

# Health Education and Public Health

2019; 2(2): 170 – 180 . doi: 10.31488 /heph.119

## Review

# A Review of Public Health in Vietnam: 50 Years after Agent Orange was Sprayed

Alvin L. Young\*

I. Consulting, Inc. 1810 Tranquility Drive, Cheyenne, Wyoming, USA

\*Corresponding author: Young AL, Consulting, Inc. 1810 Tranquility Drive, Cheyenne, Wyoming, USA, E-mail: youngrisk@aol.com

Received: February 11, 2019; Accepted: March 25, 2019; Published: March 28, 2019

## Abstract

Normal relations between the United States and the Socialist Republic of Vietnam in 1995 permitted scientists, public health experts, and the media to visit Vietnam and assess the impact of the use of Agent Orange on the ecology and peoples of Vietnam. Evaluation of scientific data suggested that exposures to the toxic contaminant 2,3,7,8-Tetrachlorodibenzo-p-dioxin, TCDD, in 2,4,5-T-containing tactical herbicides sprayed on jungle forests and mangroves were minimal for Vietnamese located in villages near combat areas. Although some tactical herbicides were sprayed in rural areas, the largest sources of polychlorinated dioxins and furans (PCDD/Fs) were from biomass-burning, especially from municipal dumpsites. In urban areas, sources of PCDD/Fs were from industrial emissions. Measurable levels of PCDD/Fs in Vietnamese were in those residents associated with dioxin “hotspots”, specifically from former airbases where Agent Orange had been stored or loaded on US military aircraft. These hotspots were characterized by high soil and sediments levels of 2,3,7,8-TCDD that persisted in spill sites for over 50 years. Movement of low levels of PCDD/Fs into food sources were a continuing source of contamination for residents adjacent to hotspots. However, levels of TCDD in their tissues were generally comparable to other Asian populations. Allegations of cancers, other diseases, and horrific birth defects due to residual dioxins on the public health of those communities have not been validated. The US and Vietnam have committed to remediation of the hotspots in Southern Vietnam. The Public Health of the Vietnamese continues to improve due to regulatory actions of Vietnam’s government to reduce industrial and atmospheric pollution.

Keywords: agent orange, tactical herbicides, Vietnam, dioxin hotspots, operation ranch hand, pcdd/fs sources, vietnamese health studies

## Introduction

For five decades there have been continuous controversies over the use of Agent Orange and other tactical herbicides sprayed for defoliation and crop destruction purposes during the Vietnam-American War (Vietnam War) from 1961 - 1971. Indeed, few occupational health issues have sustained such international attention as have the use of herbicides in Southeast Asia. The opening and establishment of normal relations in 1995 between the United States and the Socialist Republic of Vietnam provided an opportunity for scientists, public health experts, and the media to visit Southern Vietnam, and attempt to assess the long-term impact of the defoliation program. Since that time, there have been hundreds of negative and sensational stories circulated, hundreds of articles published, and an extensive propaganda campaign by the government of Vietnam and others. The search for facts and truths regarding public health in Vietnam as related to the use of Agent Orange has been lost among the continuous media coverage. This article attempts to analyze the public health threat that was posed by the spraying of Agent Orange over 50 years ago.

## Background

To conduct an assessment of the potential public health threat that Agent Orange and its associated dioxin contaminant may have caused after the Vietnam War required documentation on the military herbicides that were used in the Vietnam War; how exposure occurred and was measured; the persistence of the dioxin contaminant; toxicology of the herbicides and dioxin contaminant; validity of the cause of diseases reported; and the politics associated with Agent Orange.

## Use of Herbicides

The history of the use of tactical herbicides in Southern Vietnam by the United States Military is well documented [1-3]. The “initial deployment” of the use of herbicides occurred from 29 December 1961 to 18 March 1965 and involved a small cadre of United States Air Force (USAF) and US Army Special Forces units supported by the Army of Vietnam (ARVN) and Air Force of Vietnam (VNAF). Approximately 3 million liters of 2,4,5-T (2,4,5-trichlorophenoxy acetic

acid) and 2,4-D (2,4-dichlorophenoxy acetic acid) were sprayed primarily along roadways and lines of communication [4]. The “operational phase” of Operation RANCH HAND began on 29 March 1965 and ended on 7 January 1971 [4]. During this phase, three tactical herbicides were specifically deployed to reduce ground and forest cover as a means of improving visibility and reducing combat casualties [5]. Tactical herbicides referred to herbicides that were used in combat operations, not routine weed control programs on military bases. The three tactical herbicides sprayed in Operation RANCH HAND, the USAF Aerial Defoliation Program, were; Agent Orange (2,4-D and 2,4,5-T: 43.33 million liters); Agent White (Picloram, 4-amino-3,5,6-trichloropicolinic acid, and 2,4-D: 21.8 million liters); and, Agent Blue (Cacodylic acid: 6.1 million liters) [4]. Approximately 14% of South Vietnam’s woody vegetation were sprayed one or more times [6].

### Determination of Exposure to Tactical Herbicides

Four basic ways have been described to assess potential exposure to the tactical herbicides sprayed in Southern Vietnam. The first approach was the use historical contemporary military records to assess the effectiveness of the policies and procedures for conducting the spray missions, thus minimizing exposure to ground troops. The second approach was to develop models using the characteristics of the spray system, the pathway of the aircraft, and location of ground troops. The third approach was to use surrogates of exposure. The fourth approach was to identify a marker and track that marker through the environment and human populations.

It is estimated that approximately 95% of all defoliants (tactical herbicides) were sprayed by fixed wing aircraft assigned to Operation RANCH HAND. The remaining 5% were sprayed by the US Army Chemical Corps, primarily from helicopter spray systems [5]. A critical review of contemporary military records was published in 2004 [5]. The conclusion: Detailed policies and procedures for approval and execution of spray missions ensured that friendly forces were not located in the areas targeted for spraying. Fighter aircraft assigned to accompany each spray mission frequently suppressed much of the hostile fire with bombs and other ordnance. Confirmed clearance of the target was necessary to avoid friendly casualties. Historical records establish that these policies and procedures were strictly followed. Exposure of troops whether from direct spraying or movement through areas recently sprayed was very unlikely [5].

Methodologies using Geographic Information Systems (GIS) were developed for characterizing exposure of ground troops in Vietnam to Agent Orange and other herbicides based upon historical reconstruction from military records [5, 7]. However, the use of records of wartime military troop movements in Vietnam and herbicide operations had specific limitations in exposure reconstruction. Specifically: Of particular importance was the accuracy of the geographic data, i.e., the maps used by the aircrews and ground troops. Electronic navigation aids gave aircrews the bearing of their aircraft from a transmitter (always in friendly territory) and in some cases approximate distances but were incapable of fixing the location of aircraft with precision. To fix locations within one nautical mile (1,850 m) for a

plane 32 km from a TACAN transmitter would have been exceptional. The signals were not ordinarily received at the low altitudes flown on spray missions. Navigation by the crew using visual orientation and reference to a map was the only means that aircrews on missions had for establishing their location. In turn, this was dependent on the inherent precision of the map, the accuracy with which it depicted the surface features, and the skill of the individual pilot or navigator [5].

