Item ID Number 05325 ☐ **Not Scanned**

Author Roberts, Patricia A.

Corporate Author

Report/Article Title United States Environmental Protection Agency (EPA) Before the Administrator, In re: The Dow Chemical Company, et al. FIFRA Docket Nos. 415, et al., Direct Testimony of Dr. Ian C.T. Nisbet

Journal/Book Title

Year 1980

Month/Day May 15

Color ☐

Number of Images 68

Descripton Notes

Received
20 May 80

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

BEFORE THE ADMINISTRATOR

In re: The Dow Chemical Company, et al.	FIFRA Docket Nos. 415, et al.

DIRECT TESTIMONY OF DR. IAN C.T. NISBET*/

Dorothy E. Patton
Kevin M. Lee
Patricia A. Roberts
Richard P. Bozof
Timothy D. Backstrom
Andrew G. Gordon
Karl O. Bayer

Counsel for Respondent

U.S. Environmental Protection Agency
401 M Street, S.W.
Washington, D.C. 20460

*/ EPA Exhibit No. 494

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

BEFORE THE ADMINISTRATOR

In re: The Dow Chemical Company, et al.	FIFRA Docket No. 415, et al.

DIRECT TESTIMONY OF DR. IAN C.T. NISBET */

My name is Ian Christopher Thomas Nisbet. I am Principal Science Advisor to Clement Associates, Inc., Scientific Regulatory Consultants, Washington, D.C. I hold a Ph.D. degree in Physics and Statistical Mechanics from the University of Cambridge and I am a professional environmental scientist.

For the past ten years I have specialized in the study of the fate and transport of toxic chemicals in the environment, and the assessment of their effects on ecosystems and human health. Within this broad area of environmental science, I have specialized particularly in the assessment of human exposure to toxic chemicals. As explained more fully in this statement, I have taken a leading part in the development of this inter-disciplinary branch of environmental science. I have served on numerous panels and committees of the National Academy of Sciences/National Research Council and other national and international scientific bodies. Between 1975 and 1978, I was chairman of a committee of the National Research Council on Scientific and Technical Assessments of Environmental Pollutants. I am now a member of the National Research Council's Board on Toxicology and Environ-

*/ EPA Exhibit No. 494

mental Health Hazards. A curriculum vitae is attached to this statement.

Clement Associates is a consulting firm which specializes in the provision of expert scientific assessments of toxic chemicals in the environment. As Principal Science Advisor to the company since it was founded in 1976, I have had overall responsibility for the development of its scientific programs and for the scientific quality of its reports. In this position I have overseen work in the areas of environmental chemistry, environmental transport, exposure assessment, metabolism, pharmacokinetics, toxicology, ecotoxicology, epidemiology, statistics, risk assessment, and regulatory analysis. My primary role is to integrate the work of experts in these scientific disciplines and to ensure that multi-disciplinary work is coherent, consistent, and of high scientific quality. My own specific expertise is in the areas of exposure assessment and risk assessment, and I am directly responsible for the work of a small team within the company which develops these assessments.

Since 1976, I have been involved in a number of detailed exposure assessments as part of my work for Clement Associates and for committees of the National Research Council. This work is listed and summarized in my curriculum vitae. The assessments most relevant to this proceeding are those for heptachlor and chlordane, chloroform and carbon tetrachloride, PCBs, mirex and kepone, diflubenzuron, and hexachlorobenzene. In this and other work I have been directly involved in the interpretation and

synthesis of data from monitoring programs, field trials, metabolism studies, bioaccumulation studies, and ecosystem modeling.

In January 1979, under contract to the U.S. Environmental Protection Agency (EPA), Clement Associates undertook a full-scale critical review of data submitted to the Agency in response to its notice of Rebuttable Presumption Against Registration (RPAR) of 2,4,5-T. I was the director of this project, which involved a team of experts within Clement's staff, together with a number of outside consultants. Clement's report to EPA was completed in May 1979, and one part of it has been admitted as an exhibit in these proceedings (See, Exhibit 142). I was the primary author of the exposure sections in Chapters I and II, and of the risk assessments in Chapter VIII. I also took part, to a lesser or greater extent, in the synthesis and editing of material for the other chapters and I wrote the Executive Summary.

Although I have thus been involved with assessment of many aspects of 2,4,5-T and TCDD, my testimony in these proceedings is limited to the assessment of human exposure to these two chemicals and to silvex. The first part of this statement is an introduction to the science of exposure assessment and its relationship to risk assessment; the second part is an assessment of likely human exposure to the three chemicals. The second part parallels the exposure analysis in Chapters I and II of the Clement report of May 1979, but updates and extends this analysis to incorporate new data which have become available in the interim.

INDEX

Page

I. The Science of Exposure Assessment..... 5

II. Exposure Assessment for 2,4,5-T, Silvex, and TCDD..... 15

A. General Population Exposure to 2,4,5-T..... 16

1. Air..... 16

2. Water..... 18

3. Food..... 20

4. Wild Fish and Game..... 24

5. Target Monitoring..... 24

6. Summary..... 25

B. General Population Exposure to Silvex..... 26

C. General Population Exposure to TCDD..... 28

1. Beef and Milk..... 29

2. Rice and Fish..... 34

3. Wild Game..... 36

4. Air and Water..... 37

5. Total Exposure via All Routes..... 39

6. Human Tissue Monitoring..... 40

7. Concluding Comments..... 43

D. Occupational Exposure to 2,4,5-T..... 44

E. Occupational Exposure to Silvex..... 46

F. Occupational Exposure to TCDD..... 47

G. Accidental Exposure to Sprays..... 47

III. Summary and Conclusions..... 48

I. THE SCIENCE OF EXPOSURE ASSESSMENT

Humans may be exposed to toxic chemicals, including pesticides, by a variety of different routes. The routes usually considered to be most important are ingestion of food and drinking water, and inhalation of ambient air. Concentrations of toxic chemicals in these media usually vary greatly both in space and in time. For example, concentrations of toxic chemicals in air are usually much higher in the workplace or close to points of release than in the ambient air. Concentrations of toxic chemicals in food vary greatly, depending on the specific food commodity being considered and the history of its growth and processing. For pesticides, absorption through the skin is often an important route of exposure, resulting either from direct contact with the chemicals during manufacture, processing, or use, or from contact with contaminated foliage, soil, dust, or surfaces after use. For specific chemicals, other routes of exposure may sometimes be important, for example, dermal absorption from soaps or cosmetics, inhalation of cigarette smoke, or ingestion of mothers' milk. The primary task of an exposure assessment is to estimate the likely magnitude of human exposure to a chemical via all of these routes, and to derive some measure of the variability of these exposures in time, in space, and within the human population.

For regulatory purposes, it is often desirable to determine what fraction of human exposure is attributable to each major use of a chemical, or to each source of release into the environment. For this purpose it is necessary to trace the pathways of movement

of chemicals from the points at which they are released into the environment to the points at which they come into contact with humans. To do so usually requires knowledge of the quantities of the chemical that are released; the places and times at which it is released; the environmental conditions at the points of release; the behavior of the chemical in air, water, soil, and other media; its persistence in each of these media; the mechanisms and products of its degradation in the environment; rates and routes of transport within and between media; factors controlling uptake by plants and animals; and rate of intake of various items by humans (including dietary patterns, breathing rates, consumption of water, etc.).

Although exposure assessments may be conducted independently of risk assessments, the usual purpose of assessing exposures is to determine whether or not they are of sufficient magnitude to pose significant risks, either to the population as a whole or to more highly exposed individuals or sub-groups. For this reason it is usually valuable to conduct exposure assessments and risk assessments in parallel, if not together. In the first place, unless it is known what doses may be considered toxicologically insignificant, it is not possible to neglect any route of exposure, however minor it may be. Secondly, to compare an estimate of human exposure with a level of dosage that is known to pose risk requires that each be expressed in commensurable units. "Exposure" is a complex concept which is not synonymous with that of "dose": the former includes not only the quantity

of a chemical which reaches an individual, but also the routes of exposure, the fraction absorbed into the body after exposure by each route, the schedule of exposures (including fluctuations in time), and the distribution of exposure among the population. For use in risk assessment, it is usually desirable to express estimates of exposure in the same units as those of dose. Thus, one useful measure of exposure is the average quantity absorbed per unit time by the average individual. However, it is always desirable to include some measure of variability in exposure. For some types of effect (such as acute toxicity or teratogenicity), it may be more important to estimate peak exposure to individuals ("worst case" exposures) than long-term averages.

In some cases the most useful measure of hazard to individuals may be the concentrations of the chemical in body tissues. In such cases it is necessary to relate measures of tissue concentrations to measures of dose or exposure: this requires some knowledge of the pharmacokinetics of the chemical in humans, in experimental animals, or in both. In many cases available information on residues of human tissues refer to metabolites rather than to the parent chemical: in these cases some knowledge of the pathways of metabolism is necessary for risk assessment.

Exposure assessments may be based upon four types of data:

(i) Ambient monitoring. These data include measurements of the concentration of the chemical and/or its metabolites in samples of air, water, soil, sediment, plants, animals, food, or other substrates. The measurements may be systematic (based on

samples designed to the representative of the general environment), targeted (based on samples designed to investigate specific types of exposure), or haphazard. Such data may be converted into estimates of exposure if the rates of intake of the materials, e.g., breathing rates or dietary intakes, are known; however, where humans are exposed to a chemcial via several routes, it is difficult to convert the data on exposure to measures of total dose unless the efficiency of absorption via each route is known or can be assumed. In addition, the representativeness of the samples, the reliability of identification of the chemical residues, and the accuracy of the measurements of concentrations always need to be considered.

