest proportion was 95%.

There were relatively high levels of octachlorinated dibenzodioxins (O8CDD) in many of the samples from the aerially sprayed zones, probably reflecting the common practice of burning refuse, throughout the valley communes. These substances contributed very little to the total potential toxicity measured by the I-TEQ.

3.1.3. Studies by others

Some other workers have measured TCDDs in soils from Viet Nam. Matsuda et al. (1994) studied soils from various regions in southern Viet Nam in 1989–1991, but 80% of their 106 samples did not yield detectable concentrations. In the other 20%, they found a range from 1.2 to 59.2 pg/g. The locations and soil concentrations of TCDD were: Phu Loc (Thua Thien Hue Province) 4.37–16.8 pg/g; Ho Chi Minh City 2.98–59.2 pg/g; Tay Ninh Province 1.23–38.5 pg/g; and Song Be 6.0 pg/g. In our study, TCDD was detected in 51 of 52 soil samples from the Aluoi Valley, at somewhat comparable levels for the sprayed areas away from former bases (Tables 1 and 2). Matsuda et al. (1994) considered that leaching and runoff reduced most TCDD levels to the point of non-detection.

Aluoi Valley soil was sampled by Quynh et al. (1994) who reported TCDD of 1.0 pg/g at 20 cm depth. They found a much higher level of 62.7 pg/g at 10 cm depth in soils from Bach Ma, situated between the cities of Hue and Da Nang. The concentration dropped to 17 pg/g in the 10–20 cm depth fraction. Matsuda et al. (1994) indicated that dioxins were not detected at soil depth > 10 cm. Thus Quynh et al. (1994) showed that dioxins decrease with depth in the soil, but are still measurable, although Matsuda et al. (1994) had not detected them at depths > 10 cm.

Some results are available for industrial soils in the US. Nestrick et al. (1986) collected samples near major steel, automotive or chemical manufacturing facilities, and in the vicinity of municipal solid-waste incinerators in several mid-western and mid-Atlantic states. They found that these soils from typical industrialized areas contained TCDD levels below 10 pg/g, with a range from ND to 9.4 pg/g. Soils of the Aluoi Valley away from former military bases, but aerially sprayed, appear

Table 1
PCDDs and PCDFs in soil (0–10 cm depth) and fish pond sediment from former US Special Forces bases, Aluoi Valley, Viet Nam, 1996, 1997 and 1999

Sample location and type	PCDD (pg/g dry weight)[a]						PCDF (pg/g dry weight)[a]						Total I-TEQ	TCDD as % of total I-TEQ
	TCDD	Total T4CDD	Total P5CDD	Total H6CDD	Total H7CDD	Total O8CDD	2,3,7,8-TCDF	Total T4CDF	Total P5CDF	Total H6CDF	Total H7CDF	Total O8CDF		
A So (1996)														
Former airbase soil	110	120	7.8	13	47	460	3.6	11	8.8	8.4	28	36	112.6	97.7
Fish pond sediment	6.9	9.5	3.1	9.1	19	460	0.6	1.7	1.4	0.5	0.8	1.2	7.8	88.5
A So (1997)														
Soil														
Former airbase	897.85	897.85	7.76	24.35	68.44	563.84	10.46	30.30	23.59	8.19	19.83	16.50	901.22	99.6
Former airstrip	88.32	88.32	7.40	19.83	65.82	697.05	3.08	11.27	6.94	8.94	22.37	30.31	92.21	95.8
Manioc field	6.61	8.26	1.56	8.49	10.92	142.29	0.64	3.17	0.87	0.55	0.92	1.61	7.01	94.3
Fish pond sediment														
Fish pond #3	8.5	11	3.3	5.8	9.4	220	0.5	2.2	1.1	0.2	ND (0.4)	ND (0.6)	9.2	92.4
Fish pond #2	5.4	7.1	4.5	9.3	7.9	170	0.3	1.5	0.9	0.2	ND (0.4)	ND (0.6)	6.0	90.0
Fish pond #1	5.2	9.9	13	10	5.3	64	0.3	2.3	1.1	0.3	ND (0.4)	ND (0.6)	5.9	88.1
Fish pond #4	1.8	3.3	2.5	1.5	1.4	23	0.1	0.6	0.4	ND (0.2)	ND (0.4)	ND (0.6)	2.0	90.0
A So (1999)														
Soil														
North west	220	220	8.7	17	32	330	3.7	11	9.3	7.7	11	11	220	>99
North centre	360	370	13	16	16	310	11	25	37	3.9	4.2	4.6	360	>99
North centre (dup.)	260	260	9.2	11	13	280	7.1	17	25	3.1	4.1	3.8	260	>99
North east	260	280	23	22	10	240	20	35	20	4.4	1.5	1.2	260	>99
Centre west	24	36	28	23	7.5	120	2.1	12	9.1	1.7	1.0	0.9	26	92.3
Centre	25	37	31	25	5.2	69	1.6	6.1	5.7	1.4	0.7	0.7	27	92.6
Centre east	45	54	21	21	7.5	130	2.7	11	9	2.3	0.9	0.9	46	97.8
South west	15	20	6.5	7.6	6.8	75	0.9	6	4.8	1.5	0.9	0.8	16	93.8
South centre	5.4	7.6	3.6	4.4	5.1	77	0.4	2.3	1.6	0.6	0.5	0.6	5.7	94.7
South east	4.2	5.1	2.5	6	9.2	240	0.4	2.4	1.0	0.3	0.9	1.0	4.9	85.7
Ta Bat (1999)														
Soil														
North west	11	16	7.7	9.6	21	880	0.7	5.7	2.8	1.0	1.6	1.6	13	84.6
North centre	9.2	12	2.5	8.0	28	820	0.6	3.2	2.8	3.5	4.9	3.8	11	83.6
North east	9.4	15	7.8	8.5	13	520	1.4	11	11	2.4	2.3	2.1	11	85.5

Centre west	35	40	7.0	8.9	18	800	1.0	5.3	2.5	1.0	1.2	1.3	37	94.6
Centre	5.9	10	6.7	8.0	10	400	0.8	6.5	6.8	0.7	0.9	1.0	7.1	83.1
Centre east	4.3	8.8	1.4	5.7	18	750	0.3	2.7	1.0	0.3	0.6	0.6	5.5	78.2
South west	18	27	5.2	12	19	520	0.4	4.5	2.1	3.0	4.5	3.2	19	94.7
South centre	8.4	16	5.4	10	16	1100	0.5	4.1	2.1	0.8	1.0	1.2	10	84.0
South east	7.7	12	3.1	10	17	530	0.6	4.4	2.8	1.0	1.4	1.5	9.0	85.6
Aluoi (1999)														
Soil														
North west	11	15	6.7	13	7.0	74	0.6	3	2.1	1.0	11.8	1.3	12	91.7
North centre	12	15	4.9	11	6.1	76	0.6	2.1	1.5	0.9	1.8	1.7	13	92.3
North east	5.0	7.3	7.5	10	9.7	140	0.4	1.6	1.0	0.9	1.7	1.7	5.7	87.7
Centre west	12	15	3.9	9.5	8.0	340	0.5	2.2	1.1	0.6	0.7	0.7	13	92.3
Centre	5.7	6.9	1.7	5.7	6.7	210	0.5	1.7	0.9	0.8	1.0	0.6	6.3	90.5
Centre east	19	24	7.4	16	11	370	0.9	5.8	3.1	1.7	1.3	1.2	20	95.0
South west	11	16	13	11	6.6	260	0.5	2.7	1.6	0.6	0.4	0.6	12	91.7
South centre	19	26	18	16	7.0	320	0.7	3.4	2.1	1.1	0.8	0.8	20	95.0
South east	10	14	8.8	11	7.3	250	0.6	3.6	2.4	1.0	0.9	0.7	11	90.9

[a] ND = Not detected; for 'total I-TEQ' calculations, if ND, half the detection level was used. Parentheses enclose the sample detection limit.

to have a range of concentrations comparable to the typical levels of <10 pg/g in the US industrial soils; Aluoi market was the exception, 15 pg/g (Table 2).

Soils near the A So base showed TCDD levels that are more characteristic of soils found near highly contaminated industrialized urban areas in the US. Nestrick et al. (1986) reported TCDD ranging from 22 to 450 pg/g in soils near a Dow Chemical plant in Midland, Michigan which was involved in manufacturing chlorophenolic compounds. The Agency for Toxic Substances and Disease Registry (ATSDR, 1998) reports on a number of studies in heavily polluted locations in Missouri (including Times Beach) where TCDD levels ranged from 30 to 2200×10^3 pg/g. Apart from those highly contaminated locations, the ATSDR (1998) summarizes by stating that soil TCDD concentrations are typically greater in urban areas, with industrial soils clearly exhibiting the highest levels of contamination.

3.1.4. Environmental guidelines and standards

In the USA, the Environmental Protection Agency (US EPA) has responsibility for protecting public health and the environment. Region III of US EPA (Delaware, Maryland, Pennsylvania, Virginia, West Virginia and District of Columbia) has set 4.3 pg/g of TCDD as a residential soil guideline and 38.0 pg/g for industrial soil (US EPA, 1999a). A risk assessment is recommended for soils exceeding those levels.

In US EPA Region IX (Arizona, California, Nevada, Hawaii, Territories of Guam and American Samoa, the Northern Marianna Islands, and other unincorporated US Pacific possessions), the soil guidelines for TCDD are 3.9 and 27 pg/g for residential and industrial soils, respectively (US EPA, 1999b).