Ground troops used an entirely different series of maps, typically of 1:50,000 scale. The dense vegetation severely limited the view available to field forces even under the best of conditions, thus making accurate navigation difficult [5]. Despite these limitations, Stellman et al. developed a GIS for assessment of herbicide exposure for Vietnam [7]. The GIS framework used a records-based exposure reconstruction methodology to represent spatial and temporal relationships between instances of herbicide application and the location of (potentially) exposed individuals, military units, or other defined populations. The GIS produced as its output an “exposure opportunity vector” in a format containing an herbicide Exposure Opportunity Index (EOI) representing the number of “hits” that occurred within 0.5, 1, 2, and 5 km from the center of a GIS grid [7].

In 2008, a quantitative evaluation was conducted of the Exposure Opportunity Index model [8]. The researchers noted that there was an enormous range of EOI predictions; e.g., a location 4 km from the flight path of a high EOI mission could have about 279 times the EOI of a point on the flight path from a low EOI mission. This could be interpreted that an essentially unexposed person would be assigned an EOI score 279 times higher than a more likely exposed individual because of computational vagaries of the model. The conclusion was that the GIS-EOI model was simply not useful for exposure characterizations in the conduct of epidemiologic studies [8].

The Center for Disease Control and Prevention (CDC) developed an approach of using surrogates of exposure that involved conducting an extensive analysis of self-reported exposure histories, herbicide spraying records, and military records to include dates and map coordinates of herbicide spray missions, military unit locations, and dates of service for each Vietnam veteran. These data provided five different Agent Orange exposure scores [9].

The fourth approach to determine if exposure occurred was by using a marker that could confirm human exposure. One of the premier achievements of environmental science was the development of increasingly sophisticated methods for biomonitoring of low levels of chemicals in human tissue [9]. In the 2,4,5-T herbicide component of Agent Orange was the presence of an unwanted contaminant that persisted in both the environment and in human tissue. That contaminant was TCDD, 2,3,7,8-tetrachlorodibenzo-p-dioxin (dioxin) [10]. In 1988, the CDC compared levels of serum TCDD in 646 US Army veterans who served as ground troops in the most heavily sprayed regions of Vietnam with those of 97 Vietnam-era veterans who had not served in Vietnam. The results showed that the serum TCDD was essentially identical in both groups, both having means and medians of about 4 parts-per-trillion (ppt). Moreover, CDC found that there was no relationship between surro-

gates of exposure and the measured TCDD levels in the Vietnam ground troops [10].

#### Determination of the Extent of the Dioxin Contamination

The presence of TCDD (abbreviated 2,3,7,8-TCDD) in human and environmental samples never occurred as an isolated chemical, but always in the presence of similar poly-chlorinated compounds. The analytical methodology for the detection of dioxin involved the use of high-resolution gas chromatography (HRGC) and high-resolution mass spectrometry (HRMS). When employing HRGC-HRMS, scientists found that in the human body fat and blood serum, there were potentially seven poly-chlorinated dibenzo-p-dioxins (PCDDs) or congeners, all having chlorine atoms in the 2,3,7,8 positions on the molecules, and ten polychlorinated dibenzofurans (PCDFs) congeners, all having chlorine atoms in the 2,3,7,8 positions on the molecules [11]. Because of their highly lipophilic nature, human fat tissues retained these 17 molecules or congeners [11].

Analysis of 249 samples of Agent Orange from 4 different producers of Agent Orange during the Vietnam War, identified five congeners with the 2,3,7,8-TCDD being the predominant congener and the other four in trace quantities only [3]. The mean TCDD concentration in 1,082 samples of Agent Orange spanning the years from 1963 through 1969 was 1.88 mg/kg (parts-per-million) [12]. These data permitted the calculation that the total estimated amount of the contaminant 2,3,7,8-TCDD associated with the 2,4,5-T – containing tactical herbicides used in Vietnam was 130 kg [12]. Considering the total number of spray missions by RANCH HAND aircraft, the locations of those missions, and the total number of liters sprayed, it was likely that at least 96% to 98% of the 130 kg of TCDD were aerially sprayed over the jungles and mangrove swamps of Vietnam [12]. The remaining 2% to 4% (2.6 – 5.2 kg) were likely sprayed by the US Army Chemical Corps or were spilled at the storage and aircraft reloading sites [12]. These former storage and aircraft reloading sites in Southern Vietnam have been identified as “hot spots”, and, likely constitute a long-term source of dioxin contamination to adjacent communities [13, 14].

#### Understanding the Environmental Fate of Agent Orange and 2,3,7,8-TCDD

To understand the potential threat to public health, it was necessary to address the environmental fate of the herbicides, including changes in TCDD content over time, the persistence of TCDD and the herbicides in the environment, and the degree of likely penetration of the herbicides into the ground. In 2004, a detailed examination of the scientific literature was conducted of the environmental fate and bioavailability of Agent Orange and its associated dioxin during the Vietnam War [15]. The results of that research as reported was as follows: The evaluations of the spray systems used to disseminate herbicides in Vietnam showed that they were capable of highly precise applications both in terms of concentrations sprayed and area treated. Research on tropical forest canopies with leaf area indices (a measure of foliage density) from 2 to 5 indicated that the amount of herbicide and associated TCDD reaching the forest floor would have been between 1 and 6% of the total

aerial spray. Studies of the properties of plant surface waxes of the cuticle layer suggested that Agent Orange, including the TCDD, would have dried (i.e., be absorbed into the wax layer of the plant cuticle) upon spraying within minutes and could not be physically dislodged. Studies of Agent Orange and the associated TCDD on both leaf and soil surface have demonstrated that photolysis by sunlight would have rapidly decreased the concentration of TCDD, and this process continued in shade. Studies of ‘dislodgeable foliar residues’ (DFR, the fraction of a substance that is available for cutaneous uptake from the plant leaves) showed that only 8% of the DFR was present 1 hr after application. This dropped to 1% of the total 24 hrs after application. Studies with human volunteers confirmed that after 2 hrs of saturated contact with bare skin, only 0.15-0.46% of 2,4,5-T, one of the phenoxy acetic acid compounds that was an active ingredient of Agent Orange, entered the body and was eliminated in the urine [15].

