Dioxins and Dibenzofurans in Adipose Tissue of US Vietnam Veterans and Controls

ABSTRACT

The primary reason for concern about the adverse effects of exposure to Agent Orange is attributable to its toxic contaminant, 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) or dioxin. We studied adipose tissues from 36 Vietnam veterans, a similar group of 79 non-Vietnam veterans, and 80 civilians; the tissue specimens were selected from the 8,000 archived tissues collected from the non-institutionalized general population by the US Environmental Protection Agency. The geometric mean (± standard deviation) dioxin levels in adipose tissue for Vietnam veterans, non-Vietnam veterans, and civilian controls were 11.7 (±1.7), 10.9 (±1.7), and 12.4 (±1.9) parts per trillion on a lipid weight basis, respectively. The mean levels for these groups were not significantly different from each other with or without adjustment for age of individuals, body mass index, and specimen collection year. In addition, none of the surrogate measures of Agent Orange exposure such as military branch, service within specific geographic region, military occupation, and troop location in relation to recorded Agent Orange spray was associated with the dioxin levels in adipose tissue of Vietnam veterans. Our results suggest that heavy exposure to Agent Orange or dioxin for most US troops was unlikely. (*Am J Public Health* 1991; 81:344–349)

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Introduction

From 1965 to 1970, the US Air Force sprayed more than 40 million liters of Agent Orange in South Vietnam.[1] Approximately two million American soldiers served in Vietnam during this period. Agent Orange was the name used for a phenoxy herbicide consisting of a mixture of 2,4-dichloro-phenoxyacetic acid (2,4-D) and 2,4,5-trichlorophenoxyacetic acid (2,4,5-T). The 2,4,5-T contained 1-50 parts per million of the contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), also known as dioxin. Dioxin is extremely toxic in laboratory animals, promotes liver tumors in rats, and is teratogenic in mice.[1,2] The two parent herbicides, 2,4-D and 2,4,5-T are very short-lived in the human body and do not persist in the environment.[3] Because of these characteristics and their relatively low toxicity in humans, attention has been focused on the highly persistent and toxic chemical, dioxin.

Many Vietnam veterans believe that they were heavily exposed to Agent Orange and that the exposure is responsible for health problems such as skin rashes, rare types of cancer, and birth defects of their children. Since 1979, 200,000 Vietnam veterans have come to Department of Veterans Affairs (VA) hospitals for an Agent Orange Registry medical examination because of concerns about exposure to Agent Orange.

Two questions are paramount in dealing with the veterans' concerns. The first is whether dioxin causes birth defects, immune deficiencies, cancers, or other chronic health problems in humans. The second is whether Vietnam veterans, in general, received substantial exposure to dioxin while they served in Vietnam. This study addresses the second question.

Dioxin accumulates preferentially in the body fat of animals and man. Its half-life in humans is estimated at five to 11 years.[4–7] Among the many 2,3,7,8-substituted polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) found in the environment and in samples of human adipose tissue, only 2,3,7,8-TCDD or dioxin was present in Agent Orange.[7] Dioxin levels in adipose tissues could, therefore, serve as a biological marker of exposure to Agent Orange. In fact, several studies have reported that even after approximately 20 years, dioxin levels were elevated markedly in the adipose tissue or blood serum of Vietnam veterans who handled Agent Orange.[6–8] More recently, however, the Centers for Disease Control (CDC) reported that current serum dioxin levels of Army Vietnam combat troops did not differ significantly from those of non-Vietnam veterans and that dioxin levels in Vietnam veterans did not increase with increased exposure levels estimated from military records.[9] This study was criti-

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cized on the basis that blood specimens taken almost 20 years after exposure could not represent what happened in Vietnam and that veterans selected for the study were limited to Army enlisted men who served in only one geographic region of Vietnam.[10]

The purpose of our study was two-fold: 1) to determine if a group of individuals with military service in Vietnam have significantly higher levels of dioxin in adipose tissue than either a similar group of non-Vietnam veterans or civilian peers, and 2) to determine if dioxin levels in adipose tissue were associated with specific demographic and military service characteristics.