The majority of soil samples collected in the Aluoi Valley had TCDD levels that exceeded US EPA residential guidelines for Regions III and IX. Homes have been constructed in many of the areas we sampled, and dirt floors are common throughout Aluoi Valley. TCDD levels we recorded in the valley soils would trigger recommendations for a risk assessment, if reported in US EPA Regions III or IX.

The ATSDR (1997) guideline for dioxin and dioxin-like compounds in residential soils has been set at 50 pg/g total TEQ. If soil in residential regions exceeds that, a further site-specific evaluation is recommended. Even if soil dioxin is <50 pg/g total TEQ, ATSDR indicates that a more detailed site-specific assessment might still be required, based on an assessor's concerns for overall community health in combination with other potential contaminants. If an exposure pathway is identified (e.g., food chain) the ATSDR recommends evaluating the extent of exposure and public health implications based on the likelihood, frequency, routes and levels of exposure, and information on the human populations.

Table 2
PCDDs and PCDFs in soil (0–10 cm depth) from areas aerially sprayed with Agent Orange, Aluoi Valley, Viet Nam, 1999

Commune	PCDD (pg/g dry weight)[a]						PCDF (pg/g dry weight)[a]						Total I-TEQ	TCDD as % of total I-TEQ
	TCDD	Total T4CDD	Total P5CDD	Total H6CDD	Total H7CDD	Total O8CDD	2,3,7,8-TCDF	Total T4CDF	Total P5CDF	Total H6CDF	Total H7CDF	Total O8CDF		
A Dot														
Site 1	1.0	1.0	ND (0.1)	1.5	11	540	0.3	1.7	ND (0.1)	ND (0.2)	0.5	0.7	1.8	55.6
Site 1	0.8	0.8	ND (0.1)	1.1	8.8	430	0.2	1.7	0.3	ND (0.2)	0.4	0.7	1.4	57.1
Site 2	0.4	0.6	ND (0.1)	1.1	3.0	43	0.2	0.9	0.3	ND (0.2)	0.4	0.4	0.62	64.5
Huong Lam														
Site 1	0.4	0.6	ND (0.1)	0.9	3.2	68	ND (0.1)	0.3	ND (0.1)	ND (0.2)	0.4	0.6	0.6	66.7
Site 2	ND (0.1)	0.4	0.3	3.9	32	180	0.2	0.6	0.2	2.7	6.6	6.0	0.6	–
Huong Phong														
Site 1	1.6	2.3	0.3	1.9	7.0	270	0.2	1.3	0.5	0.5	0.8	0.8	2.1	76.2
Site 2	6.7	8.4	2.0	4.4	14	210	0.4	2.6	2.1	1.1	1.2	1.2	7.3	91.8
Phu Vinh														
Site 1	3.0	3.7	0.7	2.4	11	540	0.3	1.1	0.6	0.8	0.6	1.2	3.8	78.9
Site 2	3.1	3.7	1.0	3.2	13	610	0.5	2.6	2.4	0.7	1.3	1.5	4.1	75.6
Hong Thuong	5.1	6.8	1.5	6.1	30	2200	0.3	1.3	1.5	0.7	0.9	1.1	7.7	66.2
Bo Dot Market	4.6	6.8	7.8	20	150	1100	3.2	16	15	26	34	19	9.1	50.5
Son Thuy														
Site 1	3.1	4.3	5.7	29	230	1800	0.4	2.1	4.1	19	44	36	7.6	40.8
Site 2	3.4	6.6	4.5	9.2	30	1100	NDR (0.1)	1	1.3	1.1	1.7	2.0	5.1	66.7
Hong Quang	7.9	8.9	2	5.4	4.3	67	0.4	1.1	1.9	1.5	0.9	0.6	8.3	95.2
Aluoi	2.1	3.1	1.9	8.5	4.1	28	0.2	1.3	0.7	0.4	0.4	0.5	2.7	77.8
Aluoi Market	15	21	9.5	11	19	770	0.8	7.9	4.0	0.8	0.4	0.6	17	88.2
Hong Kim	3.7	4.7	0.8	3.6	3.7	74	0.2	0.9	0.3	ND (0.2)	0.4	0.5	4.1	90.2
Hong Van														
Site 1	0.4	0.8	1.0	5.0	6.3	130	0.1	0.5	ND (0.1)	0.4	1.1	0.8	1.1	36.4
Site 2	0.3	1.0	ND (0.1)	1.4	3.5	64	0.1	0.9	0.3	0.2	0.4	0.5	0.6	50.0

[a] ND = Not detected; for 'total I-TEQ' calculations, if ND, half the detection level was used. Parentheses enclose the sample detection limit. NDR = A chromatographic peak was detected but did not meet quantification criteria; for total I-TEQ calculations, NDR is treated as ND.

At higher concentrations of >50 to <1000 pg/g total TEQ in the soil, the ATSDR (1997) guideline recommends that the area should be evaluated for bioavailability, ingestion rates, pathways, soil cover, climate, other contaminants, community concerns, demographics, and background exposures. For even higher soil levels of ⩾1000 pg/g total TEQ, ATSDR (1997) recommends stronger actions, such as surveillance, research, health studies, community education, and exposure investigations. Assessors should obtain enough information to judge whether the site is a public health hazard, which might lead to clean-up of the contaminated site.

Many of the soil samples near the A So Special Forces base area are between 50 and 1000 pg/g total TEQ. If these soils were in the USA, site-specific evaluations would be recommended (ATSDR, 1997). Given that our soil samples from the Aluoi Valley were composited from 10 subsamples, there is a high probability that values > 1000 pg/g exist, particularly on the A So base where the 901 pg/g total I-TEQ was measured. If so, that would trigger the more comprehensive assessment under ASTDR guidelines in the USA.

In BC, Canada, a site that exceeds 350 pg/g total TEQ is legally designated as contaminated for agricultural, urban park and residential purposes. Remediation must be implemented to return soils to <350 pg/g for residential and park use and to <10 pg/g for agricultural lands used for production of human food (BC Waste Management Act, 1996).

Many of the soils in the Aluoi Valley exceed PCDD and PCDF guidelines and standards set by the US EPA, ATSDR and BC and would require further assessment and/or remediation in those jurisdictions. However, western thresholds for PCDD and PCDF levels may not be conservative enough to protect human health in rural Viet Nam. The socio-economic circumstances in rural communes, dirt floors in many homes, children and many adults without footwear, their close association with the land for food production, and general sanitation are such that higher levels of exposure to contaminants occur. As a consequence, guidelines and standards should be more stringent in this environment. However, developing new guidelines and standards for application in developing countries where people are still living off the land, would be a long and likely controversial process. Given the limitations of implementing guidelines based on geographical considerations, those criteria existing in western jurisdictions should, at a minimum, be applied in regions like rural Viet Nam.

3.2. Fish pond sediment

In the A So commune, TCDD levels in bottom sediments of fish ponds ranged from 1.8 to 8.5 pg/g. The TCDD content was high, at 88% of the total I-TEQs (Table 1), indicating that Agent Orange was the source of contamination. Presence of TCDD would be expected since the ponds were excavated from contaminated soils, then filled with water to raise carp for food.

3.3. Vegetable matter

The main vegetable foods sampled were relatively low in contaminants. TCDD was not detected in any samples of rice and manioc (cassava) root from any location sampled. Rice from A So had a total heptachlorinated dibenzodioxin (H7CDD) of 0.3 pg/g, and a total O8CDD of 1.8 pg/g. Rice from Hong Van had ND concentrations of H7CDD and only 0.2 pg/g of the O8CDD congener. Manioc root was not sampled at A So, but at Hong Thoung and A Ngo it had ND levels of all PCDDs and PCDFs, and 1.1 pg/g of O8CDD (Fig. 2).

Contamination of crops by soil particles is an avenue for human contamination, particularly if vegetables are not washed or peeled before eating (Hulster and Marschner, 1993; Startin, 1994). Recent studies have demonstrated soil-to-plant transfer of PCDDs and PCDFs in zucchini and pumpkins (Hulster et al., 1994; Neumann et al., 1999). From those findings, it is apparent that there are exceptions to the generalization that plants take up virtually no PCDDs and PCDFs through their systemic system (IARC, 1997). Lack of transfer might be expected because PCDDs and PCDFs are extremely hydrophobic and possess a high affinity to the soil organic fraction, which would limit their mobility in soil and translocation to plants. It has been stated that atmospheric deposition is the principle pathway for entrance of PCDDs and PCDFs into the human food chain through agricultural crops (McLachlan, 1999). In any case, these contaminants were low or ND in the vegetable crops sampled in the Aluoi Valley.

Vegetable cooking oil from Aluoi commune had some chlorinated contaminants, but at low concentrations and were inconsequential in terms of overall toxicity. The oil had no TCDD, but did have low levels of hexachlorinated dibenzodioxin (H6CDD, 1.0 pg/g), H7CDD (5.2 pg/g), O8CDD (19.0 pg/g), heptachlorinated dibenzofuran (H7CDF, 1.3 pg/g) and O8CDF (2.0 pg/g). IARC (1997) reports results for cooking oils which were similar to those in the Aluoi Valley (i.e., primarily hepta- and octachlorinated congeners).