The conclusion of these observations was that the prospect of exposure to TCDD from Agent Orange to personnel moving through sprayed jungles and mangroves seemed unlikely or indistinguishable from background levels due to the environmental dissipation of TCDD, little bioavailability, and the properties of the herbicides and circumstances of application by RANCH HAND aircraft [15].

In 2007, soil and water samples were collected from the Ma Da area of III Corps, an area repeatedly sprayed with Agent Orange, 1967 – 1969. The results did not provide evidence that the detected dioxins (mostly Octachlorodibenzo-p-dioxin, OCDD) that were bound to groundwater colloids were residues from the Vietnam War. Indeed, the dioxins were likely from combustion sources [16]. If contaminated soil colloids readily moved with water sources through areas repeatedly sprayed with Agent Orange, the analyses of sediments collected from coastal lagoons should provide evidence of TCDD contamination. Piazza et al. in 2010 reported on the analyses of sediments collected from nine Central Vietnam coastal lagoons [17]. Their conclusions: “Samples from nine Central Vietnam coastal lagoons, together with three soils and sediments collected in two freshwater reservoirs of the Thua Thien-Hue Province, were analyzed for polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/Fs). Total concentrations were low, from 192 to 2,912 pg/g (parts-per-trillion) and depth profiles in Tam Giang-Cau Hai sediment cores showed only minor changes over time in PCDD/Fs input and composition. OCDD (not found in Agent Orange) was the prevailing congener (approximately 90%), indicating combustion as the main PCDD/Fs source to these coastal systems. The 2,3,7,8-tetrachloro-p-dioxin (TCDD) largely sprayed together with Agent Orange over the study area during the war (1961-1971), was absent or very low. These results support the hypothesis of rapid degradation soon after spraying” [17].

Studies of residual sedimentary TCDD were conducted in 2010 from Can Gio, Southern Vietnam in an area known for the defoliation of its mangrove forests by aerial spraying with Agent Orange during the Vietnam War [18]. The results indicated that the TCDD potentially from Agent Orange occurred in only low concentrations, while the main contributors to the Toxicity Equivalent Quotient (TEQ) values were from natural



sources [18].

### Analyses of Hotspots in Southern Vietnam

The persistence of 2,3,7,8-TCDD in soils has been thoroughly documented, not only in Southern Vietnam, but in locations where Agent Orange was sprayed or stored in the United States. In 2004, an article was published on the persistence of TCDD on a test area of Eglin Air Force Base (AFB), Florida that had been used for developing and evaluating the defoliation spray systems deployed to Vietnam for both Operation RANCH HAND and for the helicopters used by the US Army Chemical Corps [19]. On an area of less than 3 km<sup>2</sup> during the period 1962 – 1970, approximately 75,000 kg of 2,4,5-T containing approximately 3.1 kg of TCDD were aerially disseminated [19]. For comparison, each hectare on the Eglin AFB test grid received at least 876 kg 2,4,5-T and at least 1,300 times more TCDD than a hectare sprayed with Agent Orange in Vietnam [19]. Soil samples analyzed from 1974 – 1984 contained less than one percent of the TCDD that was calculated to have been disseminated during the test period. The explanation was that most of the TCDD (99 percent) was likely photodegraded during the days immediately after dissemination. The one percent remaining that was bound in the soil persisted for at least 20 years and was essentially all found within the top 15 cm of the soil profile [19]. Studies by Crosby and Wong in 1977 confirmed that TCDD in Agent Orange applied on glass plates lost most or all the TCDD in a single day, due principally to photochemical de-chlorination [20].

The last mission of Agent Orange in Operation RANCH HAND occurred in April 1970 [5]. Following the termination of the use of Agent Orange, the USAF directed that all remaining stocks (5.24 million liters) be collected from throughout South Vietnam, re-drummed, and shipped to Johnston Island, Central Pacific Ocean, and placed in open storage from April 1972 to July 1977 when a suitable method for disposal was arranged [21]. Simultaneously, the remaining stocks of Agent Orange (3.3 million liters) that were not shipped to Vietnam remained in the US in storage at the Naval Construction Battalion Center (NCBC), Gulfport, Mississippi, from December 1969 to June 1977 [21]. Significant quantities of herbicide leaked or were spilled while maintaining the integrity of the inventories at both storage sites (>80,000 liters at Johnston Island) [21]. In 1977, the two inventories were destroyed by at-sea incineration and a site monitoring program was immediately initiated. In spill sites, the levels of phenoxy herbicides were > 62,000 ppm, while the TCDD often exceeded 0.27 ppm (270,000 ppt). Over a seven-year period, at both locations, a loss of approximately 98% of the two herbicides (2,4-D and 2,4,5-T) occurred. The TCDD was much more persistent with values ranging from 15% to 83% loss in the top 8 cm of soil surface [21]. The degradation of the herbicides, and likely the TCDD, were attributed to intense surface sunlight and temperatures, and very high concentrations of fungi that dominated the spill sites. However, the inconsistency of the TCDD concentrations found in “old” and “new” spills made the determination of loss questionable [21].

The centerpiece of the 2nd Agent Orange and Dioxin Remediation Workshop held in Hanoi, Vietnam, 18 – 20 June 2007

was a US Department of Defense report and maps given to the Vietnam’s Ministry of Defence showing the location of former tactical herbicide storage and loading sites in Southern Vietnam [3, 12]. The report and maps identified four “hotspot” that should be the focus of analytical studies of contaminated soils containing PCDD/PCDFs. These locations were former airbases where Operation RANCH HAND and/or US Army Chemical Corps stored and loaded aircraft for defoliation missions, and included Da Nang, Bien Hoa, Phu Cat and Nha Trang. Data on the quantities of Agent Orange stored and or loaded at those locations were also provided [3, 12].

The most thorough study of dioxin contamination in the vicinity of Da Nang Airbase/Airport was conducted by Hatfield Consultants, West Vancouver, British Columbia, Canada, and reported in April 2007 [22]. This Report was sponsored by the Office of the National Committee 33, Ministry of Natural Resources and Environment, Hanoi, Vietnam. The maximum soil TCDD concentration was 365,000 ppt (0.365 ppm), confirming the Airbase as a significant “hotspot”. Dioxin-contaminated sediments had migrated into the Phu Loc River and presumably into Da Nang Bay. However, the TCDD level at the point of entry into the Phu Loc River was 6.46 ppt. The Report noted that the highest concentrations of the TCDD was essentially found in the top 10 cm of the soil/sediment profiles [22]. These data were consistent with the soil data from the long-term studies conducted at the former Agent Orange Storage Site at NCBC where the highest concentrations ranged from 148,000 ppt to 510,000 ppt (median 270,000 ppt) [23]. TCDD residues (2,000 ppt) were detected in a drainage canal at distance of 3,000 m, but non-detected at 4,000 m (23). Biological samples were positive for TCDD at 3,000 m [23].