(ii) Exposure models. Where direct measurements of ambient concentrations are not available, estimates may be derived from various types of models. These range from simple models of dispersion of chemicals in air or water, through more complex models of the uptake of chemicals in air or water, through more complex models of the uptake of chemicals by plants and animals, to very complex models of the movement of chemicals among various media in the natural environment. In some cases, laboratory "model ecosystems" have been developed to investigate the behavior of chemicals in multicomponent systems designed to simulate the natural environment. In the case of pesticides, it is customary to conduct small-scale field trials which are designed to

investigate the behavior of pesticides and their metabolites under "real world" conditions, and to measure the extent of their uptake by plants and animals. Such trials customarily involve the use of the pesticide at rates of application higher than those expected to be used on a routine basis, in order to facilitate the detection and measurement of low level residues. These field trials ordinarily simplify the task of estimating exposure to pesticides via residues in food, but it is usually still difficult to estimate exposure via air, water, and other routes.

(iii) Target monitoring. In some cases measurements are available on residue concentrations of the chemical in the tissues of humans or other target organisms. Where available, such data are extremely valuable for two reasons. First, they reflect total exposure via all routes, eliminating uncertainties involved in predicting ambient concentrations or uptake efficiencies. Second, they often provide exact measures of exposures to specific organs, eliminating uncertainties involved in translating intake rates into organ doses. However, they have two offsetting disadvantages. First, it is usually impossible to determine what fraction of the tissue residues is attributable to each of the uses under consideration. Second, it is difficult to relate tissue concentration to dose unless some information is available (or can be assumed) about metabolism and pharmacokinetics of the chemical in humans.

(iv) Analogies and surrogates. In cases where information about exposure to a chemical is incomplete, it is often possible

to fill gaps by utilizing information on similar chemicals which have been studied more fully. To quote an example directly applicable to this proceeding, information on the environmental behavior of 2,4,5-T can be used to fill gaps in the information about the environmental behavior of silvex, by making the assumption that their chemical similarity will lead to similarity in environmental behavior. The validity of this assumption needs to be considered critically whenever analogies of this kind are used, and the resulting estimates of exposure should reflect any uncertainties introduced by the procedure.

The best exposure assessments are those that utilize data of all the four types mentioned above. For each chemical that has to be assessed, the quantity and quality of data of each type will be different. The task of the scientists making the exposure assessment is to make the best possible use of all the types of data, placing appropriate weight upon each and using data of each type to check inferences drawn from the others.

Ideally, an exposure assessment should provide enough information to draw inferences about the nature, magnitude, and distribution of risks to all exposed populations, and about the relationship of these risks to each of the uses of the chemical that is under review. For this ideal purpose to be achieved, the output of the exposure assessment should include numerical data on the nature, magnitude, and statistical distribution of exposures resulting from each use, including temporal fluctuations in exposure, and should identify the most heavily exposed sub-populations and the magnitude of exposure to especially sensitive

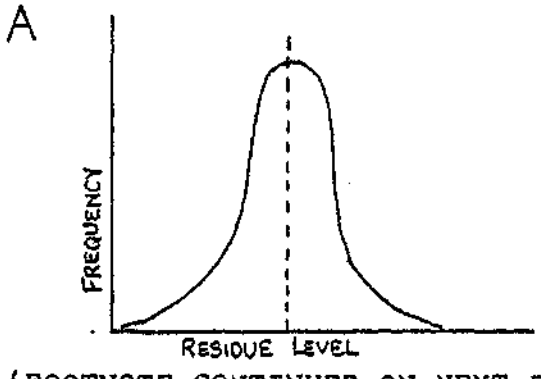
groups. To achieve such an ideal assessment requires a prohibitively large amount of data, and the risk assessor always has to settle for something less than the ideal. It is important, therefore, to take full account of the limitations imposed by incomplete, inadequate, or missing data, and to avoid presenting conclusions in greater detail than are warranted by the quantity and quality of data available. In many cases it is possible to give little more than an estimate of an "average" or "typical" exposure, together with a rough estimate of the range of variability and of the number of people likely to be exposed. Where very few data are available, it may be possible to do no more than estimate the order of magnitude of exposure. In extreme cases it may be possible to estimate only an upper limit on the plausible magnitude of exposures: this upper limit may be based on the limits of analytical detectability of the chemical in media likely to be contaminated, or upon model calculations of the dispersion and fate of the chemical in the environment. Such estimates of maximum exposures should be distinguished clearly from estimates of the <u>likely</u> magnitude of exposure, but they may be valuable in certain regulatory contexts because they represent a level below which it cannot be proved that exposures actually lie. However complete the data may be, it is always desirable to give estimates of the highest exposures likely to occur, because these are the exposures most likely to lead to adverse effects. Where the statistical distribution of exposures is known or can

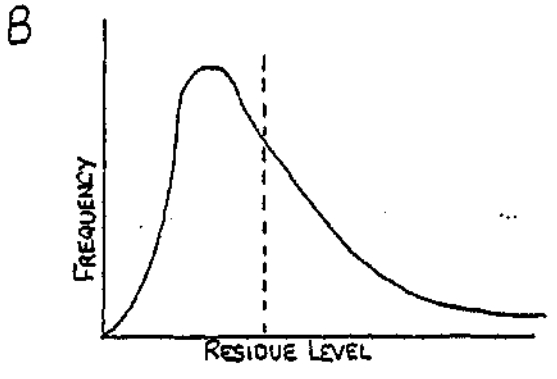
be estimated, it is possible to calculate the 90th, 95th, or 99th percentile*/ exposure and to present these as measures of "high exposures." Where the statistical distribution is not known, the concept of "highest plausible exposure" is useful, although the specification of what exposures are implausible is usually difficult to make precise.

The estimation of typical exposures and upper limits in the absence of extensive data is facilitated by certain empirical rules. Exposures to toxic chemicals are always very variable in both space and time. Whenever good statistical data are available, it has been found that the statistical distribution of residues of toxic chemicals in environmental media is strongly skewed.**/ In several cases these statistical distributions are known to

*/ The 90th percentile is the level of exposure above which only 10 percent of the population are exposed.

**/ When a numbr of samples of an enviornmental medium are analyzed, the residue levels of a chemical that are found usually vary widely. The statistical distribution of residues is a mathematical description of this variability. It describes the frequency of occurrence of various residue levels in the population that is sampled. When plotted on a graph, the distribution may be either symmetrical (A) and skewed (8).





(FOOTNOTE CONTINUED ON NEXT PAGE)

fit very closely to the mathematical form known as a lognormal distribution. In the absence of extensive data, it is a good assumption that the distribution of exposures will be skewed, and a plausible assumption that it will be close to lognormal. If 10-100 samples of an environmental medium are taken, it is then reasonable to assume that the highest residue level found is likely to be between three and ten times the mean. Although this "rule of thumb" is obviously not a precise quantitative tool, it is often useful in deriving mean exposure levels from maximum levels, or *vice versa*.

The foregoing introduction will make it clear that exposure assessment is a complex multi-disciplinary task. It requires the critical review and assessment of data from many scientific disciplines: environmental monitoring, analytical chemistry, environmental chemistry, pesticide residue kinetics, metabolism, pharmacokinetics, bioaccumulation, and statistics. It requires some background knowledge of relevant aspects of process technology, effluent controls, industral hygiene, waste disposal, agricultural practices, meteorology, hydrology, soil science, ecology, food technology, human physiology, and dietary patterns,

---

(FOOTNOTE CONTINUED FROM PREVIOUS PAGE)
In a symmetrical distribution (A), there is an equal probability that a measurement will fall above or below the mean (indicated by a dotted line). In a skewed distribution (B), most measurements fall below the mean, but a few fall well above it. Most residues of chemicals in the environment follow skewed distributions. It is an empirical observation that they often agree closely with the mathematical form known as lognormal (See Ref. 1).

including the consumption of specialist commodities such as fish and wild game. It also requires broad general knowledge of natural and social factors that affect exposure, and good scientific judgment, especially in the drawing of conclusions from incomplete information without either underestimating or overestimating the precision of results. For these reasons, the quality of exposure assessment depends greatly on experience and upon broad knowledge of the relevant fields. In conducting exposure assessments I rely upon an informal group with expertise in several of the relevant fields, consulting specialists whenever special problems arise. Apart from my group at Clement Associates, I know of only two other exposure assessment teams with comparable experience. The distinctive feature of my approach, which I regard as a strong advantage, is that I place substantial weight on data from target monitoring and on analogies, rather than relying exclusively on data from ambient monitoring and models (as most other exposure assessors do).

To illustrate the points made in this section, I have selected four examples of exposure assessments for persistent chlorinated chemicals, each of which has some points of similarity to TCDD. References 55 and 56 are assessments carried out by myself on chlordane, heptachlor, and their metabolites, and on kepone and mirex, respectively. References 1 and 2 are assessments carried out by a group at Stanford Research Institute, on toxaphene and on mirex and kepone respectively. Reference 56 was a brief assessment which was based on the more detailed review of data in

Reference 2; comparison of the two assessments shows how the results of monitoring can be extended by means of analogies to provide reasonable scientific predictions of significant exposures that have not yet been measured. All four reports illustrate and document certain procedures and assumptions used in the next section, and provide analogies used therein.

II. EXPOSURE ASSESSMENT FOR 2,4,5-T, SILVEX, AND TCDD

In this section I present assessments of human exposure to 2,4,5-T, Silvex, and TCDD likely to result from the principal uses. With the exception of some aspects of the environmental behavior of 2,4,5-T, information relevant to assessing human exposure to these three chemicals is generally scanty. The conclusions of this assessment are limited accordingly, and are appropriately qualified.

These assessments are based upon all relevant data available to me through May 3, 1980.*/ I have reviewed data included or referenced in EPA's "Position Document I", in the assessment by the joint USDA/States/EPA 2,4,5-T Assessment Team, and in the principal rebuttal comments, including those of Dow Chemical Company. I have also reviewed published data located by means of computerized literature searches through February 1980, and I have read the testimonies of several witnesses in this proceeding, including Drs. Beroza, Tiernan, and Gross and Messrs. Harless and Dixon. For evaluation of the reliability of data on environ-

*/ These assessments supersede those in the Clement Report of May, 1979, which were based on a smaller amount of information.

mental chemistry and residue identification, I have generally relied upon the assessment in Chapter I of the Clement report, which was prepared by Dr. Beroza and has been presented with his testimony (See, Exhibits 141,142). For evaluation of the reliability of data on occupational exposure, I have generally relied upon the assessment in Chapter II of the Clement report, which was prepared by Dr. Beroza and myself. The use of these data in assessing exposure is my exclusive responsibility.