3.4. Animal tissues

PCDD and PCDF analyses in the Aluoi Valley indicated that fish and duck fat contained the highest levels of TCDD of all animal tissues tested (Fig. 2). Extremely high levels were found in fatty tissue in ducks

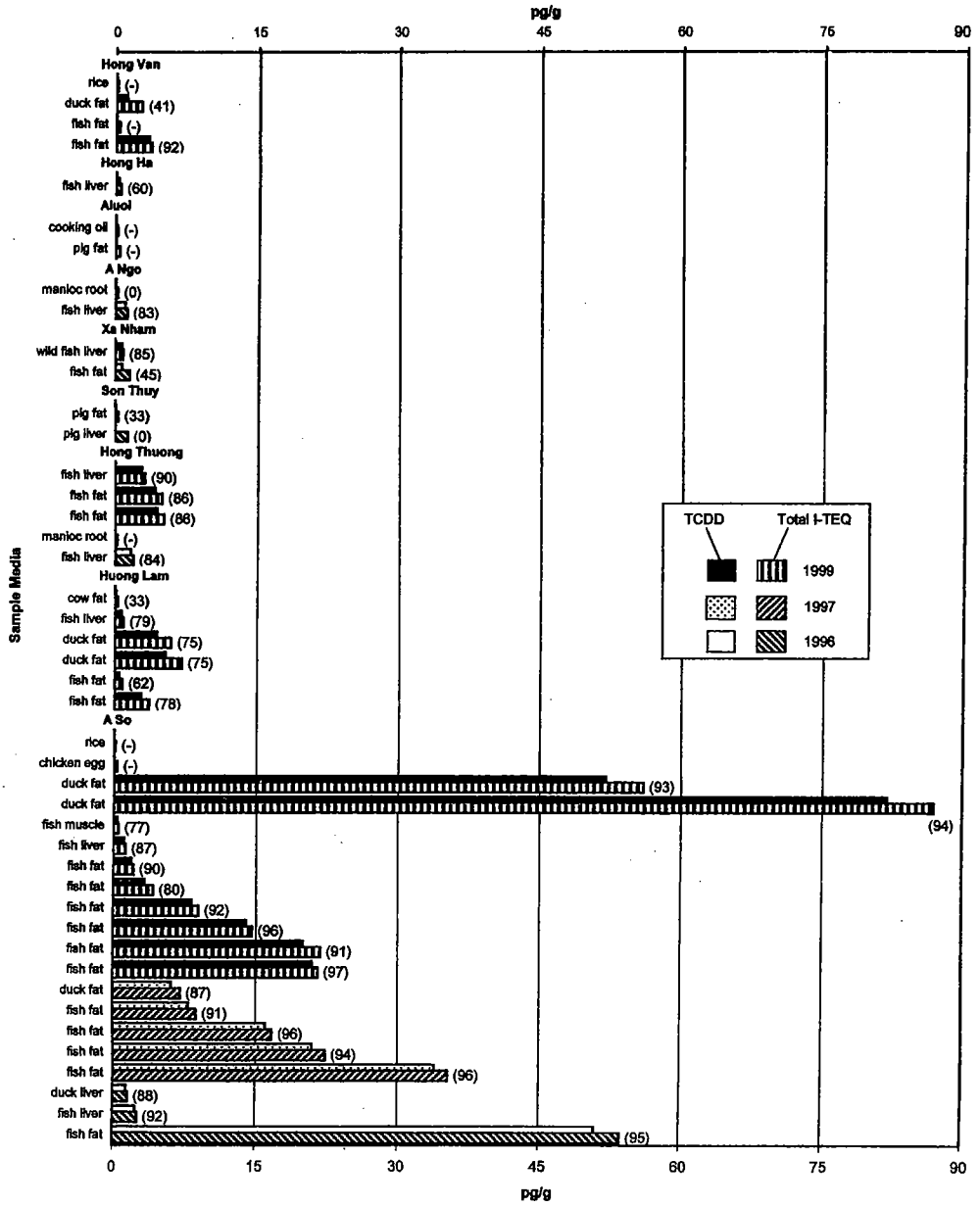

Fig. 2. TCDD and total I-TEQ concentrations in vegetable matter (pg/g dry weight) and animal tissues (pg/g wet weight) collected from communes in the Aluoi Valley, Viet Nam, 1996, 1997 and 1999. Parenthesis enclose percent contribution of TCDD to total I-TEQ.

and fish (carp) raised on the A So commune at the former US Special Forces base.

In 1996, 51 pg/g (wet weight) of TCDD was detected in fat of cultured carp from a pond on the former A So base (based on wet weight for all analyses of animals tissues). In 1997, 34 pg/g TCDD was recorded in carp fat from a pond on this base; in 1999, 21 pg/g TCDD in carp fat and 82 pg/g in duck fat from A So animals was measured. A second duck fat sample yielded 52 pg/g TCDD in 1999 (Fig. 2).

Other communes in the valley had noticeably lower levels of TCDD in the animal foods tested. Huong Lam commune, adjoining A So, had the highest TCDD among other communes in the valley, with duck fat

measuring 5.3 pg/g. With increasing distance toward Hong Van in the northern sector of the valley, levels of TCDD declined. Even in Hong Van, the least sprayed area, duck fat and fish fat contained TCDD (1.1 and 3.4 pg/g, respectively), but at much reduced levels (Fig. 2).

The contribution of TCDD to total I-TEQs in A So foods ranged from 77% to 97%, indicative of Agent Orange as a source. Similar high percent contributions of TCDD to total I-TEQs were recorded throughout the valley.

It is known that animal food can play a significant role in the transfer of PCDDs and PCDFs into humans (Pohl et al., 1995). Fish, poultry, beef and pork may become contaminated, if they are exposed to contaminated lands. Fish ponds on the former A So base were excavated in TCDD-contaminated soils, and the contaminant was, therefore, present in pond sediments (Table 2). Through bioaccumulation and biomagnification, TCDD migrated and was retained in cultured fish and in ducks that frequented these ponds. When feeding, ducks filtered sediments or suspended particles in these ponds, ingesting TCDD-contaminated soil particles. People in the Aluoi Valley consume animal fats regularly; fat from fish and ducks is a direct conduit for TCDD transfer to humans.

Constituents of the human food chain are being exposed to TCDD up to the present day in the Aluoi Valley, particularly near the former A So Special Forces base. Over 29 years have passed since the cessation of spraying, and TCDD continues to accumulate in animal tissues used as human food. There is potential for a particularly high level of TCDD exposure for humans living near A So.

3.5. Findings by others and consumption guidelines

Data for foodstuffs collected throughout Viet Nam in the mid-1980s were summarized by Schecter et al. (1989a, 1990c), Quynh et al. (1994) and Cau et al. (1994). The highest levels of TCDD reported from southern Viet Nam in these papers were for turtle ovaries (250 pg/g), turtle liver (88.0 pg/g), turtle gall bladder (39.0 pg/g) and snake (11.58 pg/g). Other TCDD values reported in these investigations were relatively low; many of them were lower than values determined in foodstuffs from the Aluoi Valley during the present investigation.

Dai et al. (1994b) and Cau et al. (1994) reported that by 1998, dioxins had decreased significantly in southern Viet Nam and levels in foodstuffs were considered comparable to those for other nations. However, both authors offered the generalization that residual dioxin contamination nonetheless remained a threat to human health in Viet Nam. Our data do not confirm low levels in some important foods, but rather, indicate potentially elevated levels of TCDD for areas of localized soil contamination.

The toxicity of TCDD specifically, and PCDDs and PCDFs in general, has prompted organizations such as the WHO and various countries to develop and adopt tolerable daily intakes (TDI) for PCDDs and PCDFs in foods, based on I-TEQ of TCDD.

The Canadian and the Japanese governments are presently applying a TDI of 10 pg TEQ per kg body weight per day (10 pg TEQ/kg bw/d) (Government of Canada, 1993; Health Canada, 1996; IARC, 1997). This value was originally recommended by the WHO (WHO/EURO, 1991) based on liver toxicity, reproductive effects, immunological effects, and on information on kinetics in humans and experimental animals. In Germany, the same limit is being used; if daily intake is higher for an extended period of time, actions are necessary to counter exposure (Schulz, 1994).

In Canada, if the upper threshold of 10 pg TEQ/kg bw/d is exceeded on a regular basis, a risk assessment/risk management process is triggered. An individual consumer is assumed to have a standard weight of 60 kg. The probable daily intake (PDI) of animal liver or fat tissues is set at 20 g tissue/day. Accordingly, a given sample that showed 30 pg/g as total I-TEQ of PCDDs and PCDFs would achieve the TDI of 10 pg TEQ/kg bw/d, without allowing for any other sources. A similar calculation for muscle tissue, at a set consumption rate of 40 g/day, means that a concentration of 15 pg/g total I-TEQ in the muscle would yield the Canadian TDI. A risk assessment/risk management process would be activated in Canada, if either the 30 pg/g total I-TEQ in liver or fat, or 15 pg/g total I-TEQ in muscle tissue were exceeded in a tissue sample.

The Netherlands uses a more restrictive TDI of 1–3 pg TEQ/kg bw/d (Birnbaum and Slezak, 1999; Patandin et al., 1999a). The US EPA has proposed a virtually safe dose of 0.0064 pg TEQ/kg bw/d (McLachlan, 1993; Patandin et al., 1999a).

The WHO has recently revised the recommended TDI, reducing the value from 10 pg TEQ/kg bw/d to a range of 1–4 pg TEQ/kg bw/d (WHO/EURO, 1998a,b) based on new epidemiological and toxicological data, particularly information focussing on neurodevelopment and endocrinological effects. The WHO has stressed that the upper value of the range (4 pg TEQ/kg bw/d) should be considered the maximum TDI, and that "the ultimate goal is to reduce human intake levels below 1 pg TEQ/kg bw/d". These new levels recommended by WHO are considerably lower than the TDIs currently in use by several countries, are somewhat comparable to the Netherlands limits, but are not nearly as low as those of the US EPA.