Methods

Selection of Study Subjects

Our study used adipose tissue specimens that had been collected from the general population by the US Environmental Protection Agency (EPA). The EPA has conducted the National Human Adipose Tissue Survey (NHATS) since 1970 to monitor the human body burden of pesticides and other selected chemicals. Up to 1,000 adipose tissue specimens have been collected annually from pathologists and medical examiners across the country and analyzed by the EPA for the selected chemicals. After analysis, the unused tissue specimens were sent to a central facility to be stored at 0°C to −20°C.

The NHATS utilized a probability sample of the Standard Metropolitan Statistical Areas (SMSA) that is designated to represent a sample of the US population in terms of age, sex, and race.[11] The target population for the NHATS program was all non-institutionalized persons in the conterminous US. However, due to the invasive nature of collecting adipose tissue samples, the sampling population was limited to individuals who died from external causes (90 percent) and surgical patients (10 percent). Within each SMSA, hospitals or medical examiners were identified and asked to contribute tissue specimens according to the design specifications of age (0–14 years, 15–44 years, 45+ years), sex and race (White, non-White). Since the vast majority of Vietnam veterans were men born between 1936 and 1954, this study was restricted to specimens from men born in that period.

An Inventory File was created for the 8,000 specimens that were recorded to have an adequate amount of tissue. It was found that 528 of 8,000 specimens were from males born between 1936 and 1954. The hospitals or medical examiners who originally collected the 528 specimens were recontacted to obtain enough identifying information on the donors to determine their military service status. The collection effort yielded information for 494 or 94 percent of the 528 specimens.

The military service status for these 494 men, including any Vietnam service, was determined by reviewing records archived at the National Personnel Records Center (NPRC) in St. Louis and military records maintained at other locations. From this effort, 134 men (or 27 percent of 494) were initially found to have served in the military, 40 of whom served in Vietnam. According to the 1980 Census, one would expect about 34 percent of men in this age group to be veterans. All 40 Vietnam veterans were selected for the study. From the 94 remaining veterans, 80 were selected randomly for the non-Vietnam comparison group. Two civilians were closely matched to each Vietnam veteran by birth year (±two years) and sample collection year (±two years). Age and sample collection year were considered important matching variables because of probable accumulation of dioxin in the body with each year of exposure and the possibility of degradation of fat or dioxin while in storage. All adipose tissue specimens were analyzed during 1987. Demographic data were taken from the NHATS file and the official death certificates. Body mass index (BMI) was calculated from weight and height as follows: BMI = weight in Kg/height in m^2.

Determination of Opportunity for Agent Orange Exposure

A precise estimate of the exposure of each Vietnam veteran to Agent Orange is not considered feasible based on either military records or self-reported data. In this study the probable opportunity for exposure was determined from the following: branch of service, military occupational specialty code (MOSC), and location of the individual's unit in Vietnam in relation to recorded Agent Orange spray. Ground troops in Vietnam might have had a higher probability of contact with Agent Orange than other Vietnam veterans due to the nature of their military operations. Ground troops engaged in combat were assumed more likely to be in herbicide-sprayed areas. As another surrogate for exposure, a veteran's military unit was assigned to one of the four broad military regions in Vietnam. According to the records of the US Air Force Ranch Hand Operation, 20 million liters of Agent Orange were sprayed in Military Region III from 1965 to 1970. During the same period, Military Regions II, I, and IV received 9.5, 8.3, and 4.5 million liters, respectively. Finally, troop locations (company size units) were determined on a 100-meter grid map of Vietnam at intervals of 90 days or less. Computer matching of troop location with respect to time and distance from recorded herbicide spray tracts was carried out using the HERBS tape and services HERB tape databases. The HERBS tape contained information on most of the herbicide missions flown by fixed-wing aircraft from 1965 to 1971, and on crop destruction missions flown by helicopter between 1968 and 1971. The tape contained information on the type of herbicide, gallons, dates, and where spray runs started and ended. Services HERBS Tape prepared by US Army and Joint Services Environmental Support Group identified and documented an additional six million liters of herbicide sprayed mainly by Army personnel around the perimeter of base camps, fire bases, air bases, and other fixed military installations. The opportunity for Agent Orange exposure was determined in two ways: an individual's company was ever located either within two kilometers (km) of a recorded Agent Orange spray tract within three days of application or within eight km of a spray tract within 90 days application.