A. General Population Exposure to 2,4,5-T

For the reasons stated in Exhibit 142, I accept the evidence that residues of 2,4,5-T and its metabolites are transitory in the environment and that they do not build up or accumulate in soil, vegetation, or animals. However, it is difficult to estimate general population exposure because there are few systematic surveys. In particular, there are extremely few residue studies or field trials which directly relate residues in human food to current uses of 2,4,5-T. Accordingly, estimation of average levels of exposure is extremely difficult. The following assessment is, therefore, concerned primarily with estimation of exposures to highly exposed subgroups and of maximum plausible exposures. It is based primarily on ambient monitoring studies, using a target monitoring study as an independent check.

1. Air

Results of the National Air Monitoring Program, conducted in 1970-72, were summarized in Reference 6. Esters of 2,4,5-T were detected in ambient air in 8 of 28 states where samples

were taken. At four urban sites, reported concentrations ranged from 4.2 to 43 ng/m^3, with a mean of about 22 ng/m^3; at a rural site in Oklahoma, reported concentrations ranged from 42 to 161 ng/m^3, with a mean of about 100 ng/m^3 (Ref. 6, Table 2). Average levels in various states ranged from 0.5 to 14.6 ng/m^3 (Ref. 3, Table 1). As in other cases considered later, the wide variability in concentrations of 2,4,5-T, both in space and in time, makes it difficult to estimate an average or typical concentration. Taking 2 ng/m^3 as a representative mean level (exceeded in only 3 states), and assuming that a person breathes 25 m^3 of air in a day, the typical daily intake by inhalation would be 0.05/ug. However, at the urban sites the typical daily intake would have been of the order of 0.5 ug, and at the rural site it would have been of the order of 2 ug.

In areas where 2,4,5-T is actually used, much higher concentrations have been reported in air. In the Blodgett Forest Study (Ref. 51), 2,4,5-T was applied to a small test plot at a rate of 3 lbs/acre. Concentrations of 2,4,5-T (measured as the propyleneglycol butyl ether esters) in air peaked on the day after application (28 ug/m^3 in the morning, 161 ug/m^3 in the afternoon), and were still high on day 21 (0 ug/m^3 in the morning, 13 ug/m^3 in the afternoon). In another study, concentrations of 2,4,5-T in air near treated wheat fields ranged up to 3.4 ug/m^3, with mean levels of 13 ng/m^3 in the vapor phase and 36 ng/m^3 in aerosol form (Ref. 7). On the basis of these data, Johnson (Ref. 8) estimated that daily intakes by persons inhaling

the air would range up to 1.8 ug. The USDA/ States/EPA Assessment Team (Ref. 4), basing their conclusions on more extensive data for 2,4-D, estimated that average intakes in high use areas would be about 3 ug per person per day. (These figures are based on the assumption of a breathing rate of 30 m^3 per day, and would therefore be appropriate for physically active persons). Both of these estimates appear reasonable for average intakes of persons living in or near treated agricultural areas. However, the study in Reference 7 indicates that transitory intakes could range up to 100 ug/ person/day. In forested areas, the Blodgett Forest Study indicates that intakes could range up to 3,000 ug/person/day*/ following application of 2,4,5-T, and could continue at a high level for several weeks.

All these figures are estimates of the quantity of 2,4,5-T likely to be taken into the lungs by inhalation in the course of a day. The amount absorbed into the body is likely to be somewhat less, but I have not seen data on the fraction that is absorbed.

2. Water

2,4,5-T has been reported in surface waters in a number of studies. In two surveys conducted by the U.S. Geological Survey, 2,4,5-T was detected in 10-20 percent of ambient water

*/ This figure is obtained by multiplying the average air concentration of about 100 ug/m^3 observed on day 1 in the Blodgett Study by 30 m^3/day for the breathing rate of a physically active person.

samples from western states, usually at concentrations of 0.01-0.02 ppb, but ranging up to 0.40 ppb (Ref. 9,10). Other studies have been summarized in the testimony of Mr. Thomas E. Dixon (Ref. 11). In the National Surface Water Monitoring Program, 2,4,5-T was detected at levels between 0.03 ppb and 12.9 ppb in 10 of 2,000 samples collected since 1976. The positive findings were in rivers in central and southern states. In the "Modified Cotton Survey." 2,4,5-T was detected at levels betwen 0.03 and 13.75 ppb in 7 of 69 samples collected from rivers in southern states. Six of the positive findings were from the Lubbock area of Texas. Dixon (Ref. 11, pp. 10-17) also summarized data from the STORET data system. 2,4,5-T was reported at least once at 18 percent of the 636 stations which had analyzed for it on five or more occasions, usually at levels less than 0.1 ppb.

Dixon (Ref. 11, pp. 17-30) also summarized a number of studies in which 2,4,5-T levels were measured in streams during and after spray applications to forested areas. 2,4,5-T was found in stream water in all of these studies at concentrations ranging up to 5 ppb but in one case reaching 50 ppb and in another case 550 ppb (Ref. 11, Tables 5 and 6). In the Blodgett Forest Study (Ref. 51), 2,4,5-T was found in a watering trough in the sprayed area at a concentration of 364 ppb (measured as the propyleneglycol butyl ether ester) on the day after spraying; concentrations of the acid in the water declined very slowly (to 132 ppb after 30 days and to 2.2 ppb after 7.5 months).

2,4,5-T was also detected in water from a spring outside the sprayed area, at concentration of 1 ppb and 0.3 ppb on days 0 and 1, respectively.

These data indicate that 2,4,5-T occurs fairly widely in surface waters in the U.S., usually at levels below 0.1 ppb. Although I am not aware of any reports of 2,4,5-T in public drinking water supplies, there is obviously a potential for its occurrence at low levels in supplies drawn from rivers. However, exposures via this route would generally be very small. Taking 0.03 ppb as a representative level in waters where 2,4,5-T occurs, and assuming an average intake of 2 liters of water per person, the daily intake by the typical consumer would be the order of 0.06 ug.

In streams subject to direct spraying or run-off from treated areas, 2,4,5-T occurs sporadically at much higher levels, ranging up to 5 ppb and even to 500 ppb in certain circumstances. This indicates a potential for large intakes (10-1,000 ug/person/day) by persons who draw their water directly from such sources. It seem likely that the number of persons who do so is small, but they might include campers and persons who collect surface water for drinking.

3. Food

Calculations based upon model studies have been used to argue that residues of 2,4,5-T in meat and milk resulting from use of 2,4,5 T on pasture and rangeland would be very low (Ref.

5, pp. 68-72). Although these arguments are plausible, their validity depends on the assumption that label restrictions on the placement and removal of animals on treated areas are observed consistently.*/ I have seen no evidence on this point.

It is unfortunate, also, that there appear to be no direct measurements of 2,4,5-T residues in meat or milk after use under current practices. The lack of adequate field residue studies under actual or simulated field use conditions is a serious deficiency in the data on 2,4,5-T. Such studies are needed before firm estimates can be made of the likely exposure of highly-exposed groups, for example, those deriving their meat and milk exclusively from regions where 2,4,5-T is used.

For the general population, two indirect sources of information are useful. In the FDA Total Diet Survey ("Market Basket" Survey), up to 30 total diet samples have been analyzed each year since 1964 (Ref. 12-22.). To date there have been only three positive findings for 2,4,5-T and two for silvex, as follows:

---

*/ Generally, a one week interval after application is required before dairy cows can be allowed to graze on treated land; beef cattle must be removed from treated land at least two weeks before slaughter.

Chemical	Fiscal Year	Food Composite	Residue Level (ppm)
Silvex	1966	Dairy Products	0.018 (in fat)
Silvex	1966	Dairy Products	0.029 (in fat)
2,4,5-T	1967	Dairy Products	0.190 (in fat)
2,4,5-T	1967	Meat, fish and poultry	0.003 (in fat)
2,4,5-T	1968	Dairy Products	0.008 (in fat)

All these findings were in food composites purchased in the Boston area. There have been no positive findings since 1968.

Unfortunately, this program has a number of limitations, which are discussed more fully in Reference 55:

(1) Only 30 Total Diet samples are collected each year, and only one of the cities where samples are collected is adjacent to rangeland.

(2) Samples of dairy products and of meat and fish are composited, so that residues in individual samples are diluted prior to analysis.

(3) In 1971 the analytical procedure for these composites was changed so that they are now analyzed on a whole-product basis instead of on a fat basis as before; this further reduces the sensitivity.

These factors make the sporadic positive findings in the above table very difficult to interpret. We cannot tell whether the lack of residue findings since 1968 is due to an improvement in agricultural practices, or to the change in analytical procedures in 1971. Assuming the latter, these data would indicate that 2,4,5-T may occur sporadically in milk fat at levels up to 0.19 ppm and in other animal fats at levels up to 0.003 ppm.

These would correspond to daily intakes up to 11 ug/person and 0.1 ug/person respectively for a typical consumer (See Ref. 23, 24 for dietary intakes). These would represent peak intakes which might occur sporadically.