Table 3
Age of donors in whole human blood analyses for PCDDs and PCDFs, Aluoi Valley, Viet Nam, 1999

Commune	Males		Females	
	⩾ 25 yearrs	<25 years	⩾ 25 years	<25 years
A So				
Median	50	20	35	20
Mean ± SD	48 ± 16	20 ± 3.1	40 ± 13	20 ± 2.7
Range	25–79	16–24	25–68	15–24
n	48	30	44	41
Huong Lam				
Median	29	21	28	22
Mean ± SD	32 ± 8.6	21 ± 2.4	30 ± 6.5	22 ± 1.9
Range	25–60	15–24	25–49	17–24
n	31	33	29	27
Hong Thuong				
Median	48	19	40	22
Mean ± SD	48 ± 14	20 ± 2.7	40 ± 11	22 ± 2.0
Range	25–78	15–24	25–75	17–24
n	43	27	37	25
Hong Van				
Median	55	21	43	20
Mean ± SD	50 ± 14	21 ± 2.3	42 ± 12	20 ± 2.6
Range	25–70	16–24	25–60	15–24
n	37	40	27	37

3.6. Whole human blood

The 556 individuals sampled for blood PCDD/PCDF were reasonably well balanced among the four locations and four categories of age and sex, with 25 individuals representing the smallest category and 48 representing the largest (Table 3).

Results confirmed that humans, at the top of the food chain, can bioaccumulate and biomagnify chemical contaminants. The highest TCDD levels in blood, 41 and 31 pg/g lipid, were recorded at the A So commune near the former military base (Table 4), with males being highest for both age categories. The lowest levels were found at Hong Van, the northernmost location; values were either ND or did not meet quantification criteria (NDR).

There was a statistically significant difference among TCDD levels from the four communes (ANOVA; [1] $F[3,7] = 37.9$, $p = 0.0001$), between levels recorded for the sexes ($F[1,7] = 27.4$, $p = 0.001$), and between levels recorded for age categories ($F[1,7] = 9.3$, $p = 0.019$). A statistically significant interaction existed between sex and commune location ($F[3,7] = 7.6$, $p = 0.013$).

Whole human blood from A So had significantly higher TCDD concentrations than blood from Hong Van, Huong Lam and Hong Thuong, with the most significant difference between A So and Hong Van ($p < 0.01$; Student–Newman–Keuls Test; Hicks, 1973). Males had higher concentrations of TCDD than females ($p = 0.001$). This sex difference is likely related to the higher caloric intake of males, their greater exposure to soil, and a practice of living off the land away from the home, for days. In addition, breast feeding by females is an avenue for the elimination of TCDD, thereby reducing their overall body burdens (Schecter et al., 1990a; Abraham et al., 1998; Schecter et al., 1998).

Older people had higher concentrations of TCDD in their blood ($p = 0.019$), presumably because they were exposed for a longer time. However, people <25 years of age (i.e., born after the war) also had TCDD levels that were clearly elevated. There was relatively little difference in TCDD levels between the two age categories for females, particularly in A So (16 pg/g vs. 14 pg/g; Table 4). This lack of difference may be attributed to younger women becoming lactating mothers at an early age, while older women are continuing to bear children and off-loading TCDD during breast feeding.

The significant interaction between sex and commune location ($p = 0.013$) reflected the major differences among males compared to females. Concentrations of TCDD in males generally decreased successively from A So to Hong Thuong to Huong Lam to Hong Van. The potential for increased exposure to contaminated soils and/or food in A So would easily account for the higher human levels at A So.

[1] Where ND or NDR appears in Table 4, half the detection level was used in the ANOVA.

Table 4
PCDDs and PCDFs in pooled whole human blood, Aluoi Valley, Viet Nam, 1999

Commune and donor (years of age)	# in pool	PCDD (pg/g lipid)[a]						PCDF (pg/g lipid)[a]						Total I-TEQ	TCDD as % of total I-TEQ
		TCDD	Total T4CDD	Total P5CDD	Total H6CDD	Total H7CDD	Total O8CDD	2,3,7,8-TCDF	Total T4CDF	Total P5CDF	Total H6CDF	Total H7CDF	Total O8CDF		
A So															
Males (⩾ 25)	48	41	41	ND (2.2)	ND (2.6)	ND (2.6)	72	ND (3.0)	ND (3.0)	ND (2.2)	29	ND (4.3)	7.4	45.9	89.3
Males (<25)	30	31	31	ND (1.7)	ND (1.7)	18	49	ND (2.4)	ND (2.4)	ND (1.7)	25	ND (2.1)	NDR (3.8)	35.0	88.6
Females (⩾ 25)	44	16	16	ND (1.7)	ND (1.7)	ND (1.7)	42	ND (3.3)	ND (3.3)	ND (1.7)	ND (2.3)	13	ND (3.3)	18.3	87.4
Females (<25)	41	14	14	ND (1.8)	2.1	28	86	ND (1.8)	ND (1.8)	5.0	16	23	7.5	16.6	84.3
Huong Lam															
Males (⩾ 25)	31	17	17	ND (2.2)	7.0	36	120	ND (2.2)	ND	ND	55	65	6.5	25.6	66.4
Males (<25)	33	9.0	9.0	3.9	13	21	74	ND (1.6)	ND (1.6)	5.5	36	47	6.5	19.8	45.5
Females (⩾ 25)	29	5.3	5.3	2.9	25	49	130	ND (2.9)	ND (2.9)	24	89	130	NDR (2.6)	22.0	24.1
Females (<25)	27	ND (9.2)	ND (9.2)	ND (2.1)	3.8	ND (7.5)	110	ND (6.7)	ND (6.7)	ND (2.1)	23	ND (11.0)	ND (16.0)	10.0	–
Hong Thuong															
Males (⩾ 25)	43	21	21	5.6	15	24	120	ND (1.9)	ND (1.9)	ND (1.9)	49	53	3.7	32.3	65.0
Males (<25)	27	8.6	8.6	ND (1.7)	10	44	180	ND (1.7)	ND (1.7)	3.4	26	32	ND (5.9)	15.1	57.0
Females (⩾ 25)	37	12	12	4.0	14	29	170	ND (2.0)	ND (2.0)	5.2	50	77	NDR (4.4)	24.6	48.8
Females (<25)	25	7.6	7.6	ND (2.8)	5.2	26.0	87	NDR (3.4)	ND (3.4)	ND (2.4)	10	21	4.1	11.5	66.1
Hong Van															
Males (⩾ 25)	37	ND (4.0)	ND (4.0)	ND (1.2)	4.7	11	83	ND (1.9)	ND (1.9)	ND (1.2)	16	25	2.6	5.41	–
Males (<25)	40	NDR (3.5)	ND (3.5)	1.6	ND (2.7)	10	64	ND (2.3)	ND (2.3)	ND (1.9)	20	32	NDR (3.5)	7.67	–
Females (⩾ 25)	27	ND (4.3)	ND (4.3)	ND (1.8)	ND (3.2)	15	120	ND (4.6)	ND (4.6)	ND (1.8)	10	23	ND (5.4)	5.95	–
Females (<25)	37	ND (1.9)	ND (1.9)	ND (1.6)	ND (1.6)	ND (1.9)	82	ND (2.6)	ND (2.6)	ND (1.6)	7.7	ND (4.2)	3.2	3.53	–

[a] ND = Not detected; for 'total I-TEQ' calculations, if ND, half the detection level was used. Parentheses enclose the sample detection limit. NDR = A chromatographic peak was detected but did not meet quantification criteria; for total I-TEQ calculations, NDR was treated as ND.

TCDD congeners contributed 84.3–89.3% of the total I-TEQs in the blood samples from A So. In the other communes the maximum contribution of TCDD was 66.4%. The higher contribution of TCDD at A So can be related to the preponderance of TCDD in the soils and foods at that commune.

In work by others, pooled blood from Viet Nam south of the former demilitarized zone, showed TCDD levels ranging from 1.0 pg/g lipid (Tay Ninh Province) to 33 pg/g lipid (Can Tho Province), as reported in reviews by Dai et al. (1994a, 1995), Schecter et al. (1992a), and Schecter (1994b). The blood levels of TCDD reported for northern Viet Nam were <2.4 pg/g lipid (Ha Noi), and 2.9 pg/g lipid (Thanh Hoa Province). Northern Viet Nam was not sprayed with Agent Orange. The levels of TCDD that we recorded in blood from people in the Aluoi Valley, particularly in the A So commune, were comparable to the highest reported in the reviews. A very high level of 271 pg/g lipid was reported for TCDD in blood by Schecter et al. (2001). This was for an individual living near the former US airbase at Bien Hoa, where a major spill of 7500 US gallons of Agent Orange had been reported for March 1970.

3.7. Human breast milk

Breast milk from the A So commune had the highest individual TCDD level (19 pg/g lipid; total I-TEQ = 21.9 pg/g lipid; Table 5). Mean TCDD level was 14.6 pg/g, the highest of any of the communes. TCDD contributed from 85.1% to 96.3% of total I-TEQ in A So, a further indication that Agent Orange was the principle source of contamination. The lowest levels of TCDD and total I-TEQ were recorded for breast milk from the reference area, Hong Van.