A recent publication by Thuong et al. 2015 provided in-depth analyses of the contamination of Bien Hoa Airbase to include both the loading and storage sites for RANCH HAND missions, and the PACER IVY site, where 11,000 drums of Agent Orange were re-drummed and shipped to Johnston Island in April 1972 [12, 24]. The TEQ concentrations of PCDD/Fs in soils and sediments varied from 7.6 to 962,000 pg/g and 17 to 4,860 pg/g (ppt), respectively [24]. The authors reported that from 95 to 99% of the TEQ was attributed to the 2,3,7,8-TCDD. The highest concentrations were associated with the spilling of 28,000 liters of Agent Orange in March 1970, and the PACER IVY re-drumming site [3, 12, 24]. Significant concentrations of OCCD were found in sediments from selected sites in and around Bien Hoa Airbase, suggesting combustion emission sources in the area [24]. Indeed, biomass-burning and open burning of municipal wastes at low temperatures, especially at dumpsites, have been identified as the major sources of PCDD/Fs in rural areas, and the PCDD/Fs in urban areas were associated with industrial emissions [25, 26]. These observations were important for understanding the dioxin profiles in the Vietnamese population.

In the 2009 Final Report prepared by Viet Nam – Russia Tropical Center with the technical support from Hatfield Consultants, the Agent Orange dioxin hotspots at Phu Cat Airbase in Phu Yen Province were investigated [27]. Dioxin concentrations in the storage area for the tactical herbicides and adjacent to the runway exceeded 230,000 ppt TCDD, but soils

collected in the Loading and Washing Areas were considerably lower and, from the viewpoint of the authors, likely did not represent a threat to human health or the environment [27].

An additional “hotspot” has been studied in Thua Thien Hue Province in Central Vietnam [28]. The A-So Airbase, located in the Dong Son Commune of A-Luoi District, was used by the US Army Chemical Corps to store Agent Orange for selected helicopter missions on the Ho Chi Minh Trail. As with both Da Nang and Bien Hoa Airbases, numerous soil and sediment samples analyzed for PCDD/Fs were found to range from 2.7 to 746 ng TEQ/kgdw (ppt) [28]. TCDD was the predominant congener, however, all seventeen 2,3,7,8-congeners were found in different soil layers [28].

### PCDD/Fs Contamination of Food Sources

The most extensive study of TCDD contamination of mammals, birds, reptiles, fishes, and amphibians was the 15-year ecological study conducted on Test Area C-52A, Eglin AFB Reservation, Florida, 1968 – 1983 [19]. As previously noted, the Test Area was the location where USAF tested the spray equipment later deployed to South Vietnam. On an area of less than 3 km<sup>2</sup> 3.1 kg of 2,3,7,8-TCDD was disseminated in the approximate 75,000 kg of 2,4,5-T herbicide during the years 1962 – 1970 [19]. The results of the analyses of biological samples: More than 340 species of organisms were observed and identified within the test area. More than 300 biological samples were analyzed for TCDD, and detectable residues were found in 16 of 45 species examined. Examination of the ecological niche of the species containing TCDD residues suggested each was in close contact with contaminated soil...From studies of organisms that ingested TCDD-contaminated organisms, the data suggested a simple concentration mechanism, e.g., birds eat insects contaminated with TCDD contaminated soil particles. Biomagnification, i.e., orders-of-magnitude increases of residue through trophic levels, did not appear to occur...In-depth field studies, including anatomical, histological and ultrastructural examinations, spanning more than 50 generations of the Beachmouse, *Peromyscus polionotus*, demonstrated that continual exposure to soil concentrations of 0.1 to 1.5 parts-per-billion (ng/g) of TCDD, had minimal effects upon the health and reproduction of this species [19].

The species on the test area that may have some relevance to studies of food species collected in Southern Vietnam near “hotspots” were: Southern Meadowlark (liver 440 ppt); Crickets (18-26 ppt); Insect grubs (Coleoptera, 238 ppt); snakes (whole bodies, 420 ppt); Southern toad (whole bodies, 1,360 ppt); and Spotted Sunfish (skin, 4 ppt; gonads, 18 ppt; muscle 4 ppt; and gut, 85 ppt) [29]. The Spotted Sunfish was a bottom feeder and its visceral mass was comprised largely of silt and detritus [29]. As noted, the most intensive studies were of the Beachmouse, where the liver concentrations of TCDD ranged from 300 to 2,900 ppt in mature mice and the pelts (fur) ranged from 130-300 ppt [30]. Examination of nursing pups had liver concentrations of 500 ppt TCDD, and whole-body analysis of fetuses ranged from 40 to 150 ppt [30]. Clearly, the TCDD crossed the placenta into the fetuses, and was present in the milk of the nursing females [30].

Pham et al. reported similar observations of contaminated

ducks and fish that were consumed by the study populations at both Da Nang and Bien Hoa Airbases [31]. Thuong et al. quantified the fish and duck samples from Bien Hoa reporting levels of TCDD ranging from 12.2 to 288 ppt in fish samples, and 36 ppt in ducks [24]. Dwernychuk et al. reported on the analysis of fish (carp) fat and duck fat at the A-So former airbase having levels of 27 ppt and 67 ppt TCDD, respectively [32]. The implications of these data were that food sources at or adjacent to hotspots were the primary input of PCDD/Fs for human consumption in populations near these airbases. Mai in 2004 challenged these conclusions [33]. He noted that there were numerous sources of PCDD/Fs for those found in food samples collected from the Bien Hoa and Bien Hung markets, especially since these samples were likely food produced in other areas and shipped to the markets for sale [33]. Other sources noted by Mai included untreated industrial waste from industrial zones located within Bien Hoa City, to include industries such as paper, plastic, electric, electronics, and chemicals [33].

The Background review provided data suggesting that the exposures to 2,4,5-T- containing tactical herbicides sprayed on jungle forests and mangroves were likely minimal for Vietnamese located in villages near these combat areas. Although some tactical herbicides were sprayed in rural areas, the largest source of PCDD/Fs were likely from biomass-burning and open burning, especially at municipal dumpsites. In urban areas, the likely sources of PCDD/Fs were from industrial emissions. The data suggested that the most likely exposure to PCDD/Fs to Vietnamese were to those residents living adjacent to hotspots, specifically in former airbases where Agent Orange had been stored or loaded on RANCH HAND or US Army Chemical Corps aircraft. These hotspots were characterized by high soil and sediments levels of 2,3,7,8-TCDD that had persisted in spill sites for over 50 years. Movement of the PCDD/Fs into potential food sources appeared to be a major source of contamination for communities adjacent to the hotspots.