To estimate an upper limit on average intakes, I note that the sensitivity level for quantitation of 2,4,5-T in the Total Diet Survey is stated to be 0.02 ppm (Ref. 17, p. 313; Ref. 18, p. 111). However, FDA often estimates residues at levels below this nominal sensitivity. In the case of 2,4,5-T, one such estimated residue level was as low as 0.003 ppm (Ref. 14, p. 19). Considering the characteristically skewed distribution of residue levels in this program, it is reasonable to assume that residues would have been detected at least occasionally since 1971 if the average level of 2,4,5-T exceeds 0.002 ppm (whole product basis) in either the dairy products composite or the meat, fish, and poultry composite. The average daily intake of these food items by the U.S. population is about 895 g (Ref. 23,24). An upper limit in the average daily intake of 2,4,5-T by the general population is then about 1.8 ug or about 0.03 ug/kg/day.*/

It is unfortunate that 2,4,5-T is not normally sampled in surveillance programs for pesticide residues in raw agricultural commodities conducted by FDA and USDA. These programs provide

*/ To convert intakes from ug/person to ug/kg bodyweight I have generally used 69 kg as the weight of a typical person, although in some cases I round up the resulting estimates of intake in ug/kg to the next higher digit: this is equivalent to considering persons of weight between 60 to 70 kg.

useful evidence on the occurrence of other pesticides in food (Ref. 55).

4. Wild Fish and Game

Another possible source of exposure of the general population to 2,4,5-T is via wild fish and game. The most likely source of significant exposure is via fish caught downstream from treated areas. Dixon (Ref. 11) failed to identify 2,4,5-T (at a detection limit of 5-10 ppb) in catfish and crayfish in crayfish rearing ponds in Louisiana. However, this was only a preliminary survey. In view of the fairly widespread occurrence of 2,4,5-T in water, it would be expected to occur in fish, if only at low parts per billion levels. More data would be needed to establish whether significant exposures occur via this route.

5. Target Monitoring

Another source of information on potential exposure to 2,4,5-T is the National Human Monitoring Program HANES II project, a multi-year study whose primary purpose is to measure the prevalence of certain health and nutritional indicators in the general population. As part of this program, urine samples from 7,500 persons throughout the country are to be analyzed. Of the 4,580 samples analyzed to date, 2,4,5-T has not been clearly identified in these samples; trace concentrations (<10 ppb) were found in a few samples, but the identity of these trace quantities could not be confirmed (Ref. 6). Although these results do not help to establish average exposure levels in the general populaton,

they can be used to estimate an upper limit. The failure to find 2,4,5-T at a detection limit of 10 ppb indicates that it does not often occur in the urine of the general population at this level (except perhaps sporadically or in limited groups with above-average exposure). Assuming that the average person excretes 1.5 liters of urine per day, a concentration of 10 ppb in the urine would correspond to excretion of 15 ug per day. This would not necessarily equal the amount taken into the body, beause exposures are expected to be sporadic. The data of Ramsey et al. (Ref. 25) suggest that 2,4,5-T is excreted over several days following a single exposure and that typically only one-half to one-quarter of the total quantity absorbed is excreted on any one day. Hence, the data from the HANES II survey suggest that daily intakes by the general population are unlikely to exceed 60 ug/day, even on an occasional basis, and are unlikely to exceed 15 ug/day on the average. These conclusions are limited by the incompleteness of the survey, but are based on an unusually large sample.

6. Summary

In the absence of specific studies that could provide better direct estimates, I have used the data cited above to estimate the daily intake of 2,4,5-T by the average consumer of meat and milk as not more than 0.03 ug/kg/day, and the intake of highly-exposed persons by all routes as not more than 0.2 ug/kg/day. The data from the Total Diet Survey suggest that individuals in the general population were formerly --and may still be --exposed sporadically to quantities as high as 0.25 ug/kg/day in the diet.

If intakes larger than this occur frequently in the general population, they would probably have been detected either through the Total Diet Program or the HANES II Survey. However, it is unlikely that either of these two surveys would have adequatley sampled two classes of persons with potentially high exposures: those who consume milk from cows grazing on treated pastures, and those who consume fish from waters downstream from treated areas.

My estimates of exposure are summarized in Table 1. Although some of these estimates are upper limits (based on the sensitivity of analytical methods used in surveys), more systematic surveys would be necessary to establish that actual exposure is less than these estimates.

B. General Population Exposure to Silvex

I have found few specific data that are useful in assessing general population exposure to silvex. Silvex has been found occasionally in water (Ref. 9,10,11) and in food (see Table on page 27 of this statement) at concentrations of the same order of magnitude as those of 2,4,5-T. In view of the similarity between the two chemicals, it seems reasonable to assume that human exposure to silvex would be of a similar magnitude to exposure to 2,4,5-T, at least when they are used in similar ways. Since silvex is used less than 2,4,5-T, it would be expected that fewer persons would be exposed to silvex, but the levels of exposure to these persons would probably be similar.

TABLE 1

ESTIMATES OF HUMAN EXPOSURE TO 2,4,5-T

Route of Exposure	Estimated intakes by more highly exposed persons (ug/kg/day)	Class of persons considered
Air	0.008	General population
Air	Up to 0.05	Residents of treated farmland
Air	Up to 50	Residents of treated forests
Water	0.001	General population
Water	Up to 15	Users of stream water in treated areas, sporadic exposures
Meat & milk	Not more than 0.03	General population, consuming food bought in stores
Meat & milk	Up to 0.25	General population, consuming food bought in stores
Meat & milk	No data	Consumers of milk from cows on treated pastures
Wild fish and game	No data	Fishermen, hunters, and their families
All routes	Not more than 0.2	General population, average
All routes	Not more than 1	General population, transitory exposures

silvex, but the levels of exposure to these persons would probably be similar.

Residues of silvex were also detected on apples in a controlled field trial, at levels in the range 0.01-0.1 ppm (Ref. 26). Silvex is registered for use on apples as a growth regulator. However, I do not know the extent of this use and I have not attempted to estimate resulting exposures.

C. General Population Exposure to TCDD

The following assessment of the likely magnitude of human exposure to TCDD is based upon reports of identification of TCDD in environmental samples, in human food, and in human tissues. These reports have been presented in this proceeding in the testimony of Drs. Baughman, Tiernan, Gross and Beroza, and Mr. Harless. The reliability of the identification of TCDD in these samples has been the subject of much debate, primarily because many of the reported concentrations of TCDD have been close to the limits of detectability. For this reason, confirmatory analytical techniques (which often have a lower sensitivity than the primary techniques) have sometimes given ambiguous results. On technical matters in analytical chemistry, I rely generally upon the judgment of Dr. Beroza, as represented in the Clement Report (See, Exhibit 142) and his testimony (See, Exhibit 141). However, in this assessment I also place considerable weight on other factors, including the scientific plausibility of the reported findings, their consistency with each other and with other sets of data, and analogies with other chemicals. My

judgments as to the probability that the identifications of TCDD and the estimates of residue levels are correct are based on all these considerations.

1. Pasture and Rangeland: Exposure via Beef and Milk

Three sets of data indicate that TCDD is likely to occur in meat and milk from cattle grazing on pasture and rangeland that have been treated with 2,4,5-T. Two of these sets are from monitoring studies and one is from a field trial.

The first monitoring study is from the Dioxin Implementation Plan (Beef Study Phase I). Sixty-seven samples of beef fat from cattle grazed on range treated with 2,4,5-T were analyzed at several laboratories. Interpretation of the data is complicated by the fact that the samples were distributed and analyzed in different ways, and that various criteria for consistency of results can be used. There were no consistent positive findings of TCDD in 18 cattle grazed on untreated range. Three samples from range treated with 2,4,5-T gave consistently positive results for TCDD (at 60, 20, and 20 ppt); five other samples may have had TCDD levels in the range 5-10 ppt (at or slightly below the limit of consistent detectability) and the remaining 59 gave non-positive results (i.e., less than 5 ppt) (See, Exhibit 141, p. 21). Depending on what values are assigned to the non-positive samples,

the mean residue level might be as low as 1.8 ppt or as high as 6.8 ppt.*/ I have not conducted a detailed review of data on the conditions of exposure of the cows, except to verify that the three cows with the highest residues were all raised on the same form.**/

The second monitoring study is also from the Dioxin Implementation Plan (Beef Study Phase II). Thirty-nine samples of fat from cattle grazed in Texas and Missouri have been analyzed at Wright State University (Ref. 28). Although independent results from other laboratories are not available for these samples, Wright State's duplicate analyses were extremely consistent. They obtained consistent positive results in four, of the 39 samples, at levels of 33, 14, 10 and 10 ppt. A fifth sample probably contained TCDD at 8 ppt. The remaining samples gave negative results at an average detection limit of 9 ppt.

---

*/ The higher figure is obtained by assigning the value 5 ppt to all the non-positive samples; the lower figure is obtained by assigning zero to these samples. In both cases the value 7.5 ppt was assigned to the probable findings in the range 5-10 ppt. Meselson et al. (Ref. 27) stated that only 25 samples were analyzed at a sensitivity of 10 ppt or better by more than one laboratory, and that these included 9 samples for which two or more laboratories reported positive findings (1 at ca. 65 ppt, 2 at ca .20 ppt, and 6 in the range 5-20 ppt). Using these data, the average level of TCDD would lie in the range 3.8 to 10.4 ppt.

**/ I understand that the hypothesis has been presented that these residues might have arisen from contaminated waste oil rather than 2,4,5-T. Although actual evidence would be needed to confirm or refute this hypothesis, it seems very unlikely that waste oil would have been used on a cattle pasture.

Although Dr. Tiernan (Ref. 29) did not know the origin of these samples, I have obtained data on the treatment of the range and the grazing schedule of the cows from EPA and have analyzed the results further. Of the 39 samples analyzed, residue data are missing for one (BA II 32), sampling data are missing for one (BA II 30), and 10 were controls. Treatment rates varied from 0.5 to 3 lbs active ingredient per acre, and on average less than half the total acreage of grazing land was treated. Silvex was used in at least three cases. The data sheets refer to "pasture", "range", and in some cases both, therefore, a more precise designation than "grazing land" cannot be assigned. Most or all of the animals were female; it was not stated whether any were milked. Data for the five animals with positive findings for TCDD are tabulated in Table 2. The most noteworthy results of this analysis are (i) that 3 of 5 calves placed on treated rangeland acquired detectable residues of TCDD, whereas only 2 of 22 adult cows did so; (ii) that residues were detected in 2 2 calves from range treated with only 0.5 lb/acre of 2,4,5-T. The average residue level in fat of the 27 animals from treated areas may have been as high as 10.1 ppt or as low as 2.8 ppt (see footnote, p. 29).