Statistically significant differences did exist among breast milk TCCD from the four communes ($F[3,12] = 7.31$, $p = 0.005$). However, the differences were limited to the reference area (Hong Van) being different from each of the other areas (Hong Van vs. A So, $p < 0.01$, vs. Hong Thuong, $p < 0.05$, and vs. Huong Lam, $p < 0.05$; Student–Newman–Keuls Test; Hicks, 1973).

The lack of differences among milk TCDDs from some areas might imply that environments which are not necessarily exceptionally high in contamination of soils and food may still show elevated milk levels. This might result from bioaccumulation or more specifically, from biomagnification. Some leveling of differences might have resulted from food items that originated in one area of the valley being transferred and marketed in another valley commune.

An alternative explanation could be based on the generally accepted concept that hydrophobic contaminants like TCDD can be eliminated from the body through lactation (Abraham et al., 1998; Raum et al., 1998). That could account for the lack of differences between some of the sites. That alternative explanation seems likely, but does not fit at all with the observed levels in milk of the single multiparous woman from A So, who had 10 children (data not shown in Table 5). Her breast milk had a TCDD level of 32 pg/g, the highest value measured in the survey. This woman nursed each of her children for up to 2 years, and had been nursing for 2 years prior to donating milk for testing. Her body levels of dioxins might have been expected to be low because of elimination through the milk, but that was not the case. Total I-TEQ was 34.1 pg/g, and TCDD contributed 93.8% of the total I-TEQ, a strong indicator of Agent Orange involvement. TCDD could remain high over time if continual replacement occurred through ingestion of contaminated foods, and/or she was experiencing some form of unusual exposure to a highly contaminated site. This woman gave birth to a child with spina bifida who was two years old at the time of milk sampling. We have no other clinical information on the child's condition other than the primary diagnosis. The father's TCDD blood level is also an unknown.

Work by others in southern Viet Nam where Agent Orange was used, showed that TCDD levels in human breast milk have generally declined from the early 1970s through the 1980s, according to reviews of historical data published by Schecter (1998) and Schecter et al. (2001). There appeared to be decreases in milk TCDD over three sampling periods: 1970, 333–1850 pg/g lipid; 1973, a range of 77–400 pg/g lipid; and 1985–1988, 5.2 and 11 pg/g lipid. Our measurements of TCDD in breast milk collected in 1999 are comparable to the levels reported in the mid to late 1980s. Apparently levels have been relatively stable over the last two decades, rather than declining further. Levels in northern Viet Nam were lower, with data for pooled human milk from Ha Noi in 1988 having a TCDD concentration of 2.1 pg/g lipid (Schecter, 1998; Schecter et al., 2001).

Breast-fed infants are considered a high risk group for PCDD and PCDF exposure (Jensen, 1987; Dahl et al., 1995). There is concern for the health of infants, particularly if breast milk comprises a high proportion of the infant diet (Raum et al., 1998), as is the case in the Aluoi Valley. Throughout the valley, breast-fed infants of primaparous females have an average daily intake (Table 6), up to 11 times the TDI used in Canada and up to 27 times the revised WHO maximum TDI (10 and 4 pg TEQ/kg bw/d, respectively). In fact, breast milk in all Aluoi Valley samples exceeded recognized guidelines of all jurisdictions. Milk from the multiparous female at A So had a total I-TEQ of 34.1 pg/g lipid, translating to an average daily intake by an infant of 167 pg TEQ/kg bw/d. That exceeds the Canadian guideline by 17 times, and the revised WHO guideline by 42 times.

Table 5
PCDDs and PCDFs in human breast milk from lactating primaparous females, Aluoi Valley, Viet Nam, 1999

Commune	Donor age (years)	% lipid	PCDD (pg/g lipid)[a]						PCDF (pg/g lipid)[a]						Total I-TEQ	TCDD as % of total I-TEQ
			TCDD	Total T4CDD	Total P5CDD	Total H6CDD	Total H7CDD	Total O8CDD	2,3,7,8-TCDF	Total T4CDF	Total P5CDF	Total H6CDF	Total H7CDF	Total O8CDF		
A So	22	5.6	5.5	5.5	ND (0.2)	1.3	0.2	1.8	ND (0.2)	NDR (0.2)	0.7	1.1	2.3	NDR (0.2)	6.15	89.4
	20	4.5	19.0	19.0	3.1	ND (0.89)	ND (0.89)	4.7	ND (0.67)	ND (0.67)	1.9	3.0	ND (1.1)	ND (1.3)	21.9	86.8
	18	4.0	18	18	1.5	1.8	ND (0.3)	15	ND (0.3)	NDR (0.3)	ND (0.3)	ND (0.3)	5.2	NDR (0.3)	18.7	96.3
	23	3.3	16	16	1.8	3.0	0.6	7.9	ND (0.3)	ND (0.3)	ND (0.3)	11	5.0	NDR (0.3)	18.8	85.1
Huong Lam	23	1.3	12	12	ND (0.8)	ND (0.8)	1.8	16	ND (0.8)	ND (0.8)	3.1	6.9	5.5	NDR (0.8)	14.6	82.2
	19	3.7	8.3	8.3	ND (0.3)	ND (0.3)	8.9	51	NDR (0.3)	ND (0.3)	1.9	5.4	3.8	NDR (0.3)	10.2	81.4
	28	3.6	2.9	2.9	2.7	7.7	9.2	15	1.1	1.1	8.3	23	17	4.9	10.6	27.4
	21	1.7	5.8	5.8	1.5	5.8	4.5	13	ND (0.6)	NDR (0.6)	3.2	6.3	6.0	ND (0.6)	9.33	62.2
Hong Thuong	17	1.6	11	11	2.5	7.8	11	31	1.1	1.5	5.3	25	12	2.7	17.2	64.0
	21	1.4	8.7	8.7	ND (0.35)	8.7	9.5	32	1.0	1.0	4.6	10	6.4	4.8	12.6	69.0
	22	2.7	7.7	7.7	1.6	5.4	3.5	18	0.6	0.6	0.6	5.7	3.7	NDR (0.34)	9.73	79.1
	19	2.1	11	11	2.7	7.8	7.2	30	0.6	0.6	6.1	23	13	NDR (0.24)	18.5	59.5
Hong Van	20	2.7	3.3	3.3	ND (0.4)	1.8	3.0	13	ND (0.4)	NDR (0.4)	1.8	4.8	2.4	NDR (0.4)	5.07	65.1
	23	2.1	2.2	2.2	1.2	3.0	0.5	12	ND (0.5)	ND (0.5)	ND (0.5)	4.7	3.4	NDR (0.5)	3.85	57.1
	20	3.2	5.0	5.0	4.1	14	14	43	ND (0.3)	NDR (0.3)	4.6	25	12	ND (0.3)	13.2	37.9
	19	1.8	1.4	1.4	1.2	2.0	1.9	9.4	ND (0.6)	NDR (0.6)	ND (0.6)	4.4	ND (0.6)	NDR (0.6)	2.99	46.8

[a] ND = Not detected; for 'total I-TEQ' calculations, if ND, half the detection level was used. Parentheses enclose sample detection limit. NDR = A chromatographic peak was detected but did not meet quantification criteria; for total I-TEQ calculations, NDR was treated as ND.

Table 6
TCDD in human breast milk (pg/g lipid) and average daily intake (pg TEQ/kg bw/d) of PCDDs and PCDFs by infants from primarou females, Aluoi Valley, Viet Nam, 1999

Commune	Age of child and duration of breast feeding[a]	% lipid	TCDD (pg/g)	Total I-TEQ (pg/g)	TCDD as % of total I-TEQ	Intake[b]	
						TCDD	Total I-TEQ
A So	1 y	5.6	5.5	6.15	89.4	27.0	30.1
	1 m	4.5	19.0	21.9	86.4	93.1	107.3
	1 y	4.0	18	18.7	96.3	88.2	91.6
	5 m	3.3	16	18.8	85.1	78.4	92.1
Huong Lam	7 m	1.3	12	14.6	82.2	58.8	71.5
	8 m	3.7	8.3	10.2	81.4	40.7	50.0
	3 m	3.6	2.9	10.6	27.4	14.2	51.9
	20 d	1.7	5.8	9.33	62.2	28.4	45.7
Hong Thuong	5 m	1.6	11	17.2	64.0	53.9	84.3
	6 m	1.4	8.7	12.6	69.0	42.6	61.7
	1 y	2.7	7.7	9.73	79.1	37.7	47.7
	2 m	2.1	11	18.5	59.5	53.9	90.7
Hong Van	5 m	2.7	3.3	5.07	65.1	16.2	24.8
	2 m	2.1	2.2	3.85	57.1	10.8	18.9
	1 m	3.2	5.0	13.2	37.9	24.5	64.7
	1 m	1.8	1.4	3.0	46.8	6.9	14.7

[a] y = year, m = months, d = days.
[b] Average daily intake via human milk based on a 5 kg infant consuming 700 ml of milk per day with a lipid content of 3.5% (WHO/EURO, 1989). Average daily intake = (volume of milk per day in ml) × (% lipid in milk/100) × (concentration of chemical in pg/g)/(infant weight in kg).