### Measuring Tcdd in Veteran and Vietnamese Populations

There have been numerous articles published on TCDD contamination in Vietnamese populations and on both US and Vietnamese veterans. The routes of exposure were important as to the 2,3,7,8-TCDD levels in adipose tissue, blood serum, and in mother’s milk.

### Vietnamese Populations

The first measurements of TCDD concentrations in Vietnam were of very limited value in determining the relative contribution of potential sources of dioxins. For example, in 10 out of 18 human milk samples collected in 7 sprayed areas of South Vietnam in 1970, Baughman and Meselson found TCDD levels from 132 to 1,450 ppt [34, 35]. They inferred that the “dioxin source” in the villages where samples were collected as being attributable to TCDD from Agent Orange, ignoring such sources as biomass-burning, open burning of wastes, industrial sources, and imported contaminated foods [34, 35].

Starting in the mid-1980s and continuing through 1990, Schecter and his associates began an ongoing series of studies on human adipose tissues that both addressed the question of identifying exposures to dioxin among Vietnamese, and demon-

strating the methodological complexities in assessing specific source contributions and routes of exposure when multiple PCDDs and PCDFs congeners were present in environmental matrices and adipose tissues [36, 37]. Measurements of PCDD/Fs in adipose required samples to be obtained by surgical biopsy to collect quantities for the detection of TCDD at the pg/g (ppt) level. [36]. Information about key demographic and life experience factors potentially affecting occupational or environmental exposures were not available, thereby limiting statistical analysis and epidemiology-based inferences of exposure sources. For example, the 60 adipose samples collected by Schecter et al. from 1984-1987 were from self-selected (the authors used the term “opportunistically”) Vietnamese cancer or other seriously ill patients from hospitals in Hanoi, Ho Chi Minh City, and Tay Ninh. Data on occupation and residence were not validated, and exposures were assumed to be from Agent Orange [36, 37]. Adipose tissue collected from a Hanoi hospital had levels ranging from ND to 2.9 ppt, while composite adipose tissues from Ho Chi Minh and Tay Ninh hospitals ranged from 2.0 to 103 ppt with a mean of 14.7 ppt. The value of the 103 ppt sample was considered an outlier [37]. Because the TCDD levels were lower in Hanoi, the authors assumed that the exposures were from Agent Orange, ignoring that there were fewer industrial sources in Hanoi as compared to those in Ho Chi Minh City [37].

A previous study comparing blood serum and adipose tissue TCDD levels showed that TCDD levels in serum were highly correlated with TCDD levels in adipose tissue, and thus provided a valid measure of TCDD levels in the human body [38].

Determining sources of exposures to dioxin and furans based on the patterns of PCDD/Fs observed in blood serum of Vietnamese was complicated. Many Vietnamese objected to giving more than a few milliliters of blood [39]. In a 1995 study of concentrations of PCDD/Fs in blood serum of Vietnamese, Schecter et al. reported the results of 43 dioxin analyses obtained by pooling samples from a set of 2,720 Vietnamese from the three major geographical regions of Vietnam: Northern Vietnam (n = 168), Central Vietnam (n = 490), and Southern Vietnam (n = 2,062) [39]. Although the authors attributed residence in a specific village or city for individuals from whom samples of blood were collected, it was difficult to know exactly where the patients had resided and for how long. All patients were volunteers and self-reported as having been sprayed with Agent Orange or were living in herbicide-sprayed areas. The results were as follows: blood serum samples from Northern Vietnam ranged from 1.2 to 6.1 ppt; samples from Central Vietnam ranged from 2.9 to 19 ppt; samples from Southern Vietnam ranged from 1.0 to 33 ppt TCDD [39]. Caution was warranted in assuming the samples were representative of concentrations of TCDD in blood serum of individuals from specific villages or cities, even more broadly representative of a region of from Agent Orange [39].

In the years from 1970 through 2005, the available information on concentrations of TCDD in human blood serum and adipose tissues, and the profiles of the relative concentrations of PCDD and PCDF congeners, were similar throughout Vietnam and other Asian countries including the Philippines, Cambodia, and India [26]. Indeed, these profiles were more representative

of municipal, industrial and atmospheric deposition exposures than they were to those observed in individuals who had been potentially exposed to the TCDD from Agent Orange. Thus, the determination of more extensive concentrations of 2,3,7,8-TCDD from Agent Orange in human tissues required studies of the hotspots in Southern Vietnam, or from studies of Veterans known to have been exposed to Agent Orange.

### Veteran Studies

In 1991, Kang et al. reported concentrations of PCDDs and PCDFs in adipose tissue from 36 US Vietnam veterans, a similar group of 79 non-Vietnam veterans, and 80 civilians [40]. Tissue samples were selected from the 8,000 archived tissues collected from the non-institutional US general population by the US Environmental Protection Agency, 1971-1987. The arithmetic-mean and standard deviation for concentrations of TCDD in adipose tissue of US Vietnam veterans, non-Vietnam veterans, and civilian controls were 13.4 ( $\pm$  7.4), 12.5 ( $\pm$  7.2), and 15.8 ( $\pm$  14.8) ppt on a lipid weight basis, respectively. The large standard deviation of the civilian controls was attributed to an outlier with a value of 106 ppt. Mean concentrations were not significantly different among the three groups with or without adjustment for individuals' age, body mass index, and specimen collection year [40].

The United States Air Force Health Study (AFHS) was undertaken in 1979 and involved the Air Force personnel that had been assigned to Operation RANCH HAND in Vietnam, 1961 – 1971 [41]. The study protocol was a 20-year matched cohort including 1261 RANCH HAND veterans and 19,101 comparison veterans who had served in Vietnam but had not been part of the RANCH HAND program [41]. Beginning in 1987 and through 2002, blood serum samples were obtained from both cohorts. In summary, 776 RANCH HAND personnel had a range of TCDD values from 0.4 to 618 ppt, and a median of 4.0 ppt. The match comparison 1,174 personnel had a range of TCDD values from 0.4 to 32 ppt, and a median of 4.0 ppt [41]. The authors noted that exposure to Agent Orange for RANCH HAND veterans ended in the late 1960s, and if a half-life of 7.6 years was assumed, roughly 2.5 – 3 half-lives had elapsed; the serum dioxin levels measured in 1987 would have been comparable to the low end of exposures measured in selected residents of Seveso, Italy [41, 42].