The field trial is that conducted by Kocher et al. (Ref. 30). In the last of several studies reported in this

paper */, an enclosed pasture was sprayed with 0.5 lb/acre commercial 2,4,5-T (containing an unknown level of TCDD); cattle were grazed on the pasture between days 10 and 40 after spraying, then held on an untreated area for 2 weeks. TCDD residues at a level of 3-4 ppt was detected in the fat of 3 of 7 animals analyzed. The data demonstrate that cattle can accumulate measurable residues of TCDD by grazing on treated pasture. As a quantitative measure of intake, however, the study has several limitations:

(1) The level of TCDD in the 2,4,5-T was not known.

(2) Thirty days' intake is almost certainly too short to reach a steady state level of TCDD in the fat.

(3) However, a longer grazing period might have led to a reduction in the level of TCDD residues on the pasture.

Each of these three studies suffered from some limitations, as pointed out above. However, the results of the three studies are consistent in showing that cattle grazing on pasture and/or rangeland treated with 2,4,5-T acquire measurable levels of TCDD in their fat within 1-5 months. The average levels of TCDD in the animals appear to have fallen in the range 1.8 - 10.1 ppt. I have used these data to adopt 4 ppt as the most likely average value for TCDD levels in beef fat from treated areas. **/ According

*/ In the other studies the pastures were only spot-sprayed or had been treated 7-24 months before the animals were killed: these studies are of little value for quantitative analysis.

**/ In none of the three studies on which this figure is based was the level of TCDD in the 2,4,5-T established. All three studies involved 2,4,5-T applications after 1973, at which period the TCDD level should have been below 0.1 ppm. Better designed studies would be necessary to show that a lower figure than 4 ppt would be appropriate for 2,4,5-T manufactured to present-day specifications.

TABLE 2. DATA ON FIVE COWS WITH POSITIVE FINDINGS FOR TCDD IN THE BEEF STUDY PHASE II

Sample No.	Treatment Rate (lb/ac)	Fraction of acreage treated	Date of application	Date of slaughter	Age of cow at application	Sex	Condition at slaughter	TCDD level (ppt)
BA II 5	3	300/593	6/6/75	11/20/75	4 years	F	Emaciated	8
BA II 9*	3	300/593	6/6/75	11/20/75	4 years	F	Lean	10
BA II 12	0.5	645/645	5/8/75	11/19/75	4 mo. **	?	Lean	14
BA II 20	2	60/80	6/15/75	11/21/75	6 mo.	F	Lean	33
BA II 27	0.5	143/143	5/19/75	11/19/75	8 mo.	?	Lean	10

* From same farm as BA II 5

** Not placed on range until 8/7/75, three months after application

to data obtained in feeding trials by Jensen et al. (Ref. 31,32), these levels in beef fat could correspond to levels of about 0.2 ppt in milk and 0.1 ppt in muscle. */ Table 3 shows the resulting estimates of the average intake of TCDD by persons consuming meat and milk from cattle grazed on treated range or pasture.

2. Rice: Exposure via Rice and Fish

Data relevant to estimating potential exposure to TCDD resulting from uses on rice are incomplete. The only direct studies are by Jensen et al. (Ref. 35) who analyzed rice from retail stores and from treated rice fields, and by Shadoff et al. (Ref. 36) who analyzed catfish and other samples from a pond in Arkansas which "was used as a reservoir for irrigating rice fields and which collected the drainage from rice fields previously treated with 2,4,5-T." Both studies were limited by the relatively high detection limits for TCDD and by the lack of precise information on the treatment of the areas involved with 2,4,5-T and on the levels of TCDD in the product.

Jensen et al. found no residues of TCDD in rice from retail stores at a detection limit of 10 ppt. They reported "apparent TCDD residues" at levels of 4-5 ppt in 5 of 6 samples of rice from treated fields. Shadoff et al. reported "apparent positives"

*/ Mahle et al. (Ref. 33) reported negative results in a search for TCDD in milk from cows grazing on treated pasture or range. Their limit of detection was 1 ppt. This level of sensitivity is insufficient to have detected TCDD at the level predicted in the text. Hence, the study by Mahle et al. does not provide substantial negative findings, although it may help to define an upper limit on potential residue concentrations in other tissues.

TABLE 3

ESTIMATED INTAKE OF TCDD FROM MEAT AND MILK OF CATTLE GRAZED ON TREATED RANGE OR PASTURE

	Fat	Muscle	Milk
Assumed concentration of TCDD (ppt)	4	0.1	0.2
Average consumer			
Daily intake of food (g)*	28 **	112	555
Compiled intake of TCDD (pg)	112	11	111
High consumer			
Daily intake of food (g)***	40	159	1044
Compiled intake of TCDD (pg)	160	16	209

* From Reference 24, based on 1965 household food consumption survey.

** Assuming 20 percent of total weight is fat.

*** From Reference 34, based on 1955 household food consumption survey. These figures are probably too low to represent high consumption of beef in 1980, but are the only estimates of high consumption in the literature.

of TCDD in several catfish at levels of 3-12 ppt. Although all of these findings were at or close to the limits of detection (see discussion in Exhibit 142), they strongly suggest the presence of TCDD in both rice and catfish from treated areas at levels around 4 ppt and 5 ppt respectively. The findings in catfish are particularly plausible, because TCDD has been reported in catfish in other studies (Ref. 37,38) and because catfish are known to accumulate residues of other persistent chlorinated chemicals (Ref. 1,2). Pending thorough field study under measured conditions of application of 2,4,5-T and TCDD, I have used 4 ppt and 5 ppt as the most likely average levels of TCDD in rice and catfish respectively, from treated areas. The correspondng daily intakes by average and high consumers of rice and catfish are shown in Table 4.

3. Rangeland, Forests and Rights-of-Way: Intake via Wild Game

Several studies of TCDD residues in deer have given inconsistent results. Young et al. (Ref. 40) found no residues of TCDD in deer, even in some areas where TCDD residues were high in soil and other wildlife. On the other hand, residues of TCDD at levels up to 68 ppt have been detected in fat tissues from deer and elk collected in forests treated with 2,4,5-T in Oregon and Washington (Ref. 28,37). In a controlled field trial in the Blodgett Forest in California (Ref. 41), deer were confined in an 11-acre enclosure, half of which was sprayed with 2,4,5-T at 3 lbs/acre. On sampling 2-28 days after spraying, three deer from

a total of 13 (which apparently included one or two control samples) had TCDD at levels in the range 1.6 to 4.6 ppt in their muscle tissue. Unfortunately the experiment was probably not continued for long enough to determine the full potential for accumulation of residues.

Data on TCDD in other wildlife are also inconsitent. Newton and Snyder (Ref. 42) found no residues of TCDD in mountain beavers (herbivorous mammals) 2 months after treatment of their forest habitat with 2,4,5-T; detection limits were 3-17 ppt. On the other hand, Meselson (Ref. 43) reported levels of TCDD in the range 10-237 ppt in wildlife samples (mostly insectivorous birds and mammals) from National Forests and rights-of-way.

It is difficult to use these data to estimate possible human exposure. The data in Reference 41 on deer muscle are the most directly relevant, because this is a tissue used for human food. A person eating 4 ounces (113 g) of venison containing 4.6 ppt of TCDD would ingest over 500 pg of TCDD. Although such intakes would occur only sporadically, they would be comparable in importance with other routes of exposure considered above.

4. Air and Water

TCDD has not been reported in air or water in the general environment. In certain specific situations where exposure to 2,4,5-T is very high, exposure to TCDD may be significant. For example, it was suggested above that daily intakes of 2,4,5-T by residents of forested areas might range up to 3,000

TABLE 4

ESTIMATED INTAKE OF TCDD FROM RICE AND FISH FROM TREATED AREAS

	Rice	Fish
Assumed concentration of TCDD (ppt)	4	5
Average consumer		
Daily intake of food (g)	11*	18**
Computed intake of TCDD (pg)	40	90
High consumer		
Daily intake of food (g)	200***	98****
Computed intake of TCDD (pg)	800	500

* From Reference 24.

** From References 23 and 39. 18 g is the mean daily intake of fish recorded in the 1965 household food consumption survey, and the mean daily intake recorded in a later survey of seafood consumption.

*** In the absence of specific published data on high consumption of rice. I have assumed that some persons (e.g., those of Chinese or Japanese descent) eat as much rice as the average U.S. consumption of wheat and wheat products. (Ref. 24).

**** This is the 99th percentile intake of seafood consumption reported in Reference 39. Holt (Ref. 1) on the basis of data from a third survey, used a figure of 107 g for the 99th percentile intake. He considered it applicable to rural dwellers in southern states who eat large quantities of catfish.

ug/day and 1,000 ug/day via air and water respectively. If TCDD is assumed to occur in air and water at the same ratio of concentrations to 2,4,5-T as it does in the commercial product (up to $5x10^{-8}$), then in the same circumstances intakes of TCDD might range up to 150 pg/day and 50 pg/day respectively. The validity of this assumption is questionable, because TCDD is less volatile and much less soluble in water than 2,4,5-T.*/ However, these properties may be offset by the greater persistence of TCDD.

5. Total Exposure Via All Routes

The estimates of exposure derived above are summarized in Table 5. All the estimates in Table 5 (with the exception of those for air and water) refer to consumers of food grown primarily or exclusively in areas treated with 2,4,5-T. Only a small fraction of range and pastureland in the United States is treated with 2,4,5-T and silvex each year, but I have seen precise figures only for rice. About 10-20% of the U.S. acreage used for rice is treated with 2,4,5-T and silvex each year. For the purposes of risk assessment, I would assume that the "average consumer" for which exposure estimates are presented in Table 5 is typical of a segment of the U.S. population that may include several million persons.