This potential health danger from breast feeding in southern Viet Nam is in conflict with the widespread recognition of the benefits to infants (WHO/EURO, 1988; Huisman et al., 1995; Hooper et al., 1998). These benefits include passing immunological factors to the infant, creating a bonding between mother and infant, reducing the risk of allergic reactions, providing virtually all the nutrition necessary during earlier months, and serving as a contraceptive. Accordingly, there are recommendations by numerous researchers that breast feeding be continued, because the benefits appear to outweigh the health risks at present (Tarkowski and Yrjanheikki, 1989; Rogan et al., 1991; Huisman et al., 1995; Albers et al., 1996; Rogan, 1996; Wise, 1997; Schade and Heinzow, 1998; Lutter et al., 1998; Patandin et al., 1999b). Nevertheless, concerted efforts should attempt to reduce health risks from toxic chemicals, mainly by controlling the source(s) of contaminants (Somogyi and Beck, 1993; Abraham et al., 1996; Brouwer et al., 1998; Schuhmacher et al., 1999).

Cessation of breast feeding is not a realistic recommendation for the Aluoi Valley because of economic and cultural conditions. Given that the sources of TCDD which feed into the human food chain have been identified, there is a very strong case for attempting to reduce exposure, in order to decrease the body burdens of contaminants in both adults and infants.

4. Concluding discussion

More than 35 years have elapsed since Agent Orange was introduced to the isolated Aluoi Valley, and the indigenous hill tribes inhabiting the region. Aerial applications throughout the valley, together with other forms of administering and handling the herbicide near former US military bases, resulted in chemical contamination that affects the valley to the present day; this is not an historical problem.

The highest TCDD contamination was measured on the former US Special Forces military base at A So. Other former bases in the valley had lower levels of TCDD in soils, but still generally higher than in regions which had received aerial spraying. Clearly, even those military installations that operated for a short time had appreciable problems of TCDD-contaminated soils.

In the region of highest TCDD contamination, at A So, our results fit the explanation that this congener has been transported from soil to fish pond sediment to cultured fish and duck tissues, and ultimately into humans as shown by measurements in blood and breast milk. The high levels of TCDD contributed almost all of the total toxicity of samples, confirming that Agent Orange was the principal source of this dioxin congener.

Numerous military installations were established throughout southern Viet Nam during the conflict. We

theorize that other former military installations throughout the southern regions are potential reservoirs for TCDD. We suspect that the pattern of TCDD data reported for the microcosm of the Aluoi Valley mirrors conditions on a countrywide basis, south of the former demilitarized zone. Recent data from Schecter et al. (2001) reporting high levels of TCDD in the blood of residents near the former US base at Bien Hoa further strengthen our theory.

The Bien Hoa base had a spill of Agent Orange in 1970. Other declassified documents (US Army documents, 1967) outlined actions for disposal of herbicide waste on the base. Herbicide spillage and water used to flush out herbicide tanks on aircraft could be directed to the small creek draining Bien Hoa base that feeds into the Dong Nai River. Other recommendations were to excavate spill ponds or ditches to allow gradual seepage, or create cisterns and use these as septic tanks comparable to sewage disposal systems. It was also recommended that efforts should be devoted to decreasing the amount of herbicide spillage. The US Army actions specified in the memorandum would not have destroyed the contaminants in herbicide, but would have caused them to enter the local soil, groundwater, or the wider downstream ecosystems. Similar plans probably existed at other military installations in southern Viet Nam which used Agent Orange and other herbicides. We have unconfirmed information that other former military installations in southern Viet Nam, in addition to Bien Hoa, have similar dioxin problems.

A systematic review should be undertaken of any military installation in southern Viet Nam during the conflict, where Agent Orange was used on site. TCDD measurements should be made in these areas if evidence suggests contamination. Soils, food chain elements and the human population should be assessed during such investigations. We suspect many fire bases, air bases and other former US and south Vietnamese military facilities have significant levels of TCDD in their subsurface soils. Crash sites and load-jettison sites of Agent Orange spray planes should also be investigated.

TCDD could be remobilized from soil by agricultural activities, detonation of UXO and land mines (during clearing), natural erosion forces, road construction and other infrastructural improvements. These soil disturbances could initiate transfer of TCDD into the natural communities and the human food chain, increasing the risk to human health. Impact mitigation plans should be formulated in areas where dioxin contamination exceeds western guidelines for protection of human health.

The Vietnamese Government has intervened in order to protect its citizens from TCDD contamination, contrary to Gochfeld (2001). We have submitted recommendations in an impact mitigation plan to government health departments addressing TCDD contamination in the Aluoi Valley. Our mitigation plan has prompted Vietnamese authorities to relocate 15 families from the former US Special Forces base away from the contaminated site at A So. Aquaculture ponds on the A So base have been deactivated, and no further excavation of ponds is being permitted on the former base. The 10-80 Committee and the Thua Thien Hue Provincial Health Department have provided educational material to all valley residents regarding the handling of fish and ducks, in terms of tissues that are safe to eat and those that should be discarded. Proper cleaning and peeling of in-ground vegetables, and improved general health practices have also been recommended to valley inhabitants to avoid or reduce TCDD exposure.

Similar mitigation strategies, appropriate to Vietnamese lifestyle and culture, should be implemented in other areas of the country where dioxin contamination is confirmed, and is deemed a risk to human health.

Acknowledgements

The project team gratefully acknowledges financial contributions for this study from the Canadian International Development Agency (CIDA), Environment Canada, the Canadian Space Agency, and Revenue Canada. In-kind support from the Forest Inventory and Planning Institute, Ha Noi, and Thua Thien Hue Peoples' Committee and Department of Health is acknowledged. We are most grateful for the assistance of many other Vietnamese, too numerous to mention here; but including the Thua Thien Hue Department of Planning and Investment, Ministry of Science Technology and Environment, Aluoi District Peoples' Committee, local Peoples' Committees in the communes of Aluoi District, local police, military personnel and citizens of the Aluoi Valley. They provided information, logistical support, permits and gracious hospitality during this investigation. We thank all the Aluoi Valley donors of food items, blood and milk samples.

We are particularly grateful for the assistance of Mr. Gerd Willkommen from the German Gerbera Demining Team, Viet Nam. Mr. Willkommen worked with our field crews during soil sampling to sweep areas for UXO. We also wish to thank Dr. John B. Sprague for his editorial comments.

References

Abraham, K., Knoll, A., Ende, M., Papke, O., Helge, H., 1996. Intake, fecal excretion, and body burden of polychlorinated dibenzo-*p*-dioxins and dibenzofurans in breast-fed and formula-fed infants. Pediatric Research 40, 671.

Abraham, K., Papke, O., Gross, A., Kordonouri, O., Wiegand, S., Wahn, U., Helge, H., 1998. Time course of PCDD/

PCDF/PCB concentrations in breast-feeding mothers and their infants. Chemosphere 37, 1731.

Albers, J.M.C., Kreis, I.A., Liem, A.K.D., van Zoonen, P., 1996. Factors that influence the level of contamination of human milk with poly-chlorinated organic compounds. Archives of Environmental Contamination and Toxicology 30, 285.

ATSDR (Agency for Toxic Substances and Disease Registry), 1997. Interim Policy Guideline: Dioxin and dioxin-like compounds in soil. US Department of Health and Human Services, Public Health Service. Atlanta, GA, p. 10 (with appendices).

ATSDR (Agency for Toxic Substances and Disease Registry), 1998. Toxicological profile for chlorinated dibenzo-*p*-dioxins (update). US Department of Health and Human Services, Public Health Service. Atlanta, GA, p. 678 (with appendices).

BC Waste Management Act, 1996. Waste Management Act—Contaminated Sites Legislation. BC Reg. 375/96. Province of BC, Canada, p. 103.

Bengtsson, B., 1976. Ecological effects of chemical warfare and bombing in Viet Nam. A review and possible research priorities. SAREC Report No. 3, p. 23.

Birnbaum, L.S., Slezak, B.P., 1999. Dietary exposure to PCBs and dioxins in children: Research highlights. Environmental Health Perspectives 107, 1.

Boffey, P.M., 1971. Herbicides in Viet Nam: AAAS study finds widespread devastation. Science 171, 43.

Brouwer, A., Ahlborg, U.G., van Leeuwen, F.X.R., Feeley, M.M., 1998. Report of the WHO working group on the assessment of health risks for human infants from exposure to PCDDs, PCDFs and PCBs. Chemosphere 37, 1627.

Brown, J.S., 1999. Personal communication. The US Army Centre of Military History. Fort Lesley J. McNair, DC.

Cau, H.D., Dai, L.C., Hanh, L.H., Thuy, L.B., Quynh, H.Y., Hein, N.M., Thom, L.H., Schecter, A., Constable, J., et al., 1994. Report on the levels of PCDD, PCB and other chloro-organic compounds in foodstuffs in Viet Nam. In: Cau, H.D., Dai, L.C., Minh, D.Q., Thuy, L.B. (Eds.), Herbicides in War—The Long-term Effects on Man and Nature. 2nd International Symposium, Ha Noi, 1993. Ha Noi: 10-80 Committee. Hanoi Medical School, pp. 25–39.

Cecil, P.F., 1986. Herbicidal Warfare: the RANCH HAND Project in Viet Nam. Praeger, New York, p. 290.

Courtney, K.D., Gaylor, D.W., Hogan, M.D., Falk, H.L., Bates, R.R., Mitchell, J., 1970. Teratogenic evaluation of 2,4,5-T. Science 168, 864.

Dahl, P., Lindstrom, G., Wiberg, K., Rappe, C., 1995. Absorption of polychlorinated biphenyls, dibenzo-*p*-dioxins and dibenzofurans by breast-fed infants. Chemosphere 30, 2297.