Laboratory Analysis

Specimens from the three groups were assigned randomly to one of 20 batches. Each batch typically consisted of 10 study specimens and four quality control (QC) samples. The QC samples provided data on method accuracy and precision. In addition, the external quality control audit samples were prepared by another laboratory and incorporated as blind samples into the various batches. Study specimens and QC samples were coded with a unique laboratory number and submitted to the analysts as blind samples. Following sample cleanup and preparation, instrumental analyses were achieved using a high resolution gas chromatograph coupled to a high resolution double-focusing mass spectrometer. Overall method accuracy and precision for 2,3,7,8-TCDD analyses of spiked lipid samples were 113 percent recovery and 8.8 percent coefficient of variation.

The analytical protocol provided for the detection and quantitative determination of 17 congeners of 2,3,7,8-substituted

TABLE 1—Distribution of 2,3,7,8-TCDD Levels in Adipose Tissue by Military Service Status, in pg/g of the Total Extractable Lipid (ppt)

Status	N	Arithmetic Mean ± SD[a]	Geometric Mean	Percentile 25th	50th (median)	75th	90th	95th
Vietnam Veterans	36[b]	13.4 ± 7.4	11.7	7.8	10.0	17.3	26.8	30.4
Non-Vietnam Veterans	79[c]	12.5 ± 7.2	10.9	7.6	11.4	14.8	19.8	25.3
Civilians	80	15.8 ± 14.5[d]	12.4	7.9	11.8	18.0	30.5	43.4

[a]Standard deviation
[b]Four of the 40 men initially classified as having served in Vietnam were excluded from analysis because two veterans' specimens had less than 20% extractable lipid content, one veteran did not have adequate amount of tissue for analysis, and one veteran served only in Thailand.
[c]One of the 80 men initially classified as having served in the military was excluded because his military service could not be documented unequivocally.
[d]The large standard deviation was attributed to an outlier with a value of 106. The value was verified. The occupational history of this individual is unknown. He was listed as a "laborer" on his death certificate. Analysis conducted without this value resulted in an arithmetic mean of 14.7 (± 10.3) and a geometric mean of 12.2. There was still no statistically significant difference between the groups (p = 0.49).

polychlorinated dibenzo-p-dioxins (PCDD) and dibenzofurans (PCDF). The minimum measurable concentration ranged from 1 picogram per gram (pg/g) for 2,3,7,8-TCDD and 2,3,7,8-TCDF, to 5 pg/g for OCDD and OCDF based on a 10 gram aliquot of human adipose tissue. This protocol was evaluated for method performance prior to being used in this study.[12]

Statistical Considerations

Multiple comparisons and testing for differences were done by using the F test in one way analysis of variance (ANOVA) and analysis of covariance with adjustments for demographic variables such as age, collection year, and body mass index.[13] A paired t-test was conducted to compare the means of Vietnam veterans with their matched civilian controls.[14] In all analyses, the dioxin values were transformed to a natural logarithmic scale because the dioxin values were found to have approximately log-normal distributions in this study and another study.[15]

A stepwise linear regression model was also used to determine whether dioxin levels were associated with demographic and military service characteristics.[16] Factors considered *a priori* as covariates were age, adipose tissue sample collection year, race, and body mass index. A regression model specific to Vietnam veterans included such covariates as military occupation, calendar year of tour in Vietnam, geographic region in Vietnam, time of and distance from recorded Agent Orange spray, and adipose tissue sample collection year.