To place the AFHS in perspective with the Seveso experience, it was necessary to describe the Seveso Accident. The Seveso explosion occurred on 10 July 1976 and as a result, residents experienced among the highest human exposure concentrations to TCDD ever recorded [42]. The Chemical plant involved was manufacturing 2,4,5-trichlorophenol, and it was estimated that more than 15 to 30 kg of TCDD was disseminated over an area of 18 km<sup>2</sup> [42]. In 1996, the Seveso Women's Health Study was initiated. The Study involved 981 who were newborn to 40 years of age in 1976 and who had resided in the most exposed zones. Blood samples were collected and stored immediately after the accident. Within the cohort there was a wide range of TCDD concentrations in serum from 2.5 to 56,000 ppt, with a median of 56 ppt. For those females who were diagnosed with chloracne, the range of TCDD was from 144 to 5,700 ppt, with a median of 1,240 ppt TCDD [42].



In 2014, a study was reported of serum dioxin levels in Vietnamese men more than 40 years after herbicide spraying [43]. Two cohorts of men who were veterans of the war were recruited for the study in 2010 and 2011. The study area was the dioxin hotspot associated with the storage and loading of Agent Orange at Phu Cat Airbase located in Binh Dinh Province. Soil levels of TCDD were reported as high as 236,000 ppt at the site [43]. There were 97 men recruited from the hotspot area and 85 men from an unsprayed area in Northern Vietnam. The TEQs of PCDDs/PCDFs + PCBs ranged from 29 to 41.7 pg/g lipid for those men living closest to the hotspot area, while the TEQs of PCDDs/PCDFs+ PCBs of the men from Northern Vietnam was 13.6 pg/g lipid [43]. The authors acknowledged that the men living closest to the former airbase may have been exposed to both Agent Orange and other sources of dioxin-like compounds, e.g., combustion sources [43].

### Other Hotspot Studies

Dwernychuk et al. in 2002 reported on blood serum and mother's milk studies conducted in four villages in the A-luoi Valley of Southern Vietnam [32]. Three of the villages were located near the sites of former US Special Forces bases, and were considered dioxin hotspot, i.e., dioxin reservoir locations [32]. Pooled whole human blood analyses for PCDD/Fs for 159 males from the 4 villages ranged from 25 to 79 pg/g lipid with medians ranging from 48 to 50 pg/g, with serum from A So base males having higher levels than pooled male samples from the other three villages [32]. Human breast milk samples were collected from 16 lactating primiparous donor females with four from each of the four villages. The concentration of TCDD in mother's milk ranged from 1.4 to 19 ppt, with the median being 8.6 ppt TCDD [32]. Again, the highest concentrations of TCDD were found in donors from A So base. The authors provided excellent details on PCDD/FS concentrations in soils, sediments, food sources, serum, and mother's milk samples [32].

Manh et al. reported in 2015 on PCDD/Fs analyses in breast milk samples from three dioxin-contaminated hotspots in Vietnam [44]. Human breast milk samples were obtained from 143 lactating primiparous donors living in the vicinity of former US airbases at Bien Hoa (n = 51), Phu Cat (n = 23), and Da Nang (n = 69) [44]. The results indicated that mothers living close to these three dioxin hotspots were exposed to TCDD. Breast milk samples from donors living close to Bien Hoa showed higher levels of TCDD, with 18% > 5 pg/g lipid, while donors living close to Phu Cat showed lower TCDD levels, with none containing > 5 pg/g lipid. In Da Nang, TCDD levels in donors from Thanh Khe (close to the airbase, n = 43) were significantly higher than donors from Son Tra (far from the airbase, n = 26) [44]. Although TCDD levels in Bien Hoa were highest among these hotspots, the total PCDD/Fs levels (mean concentrations of 9.3 pg TEQ/g lipid) were lower than observed in Phu Cat (14.1 pg TEQ/g lipid), Thanh Khe (14.3 pg TEQ/g lipid) and Son Tra (13.9 pg TEQ/g lipid) [44].

Pham et al. in 2015 reported on serum TCDD levels in Vietnamese residents living near or on dioxin hotspots in Da Nang and Bien Hoa Airbases [31]. What added significantly to their studies were detailed analyses of demographic characteris-

tics, lifestyles, and dietary habits [31]. For studies in Da Nang, 140 individual whole blood samples were analyzed, while for Bien Hoa, 42 individual serum samples were analyzed. Bien Hoa participants had higher geometric mean serum TCDD concentrations (82.43 ppt lipid) than Da Nang (11.91 ppt lipid). The Bien Hoa participants' mean serum TEQ values were approximately 3 times higher (197.9 ppt) than Da Nang participants (54.11 ppt) [31]. Although all 17 PCDD/Fs congeners were detected, the levels were different between the two locations. Older age was associated with significantly higher serum dioxin levels; participants who had worked on or been inside Da Nang Airport had significantly higher serum dioxin levels; and fish farmers working on the Da Nang Airport, and their immediate families, were weakly associated with higher serum levels. For the Bien Hoa study, raising and harvesting ducks inside the Bien Hoa Airbase was weakly associated with serum dioxin levels, while fish farmers working inside the Airbase and their families had significantly higher serum dioxin levels than other participants in the sample population [31]. Participants in these studies were exposed to TCDD for a long time at both sites, with mean residencies of 22.1 and 17.1 years on or near the Da Nang and Bien Hoa sites, respectively [31]. A review of the available literature cited above, suggested that most Vietnamese have been continually exposed to low levels of PCDDs and PCDFs before, during, and after the Vietnam War. For this population, the historical use of Agent Orange has little relevance to current concentrations of dioxins in the environment, food and human tissues due to the failure of government to effectively regulate most pollution sources, including industrial effluents and open municipal burning of wastes that are currently occurring throughout Vietnam. These sources pose an ongoing public health concern and need to be addressed.

Although over 50 years have elapsed since Agent Orange and its associated dioxin contaminant was introduced into the Vietnamese environment, there is sufficient evidence to conclude that 2,3,7,8-TCDD from Agent Orange continues to enter the environment, the food chain, and the human population living in close proximity to "hotspots" where RANCH HAND and US Army Chemical Corps aircraft were loaded and cleaned, and where Agent Orange and other 2,4,5-T-containing tactical herbicides were stored. Currently these locations that have been identified and investigated including Da Nang Airport, the former airbases at Bien Hoa, Phu Cat, Nha Trang, and at three Special Forces bases in Central Vietnam. Other locations such as the airbases at Tuy Hoa and Phan Rang remain to be investigated [12]. No estimates have been provided on the number of residents living on or near these "hotspot" locations. From the investigations by Vietnamese and other scientists conducting community surveys, individual interviews, and the number of individuals requested to be blood or mother's milk donors, the population is not large, and as Dwernychuk et al. have stated "a manageable problem" [45]. Nevertheless, the public health of these individuals must be a concern for the government and peoples of Vietnam, and the question of the risks of diseases must be addressed. The actions that have now been undertaken to conduct major remediation programs at Da Nang Airport and Bien Hoa airbase will limit the future expo-

sure of nearby residents to TCDD and other PCDD/Fs.

