DIBENZODIOXIN AND DIBENZOFURAN LEVELS IN THREE GROUPS OF VIET NAM VETERANS WHO DID NOT TAKE PART IN AGENT ORANGE SPRAYING

Peter C. Kahn, William Lewis, Therese Ghent-Guenther,
Department of Biochemistry & Microbiology
328 Lipman Hall, Rutgers University
New Brunswick, NJ 08903, U.S.A.

Marianne Hansson, and Christoffer Rappe
Institute of Environmental Chemistry
University of Umeå
S-901 87, Umeå, Sweden

Henry Velez
19-03 Maple Avenue
Fair Lawn, NJ 07410, U,S,A,

Wayne P. Wilson
New Jersey Agent Orange Commission
Box 1717
Trenton, NJ 08607-1717, U.S.A.

Michael Gochfeld
Department of Environmental and Community Medicine
University of Medicine and Dentistry of New Jersey -
Robert Wood Johnson Medical School
675 Hoes Lane
Piscataway, NJ 08854, U.S.A.

Abstract

Fifty-five U.S. Viet Nam veterans comprising twenty marines, twenty members of patrol boat crews, and fifteen army troops, all of whom were exposed to Agent Orange during the war, as well as fifteen unexposed Viet Nam veterans were examined for blood dioxin and dibenzofuran congener levels.

Introduction

Several groups of persons occupationally or accidentally exposed to chemicals containing 2,3,7,8-TCDD have been shown to carry elevated body burdens. Even workers exposed thirty years earlier still show adipose tissue levels well above those seen in the general population of the industrial world (Nygren, et al. 1986; Schecter and Ryan, 1988), for, as has been learned over the last several years, dioxin is eliminated from the human body slowly (Poiger and Schlatter, 1986; Pirkle, et al., 1989). Patterson, et al. (1986, 1988) found adipose levels as high as 750 pg/g in

factory workers who had been involved in trichlorophenol production some years before sampling. Similar results were seen by Fingerhut, et al. (1989).

In Viet Nam veterans Gross, et al. (1984), Kahn, et al. (1988), and Pirkle et al. (1989) have examined men who handled Agent Orange in the course of their wartime duties and found dioxin levels elevated above those of controls. Thereafter, the Centers for Disease Control (CDC, 1988) studied veterans who did not themselves handle herbicides, finding no elevation of body burden above controls. The ascertainment of exposure in the CDC's study of is uncertain, however. In view of the continuing interest in the possible medical consequences of wartime service, we remain concerned over the best methodology to use in inferring herbicide exposure from military records. We therefore chose three groups of veterans - one from the Army, one from the Marines, and one from the Navy - whom we believed to have been exposed to Agent Orange and assayed them for blood levels of dioxins and dibenzofurans.

Methods

Military Units

Army: Between October, 1967, and December, 1968, the Second Battalion, Eighth Cavalry, First Cavalry Division (2/8) served in a part of Military Region III (III Corps) called the "Parrot's Beak" northwest of Ho Chi Minh City (formerly Saigon) near the Cambodian border. The area is in Tay Ninh Province. Records of the Joint Services Environmental Support Group of the U. S. Department of Defense suggested that this unit's zone of operations had had a history of heavy herbicide spraying both before the unit arrived and during their time there. We selected fifteen men from 2/8. All had at least six months service with the unit between October 1, 1967, and December 31, 1968. Each was matched against a control veteran who also served in Viet Nam but whose exposure was determined to be minimal. Selection and matching are described below.

Marines: The Ninth Marine Regiment, Third Marine Division, served in Military Region I (I Corps) just south of the demilitarized zone. Like the 2/8, the Ninth Marines' area of operations Had had a history of spraying before the unit arrived in 1967 and while it was there. We chose twenty men who served between January 1, 1967, and December 31, 1969, inclusive.

Navy: From 1966 on the U. S. Navy operated small patrol boats (the "Brown Water Navy") on the rivers and canals of South Viet Nam. Missions often went far upriver and lasted for weeks at a time. The river and canal banks were heavily sprayed by herbicides, and the waterways, of course, collected material which washed from land beyond the banks. In addition, the crews often ate "off the land" - fish, local livestock, fruit, vegetables, etc. Twenty men from a variety of Brown Water Navy units were chosen.