Results

Table 1 presents the arithmetic and geometric means, and various percentile values for dioxin for the three study groups. One-way analysis of variance did not demonstrate differences in the mean dioxin levels among groups (p = 0.35). Analysis of covariance, testing the effect of Vietnam service on dioxin levels after adjusting for age, sample collection year, or body mass index did not indicate an association between service in Vietnam and dioxin levels. A paired t-test between Vietnam veterans and their matched civilian pairs did not show a difference in mean dioxin levels (95% confidence interval for the difference between two means = −1.32,1.16).

For Vietnam veterans, dioxin levels were also evaluated by four factors related to the likelihood of Agent Orange exposure (Table 2). None of the surrogate measures of Agent Orange exposure was associated with the dioxin levels in adipose tissue of Vietnam veterans. Furthermore, the mean dioxin level of seven Vietnam veterans whose specimens were taken within four years (less than one dioxin half-life estimated for humans) since their last service in Vietnam were compared to 19 non-Vietnam veterans whose tissue specimen were collected on or before 1974 (the last sample collection year for the seven Vietnam veterans), and also to their matched civilian pairs. The geometric mean dioxin levels (± standard deviation) for the Vietnam group (n = 7), non-Vietnam veteran group (n = 19), and civilian controls (n = 14) were at the levels of 16.6 (±1.6), 15.5 (±1.5), and 18.4 (±1.6) parts per trillion (ppt), respectively. There was no difference among the means (p = 0.56). The 95% CI for a mean dioxin difference between Vietnam veterans and civilian controls were −1.59 and 1.31. Stepwise linear regression analysis for 36 Vietnam veterans indicated that Vietnam service characteristics could account for only 14 percent of the variation in dioxin levels (p = 0.3), whereas collection year alone could account for 21 percent of variance (p = 0.005). Five other 2,3,7,8-substituted dioxins and 10 other dibenzofurans were measured and their mean levels were calculated from specimens with levels above the detection limit (Table 3). There were no group differences in the mean level of any of the dioxin congeners. The differences in TCDD levels in the three groups were also evaluated while adjusting for levels of other 2,3,7,8-substituted dioxin congeners not found in Agent Orange by a stepwise multiple regression technique. TCDD levels were taken as the dependent variable and Vietnam service status and other dioxin congener levels as independent variables. There was a significant association between the TCDD levels and the levels of PCDD congeners ($R^2 = 0.54$, p = 0.0001), but not with the Vietnam service status ($R^2 = 0.0025$, p = 0.3).

The levels of dioxins increased with an increase in the number of chlorine except for 1,2,3,7,8,9-HxCDD. Levels of dibenzofurans were always lower than their dioxin counterparts. In each study group, the levels of dioxin tended to be inversely related to the specimen collection year, i.e. the earlier the collection year, the higher the dioxin levels (p = 0.0001, n = 195).

Discussion

In this study, military service in Vietnam was not associated with elevated dioxin levels in adipose tissue with or without adjustment for demographic variables. In addition, no Vietnam service characteristics measured singularly or in combination was a good predictor of dioxin levels in adipose tissue. There were no consistent trends in the dioxin levels ac-

TABLE 2—Geometric Mean 2,3,7,8-TCDD Levels in Adipose Tissue by Vietnam Service Characteristics, in pg/g of the Total Extractable Lipid (ppt)

Service Characteristics	No. of Vietnam Veterans	2,3,7,8-TCDD Mean	2,3,7,8-TCDD SD[a]
Branch			
Army	20	11.6	1.7
Marine	6	12.3	1.8
Air Force	1	6.7	
Navy	9	12.4	1.5
MOSC[b]			
Non-Combat	24	11.1	1.7
Combat	12	13.1	1.6
Military Region[c]			
I Corp	11	12.4	1.6
II Corp	4	6.9	1.3
III Corp	9	11.9	1.9
IV Corp	3	14.3	1.9
Sea Duty	8	13.3	1.4
Time and Distance From[d] Recorded Herbicide Spray			
a. 3 days/2 KM			
no	31	11.5	1.7
yes	4	14.3	1.6
b. 90 days/8 KM			
no	16	11.8	1.5
yes	19	11.8	1.8