### Results of Health Studies and Policies Related to Exposure to Agent Orange AND/OR TCDD

Under the Agent Orange Act of 1991, the United States Department of Veterans Affairs (DVA) recognized 14 diseases associated with alleged exposure from 9 January 1962 to 7 May 1975 to the tactical herbicides used in the Vietnam War, including Agent Orange and its associated dioxin (2,3,7,8-TCDD) contaminant [46]. The DVA has recognized the following cancers and other health problems as “*presumptive diseases*” associated with exposure to the tactical herbicides during military service: AL Amyloidosis; Chronic B-cell Leukemias; Chloracne; Diabetes Mellitus Type 2; Hodgkin’s Disease; Ischemic Heart Disease; Multiple Myeloma; Non-Hodgkins’s Lymphoma; Parkinson’s Disease; Peripheral Neuropathy, Early-Onset; Porphyria Cutanea Tarda; Prostate Cancer; Respiratory Cancers; and, Soft Tissue Sarcoma (other than osteosarcoma, chondrosarcoma, Kaposi’s sarcoma, or mesothelioma) [46]. The federal law that applies to the DVA assumes that the 14 diseases are a result of exposure to the tactical herbicides. This “*presumptive policy*” simplified the process for receiving health care and compensation, thus allowing the DVA to forego the normal requirements of proving that a disease began during military service [46]. The procedure for determining associated diseases was outlined in the Agent Orange Act of 1991; namely, the National Academy of Sciences’ Institute of Medicine (IOM) was to conduct biennial reviews (1994–2018) of the scientific literature related to the herbicides and TCDD and determined whether the evidence concluded a positive association for a disease. The Secretary of DVA then applied a standard as mandated by Congress and the courts to create a presumption of service connection for the disease to be associated with exposure to Agent Orange or the other tactical herbicides [46]. In making the final decision on whether an association existed, Congress and Courts mandated that any resolution of doubt should favor Vietnam veterans [46].

The Institute of Medicine’s reports of linkages between herbicides or TCDD and human disease were NOT based on cause and effect relationships, but rather on “statistical associations.” The term “statistical association” was not defined but was interpreted by IOM committees as evidence of an increased risk in as little as one study for which bias, confounding and chance could be reasonably dismissed without weighing contrary or conflicting evidence [47]. In the case of TCDD, the IOM relied on the precautionary principle since the magnitude of the potential damage that it might cause in humans was uncertain [47]. No IOM report has ever reported an actual causal connection between exposure and dose to a disease [48]. As noted by Young and Young in 2017, “*the reality is that the current Agent Orange Policy by the United States is based on politics, driven by public, veteran, Congressional actions, and Court decisions*” [46]. For example, the Department of Veterans Affairs used a ‘political’ definition for Vietnam veterans. If a US veteran served in Vietnam, even for one day, that individual was considered ‘exposed’ to Agent Orange and was legally eligible for presumptive compensation and health care for any of the 14 diseases [49]. The confusion for the Vietnam and

Vietnam-Era veterans is that they and the public now regard “association” as “proof” of a disease, and they truly believe that their diseases are caused by Agent Orange, and this is echoed by the media. The Department of Veterans Affairs with the support of the Congress should task the new Health and Medicine Division of the National Academy of Sciences, Engineering, and Medicine to validate a “cause and effect relationships” with exposure to TCDD for each of the 14 diseases. There are many excellent studies that are available to negate the associations recommended by the IOM.

The Air Force Health Study (AFHS) was undertaken to determine whether the Air Force personnel involved in Operation RANCH HAND had experienced adverse health effects resulting from that service [41]. The men responsible for loading the herbicide into the 3,785-liter Internal Defoliant Dispense were frequently in direct contact with the liquid Agent Orange. The range of TCDD in their blood serum in 1987 was 0.4 to 618 ppt TCDD, and by 2002, the values ranged from 0.4 to ~155 ppt. Considering the time from exposure for most RANCH HANDers, 1967-1969, to the final serum analysis, it was likely that the highest exposed individuals had levels of approximately 1,240 ppt TCDD. Thus, the RANCH HAND population was continuing contaminated with significant levels of 2,3,7,8-TCDD for more 30-35 years. The matched retrospective cohort design for the 20-year study required the investigation of over 300 health endpoints on multiple occasions [41]. The results of the AFHS did not provide evidence of disease in the RANCH HAND veterans caused by elevated levels of exposure to Agent Orange and its associated dioxin contaminant: “Given the lack of evidence of disease or health-related endpoints associated with exposures to TCDD in the RANCH HAND veterans, the available evidence does not seem to be consistent with a causal relationship between dioxin and the health endpoints studied in the AFHS” [41].

In the past few years, there have been many high quality and sophisticated studies conducted on Agent Orange and/or 2,3,7,8-TCDD. Chang et al in 2015 reported on a critical review of the epidemiology of 2,3,7,8-TCDD and lymphoid malignancies [50]. The results of their review: “Overall, a causal effect of TCDD on Non-Hodgkin lymphoma, Hodgkin lymphoma, and multiple myeloma, or subtypes of these malignancies has not been established” [50]. Chang et al. in 2014 had reported that “Overall, epidemiologic research offers no consistent or convincing evidence of a causal relationship between exposure to Agent Orange or TCDD and prostate cancer” [51]. Krishnamurthy et al. in 2015 reported on whether Agent Orange and its associated TCDD was the cause of hepatocellular cancer in US veterans [52]. They found no significant association between Agent Orange and hepatocellular cancer, although larger studies were needed to evaluate this further in the Vietnam-Era veterans [52]. Goodman et al. in 2015 examined the dose-response relationship between serum 2,3,7,8-TCDD and diabetes mellitus (DM) [53]. Their conclusions: “Considering the discrepancy of results for low current versus high past TCDD levels, the available data do not indicate that increasing TCDD exposure is associated with an increased risk of DM” [53]. Kim et al. in 2012 described the clinical outcome for acute coronary syndrome (ACS) in Korean veterans who had served



in the Vietnam War and who had been exposed to Agent Orange. The authors stated: “In conclusion, despite the higher incidence of hypertension and hyperlipidemia in patients exposed to TCDD, exposure to TCDD did not affect the severity of cardiovascular disease and the rate of MACEs (major adverse cardiovascular events) in acute coronary syndrome” [54]. Sun et al. in 2015 reported on a study of the relationship between levels of prostate-specific antigen (PSA) and dioxins in Vietnamese men [55]. The focus of the study was on Vietnamese men who resided in the dioxin hotspot at Phu Cat and on men from a non-sprayed area. The findings suggested that PSA levels in Vietnamese men were not associated with levels of dioxins or steroid hormones in either location [55]. Lastly, Patterson et al. published an article in 2015 on skin diseases associated with Agent Orange exposures [56]. They found that Vietnam veteran’s self-reported chloracne and other skin ailments from alleged exposure in Vietnam were frequently communicated to physicians in Veteran Administration and military hospitals, yet dermatologic examination findings over 20 years after possible herbicide exposure did not support the self-reported symptoms [56].