*/ It seems likely that TCDD in both air and water will occur primarily attached to micro-particulates rather than as vapor or in solution. The exact form is unimportant, however, because micro-particulates are a significant route of exposure for other chemicals.

6. Human Tissue Monitoring

There are several monitoring studies for TCDD in human tissues which can be used as a rough check on the estimates of human exposure to TCDD, or at least to provide an upper bound to the likely magnitude of exposure. To use the human tissue data in this way it is necessary to make assumptions about the relationship between intake of TCDD and the concentrations stored in the tissues. In rats, TCDD is stored in fat after long-term feeding at concentrations 4-24 times those in the diet (Ref. 44). In cows and sheep, corresponding storage factors for TCDD in fat after relatively short-term feeding are in the range of 2-4 (Ref. 31,45). Assuming that humans would store TCDD in their fat to a similar extent (2-24 times the dietary concentration), the estimated dietary concentrations of 0.2 and 0.7 ppt for average and high consumers would correspond to fat residues of TCDD in the ranges 0.4-5 and 1.4-17 ppt, respectively. Assuming that human milk contains about 4 percent fat, and that the concentration of TCDD in lipids would be similar in human milk and in human adipose tissue, these would correspond to levels of TCDD in milk in the ranges 0.02 - 0.2 ppt for average consumers and 0.06 - 0.7 ppt for high consumers.

Several studies presented in this proceeding are consistent with this prediction. Harless (Ref. 46) reported findings of 5-16 ppt TCDD in a single sample of human adipose tissue. Meselson (Ref. 43) reported "TCDD signals" corresponding to 11-31 ppt

TABLE 5

ESTIMATES OF HUMAN EXPOSURE TO TCDD
(DAILY INTAKES IN PG/KG/DAY)

	Average consumer*	High consumer*
Air and water	No data: possibly significant locally	No data: possibly significant locally
Beef	1.7	2.5
Milk	1.6	3
Rice	0.6	12
Fish	1.3	7
Wild game	Assumed small	Data inconsistent: possibly large
Total intake (pg/kg/day)	5.2	20**
Mean concentration in diet (ppt)***	0.2	0.7

* All estimates applicable to persons who obtain all their food from treated areas.

** Assuming that individuals would not be high consumers of all types of food.

*** Assuming 1.9 kg/day food intake (Ref. 24).

TCDD in the milk lipids of 4 of 17 women living in or near areas treated with 2,4,5-T.*/ Several other studies (Ref. 36,43,47 48,49) have failed to yield consistent identifications of TCDD in the milk of women living in areas where TCDD is used. In one case the detection limit was as low as 1-3 ppt in whole milk (Ref. 50), corresponding to about 25-75 ppt in milk lipids. A common defect in all these studies is that they failed to establish any connection between the women sampled and exposure to contaminated items (except for the fact that the women lived in areas where 2,4,5-T had been used). My exposure assessment suggests that the largest exposures to TCDD would be via food grown in treated areas, rather than from general environmental contamination. Despite this limitation, the data on human tissue residues are consistent with the values predicted on the basis of my exposure estimates. Put another way, the failure to detect TCDD in human milk at a detection limit of 1 ppt (i.e., about 25 ppt in milk lipids) would place an upper limit of about 10 ppt on the possible concentration of TCDD in the diet of the women concerned. Thus the monitoring of milk from women does not help much in refining estimates of average exposure levels. However, sampling from women known to have high intakes of beef, rice or fish from treated areas could help to refine estimates of exposure to highly exposed groups.

*/ According to Reference 49, the highest value reported by Meselson was from a sample of milk from a women living in Italy. Meselson reported positive findings in milk from 3 women living in Kansas and Texas. Other laboratories analyzed these samples, but at lower sensitivity.

7. Concluding Comments

1. With the exception of a few samples of beef fat and a number of wildlife samples, all of the measurements of TCDD utilized in this exposure assessment have been at or close to the detection limits. Accordingly their precision and reliability have been questioned. Based on the criteria of analytical replicability, mutual consistency, scientific plausibility, and analogy with better studied chemicals, I would categorize the positive findings of TCDD in beef fat as of very high probability, those in fish as of high probability, and those in rice and deer as of moderate probability. My estimates of exposure levels are made with corresponding degrees of scientific confidence.

2. I estimate the exposure of typical persons consuming food grown in treated areas as of the order of 5 pg/kg/day, or about 0.2 ppt in the diet. Although this estimate is based on a very small number of confirmed measurements, I believe that it is of the right order of magnitude. If the average exposure were in fact much greater (say 20 pg/kg/day) we would expect TCDD to be found much more consistently and at higher levels in samples of food and human milk. If the average exposure were much smaller (say 1 pg/kg/day) one would not expect to find it at all.

3. Two limitations on all the studies utilized here is their failure to estabish specific links between the samples analyzed and the application of 2,4,5-T, and their failure to determine the level of TCDD in the 2,4,5-T that was used. Controlled field trials with measured application rates of TCDD (including

exaggerated application rates to overcome the insensitivity of existing analytical methodology) would be necessary to establish that human exposure to TCDD is lower than that estimated here. The complete absence of such controlled trials is surprising.

D. Occupational Exposure to 2,4,5-T

Chapter II of the Clement report included a review of the scanty data available on occupational exposure and an attempt to estimate the likely magnitude of exposure to 2,4,5-T and TCDD under various occupational circumstances. EPA's "Position Document 1" (Ref. 3) had included attempts to calculate occupational exposure by making various assumptions about conditions of application of 2,4,5-T in the field. Although we attempted to amend EPA's calculation by making more realistic assumptions, we concluded that the lack of actual data made this theoretical approach very uncertain. We placed greater weight in the results of a study by Shafik et al. (Ref. 52), in which 2,4,5-T was measured in the urine of workers. Although this study was limited by the lack of information about the circumstances of exposure, we used the results to make rough estimates of the likely magnitude of the exposure of these workers to 2,4,5-T.

Since May 1979, I have reviewed three other studies in which 2,4,5-T was measured in the urine of applications. Ramsey et al. (Ref. 25) studied 21 workers who applied a 2,4,5-T ester by various techniques; the workers used their normal spray routines and the applications lasted for 55-245 minutes on one day. The authors collected urine samples on the day before

spraying and for 5 or 6 days after spraying. On the basis of the difference in excretion rates of 2,4,5-T before and after spraying, the authors estimated the total doses of 2,4,5-T absorbed by each worker. The means of the estimated doses for workers in various job categories were as follows: flagmen, 0.002 mg/kg; supervisors, 0.011 mg/kg; helicopter pilots, variable; tractor drivers, 0.045 mg/kg; backpack sprayers, 0.063 mg/kg; mixers, 0.073 mg/kg. The highest single figure was for a mixer (0.156 mg/kg). These estimates do not appear to be very precise (because the excretion rates were very variable and were still high after 6 days, inter alia), but they are probably of the right order of magnitude.

Simpson et al. (Ref. 53) measured urinary excretion of 2,4,5-T in six forest workers who applied the chemical either with injector guns, with engine-powered knapsack misters, or with power sprays. Average concentrations of 2,4,5-T in the urine of the workers were in the range 0.160 to 1.740 mg/l. Assuming total urinary volume of about 1.5 liters/day, these workers would then have excreted between 0.24 and 2.6 mg of 2,4,5-T in the course of a day. Kolmodin-Hedman et al. (Ref. 54). reported a similar study in four workers applying 2,4,5-T with a fan sprayer mounted on a tractor. Urine was collected during a work-week and for 36 hours thereafter. The average concentration of 2,4,5-T in urine was 2.5 mg/l with a range of 0.4 to 11.4 mg/l. Again assuming total urinary volume of about 1.5 liters/day,

the corresponding excretion rates would have been between 0.6 and 17 mg/day with a mean of 3.75 mg/day.

None of these studies gives a precise measure of exposure. The study by Ramsey et al. gave very variable results and was probably terminated too soon for a complete measure of excretion. The other two studies gave insufficient data about the work practices and work history. In theory, if a worker is exposed to a similar degree every day, a roughly steady state would develop in which intake and excretion are balanced. On the other hand, if exposure is intermittent, the results of Ramsey et al. show that the material taken in over one day is excreted over at least a 6-day period; hence the amount excreted per day will be less than the amount taken in on each day of exposure. For this reason, the results of the studies by Simpson et al. and Kolmodin-Hedman et al. may underestimate actual exposures. Nevertheless, the results of all three studies are of the same order of magnitude, suggesting intake at the range 0.01-0.25 mg/kg/day by exposed workers, depending on the type of job. These figures, however could be too low or too high by factors up to 3 or more. Information on the exact conditions of exposure is inadequate for detailed analysis.

E. Occupational Exposure to Silvex

I have not found any specific studies of occupational exposure to silvex. In the absence of such data, it seems

reasonable to assume that occupational exposure to silvex would be similar in magnitude to occupational exposure to 2,4,5-T, when the two pesticides are applied in similar ways.

F. Occupational Exposure to TCDD

It seems a reasonable assumption that applicators will be exposed to TCDD and to 2,4,5-T in the same ratio as they occur in the technical mixture -- i.e. in the ratio of no more than 5×10^{-8} to 1. Accordingly, if it can be assumed that the two chemicals are similarly absorbed into the body (through the skin and/or lungs), the intakes of the two chemicals should be in the same ratio. Then an intake of 0.25 mg/kg/day of 2,4,5-T would correspond to an intake of no more than 12 pg/kg/day of TCDD. (This is in the same range as the figures derived in Section II.C) for likely dietary exposures to the general population.) Unfortunately it is not known whether TCDD and 2,4,5-T are similarly absorbed. Hence this figure is even more uncertain than the figure of 0.25 mg/kg/day 2,4,5-T from which it was derived.