Selection and Matching of Controls

Selection of exposed subjects and the matching of the Army veterans were as described by Kahn, et al. (1988). All research subjects were volunteers. For reasons of time and expense we did not attempt to obtain individually matched controls for the marines and navy men, nor did we use controls who did not serve in Southeast Asia. In previous work it was shown that unexposed Viet Nam veterans and veterans who did not serve in Southeast Asia had similar dioxin and dibenzofuran congener levels, and neither group differed from the general population (Kahn, et al. 1988; CDC,

1988). Therefore, the fifteen controls in the present study and the seventeen in our earlier work would allow comparison of group means, although pairwise comparisons for the Marine and Navy groups would, of course, not be possible.

Sampling, Sample Handling, Sample Coding, and Analysis

Research subjects came to the New Brunswick, NJ, area for several days of testing. In previous work it was shown that there is a good correlation between blood and adipose tissue content of 2,3,7,8-TCDD (Patterson, et al., 1988; Kahn, et al., 1988), so only blood was taken in this study. In addition, it has also been shown that the twenty-four hour fast before drawing blood used in our earlier work is not necessary (Hansson, et al., 1989), so the subjects were fed a low fat dinner and fasted overnight before blood drawing, following which they ate breakfast. The fasting period was approximately fourteen hours.

Blood drawing and sample handling were as described (Kahn, et al. 1988) with extreme care taken to ensure freedom from chemical contamination which might introduce artifacts into the dioxin analyses. Once frozen, all samples, which were in identical bottles, were relabeled by a team of neutral referees not connected with the research groups in New Jersey or Umeå. The researchers were therefore blind as to the exposure statuses of the subjects until the conclusion of the study.

Analysis for all dioxin and dibenzofuran congeners having four or more atoms of chlorine was according to Nygren, et al. (1988), who have also described the QA/QC protocol.

Results

Sample analysis is in progress as of this writing (May, 1990). Because the samples are coded, and the code will not be broken until all analyses are completed sometime during the next few months, the results are not yet known and will be presented and discussed at Dioxin '90.

References

CDC (Centers for Disease Control), (1988). J. Amer. Med. Assn. **260:** 1249-1254

Fingerhut, M.A., Sweeney, M.H., Patterson, D.G., Jr., Piacitelli, A., Morris, J.A., Marlow, D.A., Hornung, R.W., Cameron, L.W., Connaly, L.B. Needham, L.L., and Halperin, W.E. (1989). Chemosphere **19:** 835-840

Gross, M.L., Lay, J.O., Lyon, P.A., Lippstreu, D., Kangas, N., Harless, R.L., Taylor, S.E., and Dupuy, A.E., Jr. (1984) Environ. Res. **33:** 261-268

Hansson, M., Rappe, C., Gochfeld, M., Velez, H., Wilson, W.P., Ghent-Guenther, T., and Kahn, P.C. (1989). Chemosphere **18:** 525-530

Kahn, P.C., Gochfeld, M., Nygren, M., Hansson, M., Rappe, C., Velez, H., Ghent-Guenther, T., and Wilson, W.P. (1988). J. Amer. Med. Assn. **259:** 1661-1667

Nygren, M., Rappe, C., Lindstrom, G, Hansson, M., Bergqvist, P-A., Marklund, S., Domellof, L., Hardell, L., and Olsson, M. (1986). In Rappe, C., Choudhary, G., and Keithm L.H., Chlorinated Dioxins and Dibenzofurans in Perspective, Lewis Publishers, Chelsea, Michigan, pp 17-34.

Nygren, M., Hansson, M., Sjostrom, M., Rappe, C., Kahn, P.C., Gochfeld, M., Velez, H., Ghent-Guenther, T., and Wilson, W.P. (1988). Chemosphere **17:** 1683-1692

Patterson, D.G., Hoffman, R.E., Needham, L.L., Roberts, D.W., Bagby, J.R., Pirkle, J.L., Falk, H., Sampson, E.J., and Houk, V.N. (1986) J. Amer. Med. Assn. **256:** 2683-2686

Patterson, D.G., Needham, L.L., Pirkle, J.L., Roberts, D.W., Bagby, J., Garrett, W.A., Andrews, J.S., Jr., Falk, H., Bernert, J.T., et al. (1988). Arch. Environ. Contam. Toxicol. **17:** 139-144

Pirkle, J.L., Wolfe, W.H., Patterson, D.G., Needham, L.L., Michalek, J.E., Miner, J.C., Peterson, M.R., and Phillips, D.L. (1989). J. Toxicol. Environ. Health **27:** 165-172

Poiger, H., and Schlatter, C. (1986). Chemosphere **15:** 1489-1494

Schecter, A., and Ryan, J.J. (1988). Chemosphere **17:** 915-920