[a]Standard deviation
[b]Military Occupation Specialty Code (MOSC)
[c]Missing Military Region information for one veteran
[d]Missing information on time and distance from recorded herbicide spray for one veteran due to unknown unit location

cording to the surrogate measures of Agent Orange exposure. The mean levels of dioxin did rise slightly with combat MOSC and having been within three days/two km of sprayed areas. But the other two surrogates, branch of service and service location in Vietnam, were not associated with the dioxin levels. The small magnitude of the mean difference and the variation within each group suggest that the small difference in mean values could have been easily due to the sampling and measurement variation.

Analyses of adipose tissue from the general population of industrialized countries have indicated the presence of a number of 2,3,7,8-substituted dioxins and dibenzofurans at ppt level.[17–21] These dioxins and dibenzofurans could have originated from a number of sources: incineration of municipal waste and wood products; manufacturing, use and disposal of pesticides, herbicides and wood preservatives; and PCBs from electric transformers and capacitors. Because 2,3,7,8-TCDD was the only congener found in Agent Orange as a contaminant[1,7] knowing the levels of other dioxins and dibenzofurans would help determine whether TCDD levels in adipose tissue of Vietnam veterans were the result of Agent Orange exposure in Vietnam or exposure to other sources. For example, if most dioxins and dibenzofurans as well as 2,3,7,8-TCDD levels were found to be elevated among Vietnam veterans, contribution from sources other than Agent Orange should not be ruled out. However, if only the 2,3,7,8-TCDD level remained elevated and other PCDD levels were comparable to the comparison groups, Agent Orange would be considered as the contributor. There were no group differences in the mean levels of any PCDD congeners. The lack of group differences suggested that sources other than Agent Orange may have contributed to the Vietnam veterans' levels of 2,3,7,8-TCDD. In a study by Kahn, *et al*,[7] of 10 Vietnam veterans with heavy potential for exposure to Agent Orange (e.g. Ranch Hand personnel, Army Chemical Corps specialists), the levels of 12 other 2,3,7,8-substituted dioxins and dibenzofurans were similar to the levels found among 10 Vietnam veteran controls and seven non-Vietnam veteran controls. Only 2,3,7,8-TCDD levels were elevated approximately 10-fold among Vietnam veterans with a heavy exposure potential.

This study may have failed to detect a small difference in mean dioxin levels among groups because of the relatively small sample size. The study had an adequate statistical power (90 percent) to detect a mean difference of 5 ppt or more between groups. Elimination of dioxin from the body after Vietnam service is an unlikely explanation because dioxin levels of seven Vietnam veterans whose specimens were taken within four years from their return from Vietnam (which was considerably less than the estimated half-life of dioxin in humans) were not significantly different from their appropriate comparison groups.

Although the NHATS sampling scheme was designed to collect a representative sample of the SMSA in terms of age, sex and race, subjects selected for the study may not have represented their respective groups for several reasons. First, over 90 percent of the NHATS sample were collected from deceased persons whose death in most instances were due to traumatic injury. Second, tissue samples for this study were selected from the archived NHATS specimens rather than original NHATS samples. Third, 6 percent of the men who were eligible for the study had to be excluded because of missing personal identifiers. Despite these problems, demographic and military characteristics of the Vietnam veterans selected for the study did not differ substantially from the overall Vietnam veteran population. They were predominantly white (75 percent), draft eligible during the Vietnam war (age 18 to 25), enlisted men (89 percent), who served in the Army and Marine Corps (72 percent) with military occupational specialties related to combat support roles (67 percent). None of the Vietnam veterans in the study had a record of routinely handling or spraying Agent Orange.