G. Accidental Exposure to Sprays

A final category of exposure to be considered is that of persons who may be accidentally exposed to a direct spray application of 2,4,5-T or silvex. One example is that of a person who is in the forest or beside a rice field when a spray helicopter flies over. Calculation of potential magnitude of exposure to such persons requires many uncertain assumptions about the conditions of exposure. The most appropriate analogy would be

the exposure of flagmen, but the flagmen studies by Ramsey et al. (Ref. 25) do not appear to have had significant exposure. It is unlikely that a single accidental exposure of this kind would exceed that experienced by a tractor driver in the course of a day's work. However, in the absence of actual data, I am not able to make a numerical estimate of exposure.

H. Summary and Conclusions

1. This statement includes an assessment of the degree and extent of human exposure to 2,4,5-T, silvex, and TCDD, expected to result from various uses of 2,4,5-T and silvex.

2. Because estimation of average exposure to the general population is very difficult, this statement is concerned primarily with estimation of average exposures to highly exposed sub-groups in the population, and of maximum plausible exposures. Several different types of "high exposure" estimates are included in this statement. The various estimates in Tables 1 and 5 are not directly comparable with each other and should be read in conjunction with the discussion in the text where their applicability is explained.

3. This assessment is based primarily on ambient monitoring studies, although in some cases specific field studies of residues of 2,4,5-T and TCDD proved useful. Target monitoring (measurement of residues in human tissues) is used to estimate the magnitude of occupational exposure, and to establish upper limits on the likely magnitude of general population exposure.

4. As in all exposure assessments, the precision of this assessment is limited by the sporadic occurrence of residues of 2,4,5-T, silvex, and TCDD in the environment, and by the consequent variability in human exposure. This assessment is further limited by the fact that many reported residues were close to the limit of detection, and by the poor quality of many of the field studies.

5. Estimates of human exposure to 2,4,5-T are summarized in Section D (occupational expsoure) and Section A (non-occupational exposure). Intakes resulting from occupational exposure range up to roughly 0.25 mg/kg/day, the precise magnitude depending on the job category, the precautions taken, and the duration of exposure. There is insufficient information to estimate average exposures to the general population, except that average exposures via all routes are unlikely to exceed 0.2 mg/kg/day. Exposures via air, water, and food (meat, milk, fish, and game) may be much higher than this in areas where 2,4,5-T is used. These estimates are summarized in Table 1.

6. There is insufficient information to estimate human exposure to silvex directly. It appears reasonable to assume that the magnitude of exposure to silvex will be similar to the magnitude of exposure to 2,4,5-T when they are used in similar ways, although fewer persons are expected to be exposed to silvex.

7. Exposure of the general population to TCDD is expected to be primarily via the diet although air and water may be significant locally. On the basis of information available at

present, beef, milk, fish, and rice are likely to be significant sources of exposure. For persons who obtain most or all of their food from treated areas, average exposures to TCDD are expected to be of the order of 5 pg/kg/day (up to 20 pg/kg/day in high consumers of beef, milk, fish, and/or rice). These estimates are summarized in Table 5. Occupational exposure to TCDD is expected to range up to roughly 12 pg/kg/day.

8. There is insufficent information to estimate the magnitude of exposure to persons who are accidentally sprayed during field applications.

Ian C.T. Nisbet/pas

Ian C.T. Nisbet

REFERENCES

1. Holt, B.R. 1977. Population Exposures to Toxaphene (Chlorinated Camphenes) CRESS Report No. 36. Stanford Research Institute Final Report prepared for U.S. EPA under Contract No 68-01-4314.

2. Suta, B.E. 1977. Human Population Exposures to Mirex and Kepone. Environmental Health Effects Research Series. EPA-600/1-78-045.

3. 2,4,5-T Working Group, U.S. Environmental Protection Agency. 1978. 2,4,5-T: Position Document 1. Federal Register 43(78):17118-17147 (EPA Exhibit 235).

4. USDA-States-EPA (1979) The Biologic and Economic Assessment of 2,4,5-T. A Report of the USDA-States-EPA 2,4,5-T RPAR Assessment Team.

5. Response of Dow Chemical U.S.A. to Notice of Rebuttable Presumption Against Registration and Continued Registration of Pesticide Products Containing 2,4,5-T. 1978.

6. Direct Testimony of Dr. Frederick Kutz (EPA Exhibit 131).

7. Bamesberger, W.L. and D.F. Adams. 1966. An atomospheric survey for aerosol and gaseous 2,4-D compounds p. 219-227. In: R.F. Gould (Ed.) Organic pesticides in the environment. Ad. Chem. Series, Am. Chem. Soc.

8. Johnson, J.E. 1971. The public health implications of wide spread use of the phenoxy herbicides and picloram. Bio Science. 21(17): 899-905.

9. Marrigold, D.B., and Schulze, J.A. 1969. Pesticides in Selected Western Streams - and progress report. Pestic. Monit. J. 3(2):124-135.

10. Schulze, J.A., Marigold, D.B., Andrews, F.L. 1973. Pesticides in Selected Western Streams - 1968-71. Pestic. Monit. J. 7(1):73-84.

11. Direct Testimony of Mr. Thomas E. Dixon (EPA Exhibit 170).

12. Duggan, R.E., H.C. Barry, and L.Y. Johson. 1966. Pesticide residues in total diet samples. Science 151 (3706):101-104.

13. Duggan, R.E., H.C. Barry, and L.Y. Johnson. 1967. Pesticide residues in total diet samples (II). Pestic. Monit. J. (2):2-12.

14. Martin, R.J. and R.E. Duggan. 1968. Pesticide residues in total diet samples (III). Pestic. Monti. J. 1(4):11-20.

15. Corneliussen, P.E. 1969. Pesticide residues in total diet samples (IV). Pestic. Monit. J. 2(4):140-152.

16. Corneliussen, P.E. 1970. Pesticide residues intotal diet samples (V). Pestic. Monit. J. 4(3):89-105.

17. Corneliussen, P.E. 1972. Pesticide residues in total diet samples (VI) pestic. Monit. J. 5(4):313-330.

18. Manske, D.D., and P.E. Corneliussen. 1974. Pesticide residues in total diet samples (VII). Pestic. Monti. J. 8(2):110-124.

19. Manske, D.D., and R.D. Johnson. 1975. Pesticide residues in total diet samples (VIII). Pestic. Monit. J. 9(2):94-105.

20. Johnson, R.D., and D.D. Manske. 1975. Pesticide residues in total diet samples (IX). Pestic Monti. J. 9(4):157-169.

21. Manske, D.D. and Johnson, R.D. 1977. Pesticide and Other Chemical Residues in Total Diet Samples (X) Pestic. Monit. J. 10(4):134-148.

22. Johnson, R.D. and Manske, D.D. 1977. Pesticide and Other Chemical Residues in Total Diet Samples (XI). Pesti. Monit. J. 11(3):116-131.

23. United States, Department Agriculture. 1972. Food Consumption of Households in the United States, Seasons and Year 1965-66. pp. 8 and 9.

24. Memo: Update of Food Factor Tables, dated May 1, 1978, From R.D. Schmitt, Toxicology Branch, EPA to Acting Chief, Toxicology Branch EPA.

25. Ramsey J.C., Lvay, T.L., and Braun, W.H., Exposure of Forest Workers to 2,4,5-T Calculated Dose Levels.

26. Cochrane, W.P., Greenhalgh, R., Looney, N.E. 1976. Residues in Apples Sprayed with Fenoprop. Cdn. J. Plant Sci. 56 207-210 (Beroza Reference # 1).

27. Meselson, M.S., O'Keefe, P.W., and Baughman, R.W. 1978. The evaluation of possible health hazards from TCDD in the environment. In: Symposium on the on the Use of Herbicides on Forestry (21-22 February 1978). U.S. Department of Agriculture, Washington, D.C. pp. 91-94 (EPA Exhibit 190).

30. Kocher, C.W., Mahle, N.H., Hummel, R.A., Shadoff, L.A. and Getzendaner, M.E. 1978. A search for the presence of 2,4,7,8-tetrachlordibenzo-p-dixon in beef fat. Bull. Environ. Contam. Toxicol. 19(2): 228-236 (EPA Exhibit 162).

31. Jensen, D.J., Hummel, R.A., Mahle, N.H., Kocher, C.W. 1978. A residue Study on beet cattle consuming 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). Unpublished. The Dow Chemical Company (EPA Exhibit 159).

32. Jensen, D.J., Hummel, R.A., Higgins, H.S., Lamparski, L., Madrid, E.T. 1978. Secretion of TCDD in milk and cream following the feeding of TCDD to lactafing diary cows. Unpublished. The Dow Chemical Company. (EPA Exhibit 161).

33. Mahle N.H., Higgins, H.S., Getzendaner, M.E. 1977. Search for the presence of 2,3,7,8-tetrachlorodibenzo-p-dioxin in bovine milk Bull. Environ. Contam. Toxicol. 18(2): 123-30.

34. United States Department of Agriculture, Agricultural Research Service. 1960. High Consumption of Foods.

35. Jensen, D.J., et al. July 1978. Analysis for TCDD residues in rice grain from retail stores and from fields treated with 2,4,5-T. Unpublished. The Dow Chemical Company (EPA Exhibit 163).

36. Shadoff, L.A., Hammel, R.A., Lampurski, L., and Davidson, J.H. 1977. A search for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in an environment exposed annually to 2,4,5-trichlorophenoxyacetic acid ester (2,4,5-T) herbicides. Bull. Environ. Contam. Toxicol. 18:478-485 (EPA Exhibit 158).

37. Direct Testimony of Mr. Robert L. Harless (EPA Exhibit 212).

38. Result of Capillary Column GC/HRMS Analyses Performed on Extracts of Sediment, Soil, and Fish for 2,3,7,8-TCDD Residues. Memo to Mike Dellarco, Dioxin Project Manager, February 11, 1980, from Robert Harless. Exhibit 2220).

39. Presentation of TRF Seafood Consumption Study. September, 1973-August, 1974 (Revised january, 1975). Presented by John Malec, National Purchase Diary.