Dai, L.C., Minh, D.Q., Quynh, H.T., Thom, L.H., Thuy, L.B., 1994a. Remarks on the dioxin levels in human pooled blood from various localities of Viet Nam. In: Cau, H.D., Dai, L.C., Minh, D.Q., Thuy, L.B. (Eds.), Herbicides in War—The Long-term Effects on Man and Nature. 2nd International Symposium, Ha Noi, 1993. Ha Noi: 10-80 Committee. Hanoi Medical School, pp. 5–15.

Dai, L.C., Hanh, L.H., Giay, T., Hue, N.D., Thuy, L.B., 1994b. An attempt to calculate the daily dioxin intake through food in Viet Nam. In: Cau, H.D., Dai, L.C., Minh, D.Q., Thuy, L.B. (Eds.), Herbicides in War—The Long-term Effects on Man and Nature. 2nd International Symposium, Ha Noi, 1993. Ha Noi: 10-80 Committee. Hanoi Medical School, pp. 40–46.

Dai, L.C., Thuy, L.B., Minh, D.Q., Quynh, H.T., Thom, L.H., 1995. Remarks on the dioxin levels in human pooled blood from various localities in Viet Nam. Organohalogen Compounds 26, 161.

Dwyer, J.H., Flesch-Janys, D., 1995. Editorial: Agent Orange in Viet Nam. American Journal of Public Health 85, 476.

Gochfeld, M.D., 2001. Dioxin in Viet Nam—the ongoing saga of exposure. Journal of Occupational and Environmental Medicine 43, 433.

Gough, M., 1986. Dioxin, Agent Orange—The Facts. Plenum Press, New York, p. 298.

Government of Canada, 1993. Polychlorinated dibenzodioxins and polychlorinated dibenzofurans. Canadian Environmental Protection Act. Priority Substances List. (Assessment Report No. 1), Health and Welfare, Canada, p. 56.

Health Canada, 1996. Values presently used by contaminants toxicology section. Food Directorate, Ottawa, Canada.

Hicks, C.R., 1973. Fundamental Concepts in the Design of Experiments. Hold, Rinehart and Winston, Toronto, p. 349.

Hooper, K., Petreas, M.X., Chuvakova, T., Kazbekova, G., Druz, N., Semenova, G., Sharmanov, T., Hayward, D., She, J., Visita, P., Winkler, J., McKinney, M., Wade, T.J., Grassman, J., Stephens, R., 1998. Analysis of breast milk to assess exposure to chlorinated contaminants in Kazakstan: High levels of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in agricultural villages of Southern Kazakstan. Environmental Health Perspectives 106, 797.

Hu, K., Bunce, N.F., 1999. Metabolism of Polychlorinated dibenzo-*p*-dioxins and related dioxin-like compounds. Journal of Toxicology and Environmental Health, Part B 2, 183.

Huisman, M., Koopman-Esseboom, C., Lanting, C.I., van der Paauw, C.G., Tuinstra, L.G.M.Th., Fidler, V., Weisglas-Kuperus, N., Sauer, P.J.J., Boersma, E.R., Touwen, B.C.L., 1995. Neurological condition in 18-month-old children perinatally exposed to polychlorinated biphenyls and dioxins. Early Human Development 43, 165.

Hulster, A., Marschner, H., 1993. Transfer of PCDD/PCDF from contaminated soils to food and fodder crop plants. Chemosphere 27, 439.

Hulster, A., Muller, J.F., Marschner, H., 1994. Soil-plant transfer of polychlorinated dibenzo-*p*-dioxins and dibenzofurans to vegetables of the cucumber family (Cucuribitaceae). Environmental Science and Technology 28, 1110.

IARC (International Agency for Research on Cancer), 1997. IARC monographs on the evolution of carcinogenic risks to humans, vol. 69. Polychlorinated dibenzo-*para*-dioxins and polychlorinated dibenzofurans. World Health Organization, Geneva, p. 666.

IOM (Institute of Medicine), 1994. Veterans and Agent Orange—Health effects of herbicides used in Viet Nam. National Academy Press, Washington, DC, p. 812.

IOM (Institute of Medicine), 2001. Veterans and Agent Orange Update 2000. National Academy Press, Washington, DC, pp. 604.

Jensen, A.A., 1987. Polychlorobiphenyls (PCBs), polychlorodibenzo-*p*-dioxins (PCDDs) and polychlorodibenzofurans

(PCDFs) in human milk, blood and adipose tissue. Science of the Total Environment 64, 259.

Lutter, C., Iyengar, V., Barnes, R., Chuvakova, T., Kazbekova, G., Sharmanov, T., 1998. Breast milk contamination in Kazakhstan: implications for infant feeding. Chemosphere 37, 1761.

Matsuda, M., Funeno, H., Quynh, H.T., Cau, H.D., Wakimoto, T., 1994. PCDDs/DFs pollution in soils from Viet Nam. In: Cau, H.D., Dai, L.C., Minh, D.Q., Thuy, L.B. (Eds.), Herbicides in War—The Long-term Effects on Man and Nature. 2nd International Symposium, Ha Noi, 1993. Ha Noi: 10-80 Committee. Hanoi Medical School, pp. 55–58.

McLachlan, M.S., 1993. Digestive tract absorption of polychlorinated dibenzo-*p*-dioxins, dibenzofurans, and biphenyls in a nursing infant. Toxicology and Applied Pharmacology 123, 68.

McLachlan, M., 1999. Interpreting the accumulation of dioxins and related compounds in plants. Organohalogen Compounds 41, 325.

NATO (North Atlantic Treaty Organization), 1988. International Toxicity Equivalent Factor (I-TEF) method of risk assessment for complex mixtures of dioxins and related compounds. Pilot study on international information exchange on dioxins and related compounds. Committee on the Challenges of Modern Society, vol. 176, p. 26.

Nestrick, T.J., Lamparski, L.L., Frawley, N.N., Hummel, R.A., Kocher, C.W., Mahle, N.H., McCoy, J.W., Miller, D.L., Peters, T.L., Pillepich, J.L., Smith, W.E., Tobey, S.W., 1986. Perspectives of a large scale environmental survey for chlorinated dioxins: Overviews and soil data. Chemosphere 15, 1453.

Neumann, G., Hulster, A., Romheld, V., 1999. PCDD/PCDF-mobilizing compounds in root exudates of zucchini. Organohalogen Compounds 41, 331.

Orians, G.H., Pfeiffer, E.W., 1970. Ecological effects of the war in Viet Nam. Science 168, 544.

Patandin, S., Dagnelie, P.C., Mulder, P.G.H., de Coul, E.O., van der Veen, J.E., Weisglas-Kuperus, N., Sauer, P.J.J., 1999a. Dietary exposure to polychlorinated biphenyls and dioxins from infancy until adulthood: A comparison between breast-feeding, toddler, and long-term exposure. Environmental Health Perspectives 107, 45.

Patandin, S., Lanting, C.I., Mulder, P.G.H., Boersma, E.R., Sauer, P.J.J., Weisglas-Kuperus, N., 1999b. Effects of environmental exposure to polychlorinated biphenyls and dioxins on cognitive abilities in Dutch children at 42 months of age. Journal of Pediatrics 134, 33.

Phiet, P.H., Phuong, N.T.N., Tan, N.V., Schecter, A., 1994. Dioxin levels in 93 individual fat tissue specimens of patients living in South Viet Nam. In: Cau, H.D., Dai, L.C., Minh, D.Q., Thuy, L.B. (Eds.), Herbicides in War—The Long-term Effects on Man and Nature. 2nd International Symposium, Ha Noi, 1993. Ha Noi: 10-80 Committee. Hanoi Medical School, pp. 22–24.

Pohl, H., DeRosa, C., Holler, J., 1995. Public health assessment for dioxins exposure in soil. Chemosphere 31, 2437.

Quynh, H.T., Dung, B.T., Thuy, L.B.T., Hoa, M.T., 1994. First results on the transfer of 2,3,7,8-TCDD in nature and its persistence in human body in south Viet Nam. In: Cau, H.D., Dai, L.C., Minh, D.Q., Thuy, L.B. (Eds.), Herbicides in War—The Long-term Effects on Man and Nature. 2nd International Symposium, Ha Noi, 1993. Ha Noi: 10-80 Committee. Hanoi Medical School, pp. 81–91.

Raum, E., Seidler, A., Schlaud, M., Knoll, A., Webling, H., Kurtz, K., Schwartz, F.W., Robra, B.-P., 1998. Contamination of human breast milk with organochlorine residues: A comparison between East and West Germany through sentinel practice networks. Epidemial Community Health 52, 50S.

Rogan, W.J., 1996. Pollutants in breast milk. Archives of Pediatrics and Adolescent Medicine 150, 981.

Rogan, W.J., Blanton, P.J., Portier, C.J., Stallard, E., 1991. Should the presence of carcinogens in breast milk discourage breast feeding? Regulatory Toxicology and Pharmacology 13, 228.

Roumak, V.S., Poznyakov, S.P., Antonyuk, V.V., An, N.Q., Sofronov, G.A., 1995. Consistent deterioration of general health status in South and North Vietnamese exposed to Agent Orange. Organohalogen Compounds 25, 161.

Schade, G., Heinzow, B., 1998. Organochlorine pesticides and polychlorinated biphenyls in human milk of mothers living in northern Germany: Current extent of contamination, time trend from 1986 to 1997 and factors that influence the levels of contamination. Science of the Total Environment 215, 31.

Schecter, A. (Ed.), 1994a. Dioxins and Health. Plenum Press, New York, p. 710.