The mean background levels of dioxin reported here were generally higher than the values reported by others.[6–9] It is unlikely that this difference is due solely to an interlaboratory variation. Our laboratory (Midwest Research Institute) participated in an interlaboratory validation study for dioxin measurement and produced a satisfactory result. Furthermore, external quality control audit samples prepared by the Battelle Columbus Division were incorporated as blind samples into the various batches and analyzed for dioxin and dibenzofuran congeners. The analytical results were found to be acceptable. We believe that the difference in sampling years between this study and several other studies could account for the higher values reported in our study. We found that the dioxin levels in adipose tis-

TABLE 3—Arithmetic Mean Levels of Dioxins and Furans Detected in Adipose Tissue by Military Service Status, in pg/g of the Total Extractable Lipid (ppt)

Chemicals	Status		
	Vietnam Veterans	Non-Vietnam Veterans	Civilians
Dioxins			
2378-TCDD	13.4 (36)*	12.5 (79)	15.8 (80)
12378-PeCDD	20.6 (36)	18.3 (78)	18.3 (80)
123478/123678-HxCDD	170.4 (36)	152.9 (79)	165.1 (80)
123789-HxCDD	19.4 (35)	17.2 (79)	17.9 (79)
1234678-HpCDD	276.2 (36)	244.6 (79)	300.3 (80)
OCDD	1261.8 (36)	1108.9 (79)	1392.9 (80)
Furans			
2378-TCDF	2.9 (25)	2.4 (52)	3.3 (51)
12378-PeCDF	1.7 (8)	1.1 (17)	1.9 (16)
23478-PeCDF	23.1 (35)	22.2 (78)	23.3 (80)
123478-HxCDF	21.5 (36)	19.3 (78)	23.2 (79)
123678-HxCDF	10.7 (34)	9.9 (77)	12.0 (79)
234678-HxCDF	3.8 (26)	3.2 (73)	3.6 (78)
123789-HxCDF	1.5 (3)	0.9 (2)	0.9 (4)
1234678-HpCDF	37.4 (36)	32.9 (79)	39.1 (80)
1234789-HpCDF	2.2 (14)	1.9 (35)	2.2 (41)
OCDF	3.6 (27)	4.5 (54)	3.4 (60)

*the number in parentheses represent the number of cases in that category

sue were significantly associated with the sample collection year ($p = 0.0001$), the earlier the collection year, the higher the levels of dioxin irrespective of veteran status. In fact, this general trend was observed for other dioxins. The median sample collection year in our study was 1978, whereas in other studies the specimens were mostly collected in mid 1980s or later. The observed dioxin decline from 1971 to 1982 is consistent with the general trend for chlorinated hydrocarbon chemical compounds in human adipose tissue. The US EPA's National Human Adipose Tissue Survey Program indicates that the median levels of BHC, HCB, and PCB had been steadily decreasing over time between 1970 and 1983.[11] In Sweden, the levels of dioxin and dibenzofurans in human milk decreased significantly from 1972 to 1985.[22] The Swedish authors attributed the decline to the reduction in use of certain organochlorine compounds such as PCBs, PCP and 2,4,5-T. A study involving a large sample of specimens representative of the US population will be needed to confirm this observation.

We concluded that the results of our study did not support the hypothesis that most US troops were heavily exposed to dioxin in Vietnam. Furthermore, none of the surrogate measures of Agent Orange exposure based on military service characteristics was associated with the dioxin levels in adipose tissue of Vietnam veterans. These results are consistent with those of CDC[9] and not inconsistent with Kahn, *et al*,[7] and Schecter, *et al*.[8] □

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Kellogg Grant to Strengthen Community Health Services

A grant of $1.2 million has been awarded to the Colleges of Nursing and Medicine at the University of Illinois at Chicago (UIC) by the W. K. Kellogg Foundation to strengthen community-based health service. Emphasizing leadership training for project directors, community members and health care professionals, the project will bring together community residents and health care professionals to create partnerships in advancing leadership in community-based primary health care.

Both classroom instruction and practical hands-on experience at community-based primary health care sites will be offered. UIC faculty and community leaders will serve as mentors for program participants.

For more information, contact Susan Poslusny, Office of Leadership for Primary Health Care in Urban Communities, College of Nursing (m/c 802), the University of Illinois at Chicago, 845 S. Damen, Chicago, IL 60612; (312) 413-0068.