### The Validity of Studies of Birth Defects and Exposure to Agent Orange and Association TCDD

One of the most controversial issues associated with the spraying of Agent Orange in Vietnam has been the issue of birth defects. The Institute of Medicine’s report, *Veterans and Agent Orange, Update 1996* concluded that there was limited or suggestive evidence of an association between exposure to herbicides used in Vietnam and spina bifida in children of Vietnam veterans [47]. Accordingly, the Department of Veterans Affairs under the Agent Orange Act of 1991, recognized that the only birth defect associated with Vietnam veterans and exposure to Agent Orange was Spina Bifida, and that eligible children may receive VA benefits [49]. In the latest Institute of Medicine’s *Update 2018 Report* (now known as the Health and Medicine Division of the National Academy of Sciences, Engineering, and Medicine) stated: There is inadequate or insufficient evidence to determine whether there is an association between exposure to 2,4-D, 2,4,5-T, TCDD, picloram, or cacodylic acid and endometriosis; semen quality; infertility; spontaneous abortion; stillbirth; late fetal, neonatal, or infant death; low birth weight or preterm delivery; birth defects other than spina bifida; childhood cancers; or diseases in mature offspring or later generations. There is limited or suggestive evidence of no association between paternal exposure to TCDD and spontaneous abortion [49].

Eakenazi et al. have released the 2018 update: *The Seveso accident: a look at 40 years of health research and beyond* [57]. The article notes that early Seveso data suggested no association of residents from Zone A, the most highly contaminated TCDD zone, with birth defects. In 1983, again no increased risks were reported for all birth defects in any of the exposed zones. The Seveso Women’s Health Study established in 1996 has to date not produced evidence of any positive association of TCDD exposure and birth defects; no Spina Bifida was found [57]. In an early publication by Charnley and Kimbrough, they noted that studies of children indicated that exposure of general

populations to low levels of 2,3,7,8-TCDD did not result in any clinical evidence of disease, although accidental exposure to higher levels either before or after birth have led to minor developmental deficits [58]. Breast-fed infants have higher exposures than formula-fed infants, but studies consistently find that breast-fed infants perform better on developmental neurologic tests. Because body burdens and environmental levels of PCDD/Fs continue to decline, it is unlikely that children alive today will experience exposures that are injurious to their health [58]. The available scientific data provide evidence that the Public Health of Vietnamese continues to improve, but that improvement is most related to regulatory actions of Vietnam’s government to reduce pollution and increase the quality of potable water while reducing atmospheric emissions. The use of Agent Orange during the Vietnam War clearly impacted the ecology of Vietnam. The remaining hotspots of PCDD/Fs provide evidence that 2,3,7,8-TCDD continues to be present in residents living on or near these hotspots, but as to the claims of the horror stories of large numbers of human deaths and gross structural birth defects, the evidence is absent.

### The Politics of Agent Orange and Dioxin

Jon Franklin, Professor, School of Journalism and Communication, University of Oregon, Eugene Oregon reflected on his experience in 1980 when tasked by the editor of *The Baltimore Sun* to cover the developing Agent Orange Scandal [59]. “People were saying the defoliant had not only poisoned the Vietnamese countryside but our own soldiers. As a veteran, I was outraged. Let me remind you of the headlines. There had been hearings on Capitol Hill in which it was charged that the children of Vietnam veterans (and Vietnamese) had high rates of birth defects, and veterans (and Vietnamese) were dying of cancer, brain tumors and unexplained episodes of violence...So you can imagine my horror when ...I couldn’t substantiate anything at all. Scientific reality was one thing and social reality was something else. What was happening was all too clear. The media, which once prided itself on its truth and accuracy, now flourished on lies, half-truths and illusions about environmental poisons” [59].

An example of propaganda that has been disseminated world-wide is the following. In April 2012, Marie-Helene Lavallard, a member of the Franco-Vietnamese Friendship Association published online “A Chemical War without End: Agent Orange in Vietnam” [60]. She described Agent Orange and its dioxin contaminant as “one of the most violent and most indestructible poisons known”. She continued: “Millions of Vietnamese, soldiers, civilians, men, women, children were injured by the spreading of Agent Orange/dioxin. Ten of thousands died on the spot. Two to four million survivors according to the Vietnamese Red Cross present serious pathologies including cancer, leukemia, diabetes, and skin diseases... and often give birth to severely handicapped children” [60]. How can the public not believe these lies, when Wikipedia, the free Internet encyclopedia has described Agent Orange when the same verbiage?

In 2013, Michael Gough noted that some \$3 billion have been spent on researching possible health effects from dioxin, and the results have shown that the risks were overstated; even

the US Environmental Protection Agency's Scientific Advisory Board had concluded that the evidence that dioxin caused human cancer and other diseases was unconvincing [61]. Nevertheless, in 1988, Congressional leaders faced a dilemma as to how to satisfy concerns about Agent Orange by the veteran community, the public and the politicians. In their minds, the political answer was to do the "right thing" by providing appropriate health care and compensation, hence, the Agent Orange Act of 1991 [61].

### Conclusion

The Agent Orange Act of 1991 may be the solution, albeit one ignoring the science, for the United States, but what Public Health solution is there for Vietnam? In 2007, at the Agent Orange and Dioxin Remediation Workshop held in Hanoi [12], the American Ambassador Michael W. Marine stated: "I do not accept the term "victims of Agent Orange", the US's humanitarian assistance to the disabled in Vietnam is not based on evaluation of causes of disability." Members of Congress have recognized that the legacy of Agent Orange still impacts our diplomatic relationships. After more than 50 years since the use of Agent Orange in Vietnam, the US must now fulfill its obligation as a responsible global citizen by helping to contain the dioxin hotspots and by providing continued humanitarian assistance to Vietnam. Indeed, the United States just completed a five-year, \$110 million program that cleaned soil contaminated by Agent Orange at Da Nang Airport, and the US Agency for International Development, which has been overseeing the project, is now expected to provide \$390 million for the cleanup at Bien Hoa Airbase [62]. These actions will assist Vietnam in reducing their Public Health concerns over Agent Orange and its associated dioxin.

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To cite this article: Young AL. A Review of Public Health in Vietnam: 50 Years after Agent Orange was Sprayed. *Health Educ Public Health*. 2019; 2:2.