Schecter, A., 1994b. Exposure assessment—Measurement of dioxins and related chemicals in human tissues. In: Schecter, A. (Ed.), Dioxins and Health. Plenum Press, New York, pp. 449–485.

Schecter, A., 1998. A selective historical review of congener-specific human tissue measurements as sensitive and specific biomarkers of exposure to dioxins and related compounds. Environmental Health Perspectives 106, 737.

Schecter, A.J., Ryan, J.J., Gross, M., Weerasinghe, N.C.A., Constable, J.D., 1985. Chlorinated dioxins and dibenzofurans in human tissues from Viet Nam, 1983–1984. In: Rappe, C., Choudhary, G., Keith, L.H. (Eds.), Chlorinated Dioxins and Dibenzofurans in Perspective. Lewis Publishers, Michigan, pp. 35–50.

Schecter, A., Ryan, J.J., Constable, J.D., 1987. Polychlorinated dibenzo-*p*-dioxin and polychlorinated dibenzofuran levels in human breast milk from Viet Nam compared with cow's milk and human breast milk from the North American continent. Chemosphere 16, 2003.

Schecter, A., Kooke, A.R., Serne, P., Olie, K., Huy, D.Q., Hue, N., Constable, J.D., 1989a. Chlorinated dioxin and dibenzofuran levels in food samples collected between 1985 and 1987 in the north and south of Viet Nam. Chemosphere 18, 627.

Schecter, A., Furst, P., Kruger, C., Meemken, H.-A., Groebel, W., Constable, J.D., 1989b. Levels of polychlorinated dibenzofurans, dibenzodioxins, PCBs, DDT and DDE, hexachlorobenzene, dieldrin, hexachlorocyclohexanes and oxychlordane in human breast milk from the United States, Thailand, Viet Nam, and Germany. Chemosphere 18, 445.

Schecter, A., Papke, O., Ball, M., 1990a. Evidence for transplacental transfer of dioxins from mother to fetus: Chlorinated dioxin and dibenzofuran levels in the livers of stillborn infants. Chemosphere 21, 1017.

Schecter, A., Ryan, J.J., Constable, J.D., Baughman, R., Bangert, J., Furst, P., Wilmers, K., Oates, R.P., 1990b. Partitioning of 2,3,7,8-chlorinated dibenzo-*p*-dioxins and dibenzofurans between adipose tissue and plasma lipid of 20 Massechusetts Viet Nam Veterans. Chemosphere 20, 951.

Schecter, A., Furst, P., Furst, C., Groebel, W., Constable, J.D., Kolesnikov, S., Beim, A., Boldonov, A., Trubitsun, E., Vlasov, B., Cau, H.D., Dai, L.C., Quynh, H.T., 1990c. Levels of chlorinated dioxins, dibenzofurans and other chlorinated xenobiotics in food from the Soviet Union and the south of Viet Nam. Chemosphere 20, 799.

Schecter, A., Papke, O., Ball, M., Cau, H.D., Dai, L.C., Ming, N.Q., Quynh, H.T., Phuong, N.N.T., Phiet, P.H., Chi, H.K., Vo, D.T., Constable, J.D., Spencer, J., 1992a. Dioxin and dibenzofuran levels in blood and adipose tissue of Vietnamese from various locations in Viet Nam in proximity to Agent Orange spraying. Chemosphere 25, 1123.

Schecter, A., McGee, H., Stanley, J., Boggess, K., 1992b. Dioxin, dibenzofuran and PCB, including coplaner PCB levels in the blood of Viet Nam veterans in the Michigan Agent Orange study. Chemosphere 25, 205.

Schecter, A., Dai, L.C., Bich Thuy, L.T., Quynh, H.T., Minh, D.Q., Cau, H.D., Phiet, P.H., Ngoc Phuong, N.T., Constable, J.D., Baughman, R., Papke, O., Ryan, J.J., Furst, P., Räisänen, S., 1995. Agent Orange and the Vietnamese: The persistence of elevated dioxin levels in human tissues. American Journal of Public Health 85, 516.

Schecter, A., Ryan, J.J., Papke, O., 1998. Decrease in levels and body burden of dioxins, dibenzofurans, PCBs, DDE, and HCB in blood and milk in a mother nursing twins over a 38 month period. Chemosphere 37, 1807.

Schecter, A., Dai, L.C., Papke, O., Prange, J., Constable, J.D., Matsuda, M., Thao, V.D., Piskac, A.L., 2001. Recent dioxin contamination from Agent Orange in residents of a southern Viet Nam city. Journal of Occupation and Environmental Medicine 43, 435.

Schuhmacher, M., Domingo, J.L., Llobet, J.M., Kiviranta, H., Vartiainen, T., 1999. PCCD/F concentrations in milk of non-occupationally exposed women living in Southern Catalonia, Spain. Chemosphere 38, 995.

Schulz, D., 1994. Recent measures to further reduce dioxin impact on man and the environment in the federal republic of Germany. Chemosphere 29, 2439.

Somogyi, A., Beck, H., 1993. Nurturing and breast-feeding: Exposure to chemicals in breast milk. Environmental Health Perspectives Supplements 101, 45.

Stanton, S.L., 1985. Green Berets at war: US Army Special Forces in southeast Asia, 1956–1975. Ivy Books, New York, p. 383.

Startin, J.R., 1994. Dioxins in food. In: Schecter, A. (Ed.), Dioxins and Health. Plenum Press, New York, pp. 115–137, 710.

Tarkowski, S., Yrjanheikki, E., 1989. Polychlorinated dibenzodioxins and dibenzofurans in human milk: An interim health risk evaluation. Chemosphere 18, 1107.

Tschirley, F.H., 1969. Defoliation in Viet Nam. Science 163, 779–786.

US Army documents (declassified), 1967. Disposal of herbicide waste at Bien Hoa and Da Nang military bases. Department of the Army. The US Army Center of Military History, Fort Lesley J. McNair DC, 20319-5048, USA.

US Army documents (declassified), 1969. Accidental herbicide damage at Nha Trang, Bien Hoa, Phu Cat and Saigon military bases. Department of the Army. The US Army Center of Military History, Fort Lesley J. McNair DC, 20319-5048, USA.

US Army documents (declassified), 1970. Agent Orange spills on the Bien Hoa military base. Department of the Army. The US Army Center of Military History, Fort Lesley J. McNair DC, 20319-5048, USA.

US Army documents, 1995. Print out of aerial herbicide applications, using grid coordinates, in the Aluoi Valley. Department of the Army. The US Army Center of Military History, Fort Lesley J. McNair DC, 20319-5048, USA.

US Army documents (declassified), 2001. Action reports and memoranda regarding the A Shau Valley, Viet Nam. Authority NND 931713/903562, National Archives, Washington, DC.

US EPA (US Environmental Protection Agency), 1999a. Region III. Available from http://www.epa.gov/reg3hwmd/risk/riskmenu.htm. Accessed 27 November, 1999.

US EPA (US Environmental Protection Agency), 1999b. Region IX. Available from http://www.epa.gov/region09/waste/sfund/prg/index.htm. Accessed 3 December, 1999.

Westing, A.H., 1984. Herbicides in war: past and present. In: Westing, A.H. (Ed.), Herbicides In War, The Long-term Ecological and Human Consequences. Stockholm International Peace Research Institute, Taylor and Francis, London and Philadelphia, pp. 3–24.

WHO/EURO (World Health Organization/European Regional Office), 1988. PCBs, PCDDs and PCDFs in breast milk: Assessment of health risks. In: Grandjean, P., Tarkowski, S., Kimbrough, R., Yrjänheikki, E., Rantanen, J.H. (Eds.), Environmental Health Series Report No. 29. World Health Organization Regional Office for Europe, Copenhagen, p. 116.

WHO/EURO (World Health Organization/European Regional Office), 1989. Levels of PCBs, PCDDS and PCDFs in breast milk: Results of WHO-coordinated interlaboratory quality control studies and analytical field studies. In: Yrjanhaiki, E.J. (Ed.), Environmental Health Series Report No. 34. World Health Organization Regional Office for Europe, Copenhagen, p. 92.

WHO/EURO (World Health Organization/European Regional Office), 1991. Consultation on Tolerable Daily Intake from food of PCDDs and PCDFs, Bilthoven, Netherlands, 4–7 December 1990. Region Office for Europe Summary Report. EUR/ICP/PCS 030(S)0369n. World Health Organization Regional Office for Europe, Copenhagen, p. 5.

WHO/EURO (World Health Organization/European Regional Office), 1998a. WHO revises the Tolerable Daily Intake (TDI) for dioxins. World Health Organization European Centre for Environment and Health; International Programme on Chemical Safety. Organohalogen Compounds 38, 295.

WHO/EURO (World Health Organization/European Regional Office), 1998b. Assessment of the health risk of dioxins: re-evaluation of the Tolerable Daily Intake (TDI). Executive Summary. World Health Organization, European Centre

for Environment and Health; International Programme on Chemical Safety. WHO Consultation, 25–29 May, 1998, Geneva, Switzerland, p. 22.

WHO/EURO (World Health Organization/European Regional Office), 2001. Interlaboratory quality assessment of levels of PCBs, PCDDs and PCDFs in human milk and blood plasma. Available from http://www.who.dk/document/e70039.pdf. Accessed 12 June, 2001.

Wise, J., 1997. High amounts of chemicals found in breast milk. Biomedical Journal 314, 1505.

Zaffiri, S., 1988. Hamburger Hill. Presidio Press, Novato, CA, p. 304.