40. Young A. et al. 1978. The Toxicology, Environmental Fate and Human Risk of Agency Orange and Its Associated Dioxin. (EPA Exhibit 150).

41. Gross, M.L. 1979. Final Report. Ultratrace Analysis of Tetrachlorodibenzo-p-dioxin in Samples of Deer Tissue by Gas Chromatograph/High Resolution Mass Spectrometry U.S. Forst Service Contract No. 53-915B-8-6133. July 10, 1979. (EPA Exhibit 227).

42. Newton, M., and Snydr, S.P. 1978. Exposure of forest herbivores to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in areas sprayed with 2,4,5-T. Bull. Environ. Contam. Toxicol. 20:743-750.

43. Meselson, M. 1978. "TCDD Analysis in Environmental Samples." Attachment F, Submitted as part of 2,4,5-T RPAR rebuttal of the Environmental Defense Fund (EPA Exhibit 156).

44. Kociba, R.J., Keyes, D.G., Carreon, R.M., Wade, C.E., Dittenber, D., Kalnins, R., Frauson, L., Park, C.N., Hummel, R., and Humiston, C.G. 1978a. Results of a Two-year Chronic Toxicity and Oncogenicity Study of 2,3,7,8-Tetrachlorodibenzo-p-dioxin in Rats. Toxicol. Appl. Pharmacol. 46: 279-303 (EPA Exhibit 13).

45. Jensen, D.J., R.A. Hummel, H.S. Higgins, L. Lamparski, E. Madrid. 1978. A Residue Study on Sheep Consuming 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD). Unpublished. The Dow Chemical Company. (EPA Exhibit 160).

46. Results of Capillary Column GC/HRMS Analysis Performed on extracts of Human Adipose Tissue for 2,3,7,8-TCDD. Residues (Needle Biopsy Feasibility Study). Memo to Mike Dellarco, Dioxin Project Manager, February 26, 1980, from Robert Harless. (EPA Exhibit 218).

47. Direct Testimony of Dr. Michael Gross. (EPA Exhibit 223).

48. EPA. 1980. Result's of Mother's Milk Study (EPA Exhibit 219).

49. National Institute of Environmental Health Sciences (McKinney, J.D.) February 13, 1979. TCDD in Human Milk Study (with attachments) (EPA Exhibit 193).

50. Report of the Panel Reviewing Investigation of TCDD in Human Milk. December, 1979 (Exhibit 1063).

51. Cheney, H.U., Walby, C.M., Shields, R.E. 1979. Impact of 2,4,5-T on Blogdett Forest I. Description of an Experimental Aerial Application of 2,4,5-T.

52. Shafik, M.T., Sullivan, H., Enos, H.F. 1971. A method for determination of low levels of exposure to 2,4-D and 2,4,5-T. Intern. J. Environ. Anal. Chem. 1: 23-33 (EPA Exhibit 132).

53. Simpson, G.R., Higgins, U., Chapman, J , Bermingham, S. 1978. Exposures of council and forestry workers to 2,4,5-T. Med. J. Augt. 2(u): 536-7.

54. Kolmodin-Hedman, B., Erne, K., Hokansson, M., and Engqvist. 1979. Control of Occupational Exposure to Phenoxy Acids (2,4-D and 2,4,5-T) Vetenskaplig Skriftserie, 17 (26 pages, numbers not given)

55. Nisbet, I.C.T., 1976. Human Exposure to Chlordane, Heptachlor, and Their Metabolites. Unpublished report to the U.S. Environmental Protection Agency.

56. National Academy of Sciences. 1979. Kepone/Mirex/ Hexachlorocyclopentadiene: An Environmental Assessment, Washington, D.C., Chapter 2, pages 32-37.

CERTIFICATE OF SERVICE

I hereby certify that copies of the foregoing DIRECT TESTIMONY OF DR. IAN C.T. NISBET were hand delivered or mailed first class postage prepaid on May 15, 1980 to the following persons:

Edward W. Warren
L. Mark Wine
Richard L. McConnell, Jr.
Kirkland & Ellis
Counsel for Dow Chemical Company
1776 K Street, N.W., 12th Floor
Washington, D.C. 20006

William A. Butler, Esq.
Jacqueline M. Warren, Esq.
Counsel for Environmental Defense Fund, Inc.
1525 - 18th Street, N.,W.
Washington, D.C. 20036

Margaret M. Brienholt
Judith A. Wenker
Terrence G. Jackson
Room 2036, South Ag. Bldg.
Office of the General Counsel
U.S. Department of Agriculture
Washington, D.C. 20250

Marla Gillham
Northwest Coalition for Alternatives to Pesticides, Inc.
454 Willamette Street
Eugene, Oregon 97401

Allen A. Lauterbach
John J. Rademacher
American Farm Bureau Federation
425 - 13th Street, N.W.
Washington, D.C. 20004

Robert S. Kirk, Jr., Esq.
Counsel for Vertac, Inc.
2414 Clark Tower
5100 Poplar Avenue
Memphis, Tennessee 38137

Richard J. Wertheimer, Esq.
Arnold & Porter
Counsel for National Forest Products Association
1229 Nineteenth Street, N.W.
Washington, D.C. 20036

Stephen W. Jacobson
Joseph E. Stevens, Jr.
William Ray Price, Jr.
Lathrop, Koontz, Righter, Clagett, Parker & Norquist
2600 Mutual Benefit Life Bldg.
P.O. Box 1200
2345 Grand Avenue
Kansas City, Missouri 64108

Sonia G. Anderson
Hearing Clerk (A-110)
U.S. Environmental Protection Agency
401 M Street, S.W.
Washington, D.C. 20460

Elizabeth M. Whelan, Sc.D., M.P.H.
Executive Director
American Counsel on Science and Health
1995 Broadway
New York, New York 10023

Patricia A. Roberts

Patricia A. Roberts

May 15, 1980

CURRICULUM VITAE

IAN C. T. NISBET

BIOGRAPHICAL SUMMARY: Dr. Ian Nisbet is an environmental scientist with an unusually broad range of experience, including professional work in physics and engineering (1954-1968), in ecology and ecotoxicology (1965 to the present), and on the fate and effects of toxic chemicals (1970 to the present). He is currently employed by Clement Associates, where he supervises a wide range of work on the fate and effects of toxic chemicals, and by the Massachusetts Audubon Society, where he directs a broad program in environmental sciences. In addition to research and consulting activities, he has had extensive experience in the regulation of toxic chemicals, including the drafting of regulations and the conduct of formal and informal public hearings and court review proceedings. Dr. Nisbet is internationally recognized as an expert on the transport and fate of chemicals in the environment. He has served as a member of numerous expert committees, including several committees and boards of the National Academy of Sciences. Dr. Nisbet is now specializing in the assessment of human exposure to chemicals and in the estimation of risks, especially of cancer, posed by chemical exposure. He is also actively conducting research in ecology and ecotoxicology.

EDUCATION

Postdoctoral Fellow, Applied Mathematics and Theoretical Physics, Cambridge University, 1959-1962

Postdoctoral Fellow, Mechanical Engineering, Massachusetts Institute of Technology, 1958-1959

Postdoctoral Fellow, Aeronautical Engineering, Cornell University, 1957-1958

Ph.D., Physics and Statistical Mechanics, Cambridge University, 1958

M.A., Physics and Statistical Mechanics, Cambridge University, 1958

B.A., Mathematics and Physics, Cambridge University, 1954

EMPLOYMENT HISTORY

1979-present	Vice-President and Principal Science Advisor, Clement Associates. Responsible for scientific oversight and quality control of a rapidly growing group of scientific regulatory consultants and for conduct of specialized programs investigating human exposure and risk assessment.
1975-present	Director, Scientific Staff, Massachusetts Audubon Society. Responsible for the direction of a small group of scientists conducting a broad program of research and analysis in environmental sciences.
1976-1979	Senior Science Advisor, Clement Associates. Responsible for the development and scientific direction of consulting activities in the field of toxic chemicals.
1968-1975	Associate Director, Scientific Staff, Massachusetts Audubon Society. Developed a research program in ecology and ecotoxicology.
1973-1979	Associate in Biology, Museum of Comparative Zoology, Harvard University. Honorary appointment in recognition of research in ecology and ecotoxicology.
1970-1972	Lecturer in Biology, Harvard University. Conducted graduate seminar in ecology.
1963-1968	Lecturer and Senior Lecturer in Physics, University of Malaya. Taught courses and conducted research in biophysical ecology.
1962-1963	Research Associate, Massachusetts Audubon Society. Conducted research in biophysical ecology.
1959-1962	NATO and ICI Research Fellow, Cambridge University. Conducted research in fluid and plasma mechanics.

PROFESSIONAL ACTIVITIES

Member, Board on Toxicology and Environmental Health Hazards, and member of Subcommittee on Critical Issues of the 1980's, National Academy of Sciences/National Research Council (NAS/NRC), 1979-present

Participant, Workshop on Long Range Environmental Outlook, NAS/NRC, 1979

Participant, Workshop on Exposure Assessment for Toxic Chemicals, U.S. Environmental Protection Agency, 1978

Member, Committee on the Assessment of Polychlorinated Biphenyls in the Environment, NAS/NRC, 1978-1979

Chairman, Committee on Scientific and Technical Assessments of Environmental Pollutants, NAS/NRC, 1975-1978

Participant, Symposium on Global Environmental Monitoring System, International Environmental Programs Committee, NAS/NRC, 1977

Consultant, Scientific Committee on Problems of the Environment (SCOPE), Project on Eco-Toxicology, International Council of Scientific Unions (ICSU), 1976-1977

Member, Second Task Force for Research Planning in the Environmental Health Sciences, 1976

Participant, Conference on Air Quality and Automobile Emissions, NAS/NRC, 1975

Member, Committee on Air Quality and Stationary Source Emission Control, NAS/NRC, 1975

Member, Study Panel on Assessing Potential Ocean Pollutants, NAS/NRC, 1973-1974

Member, Working Group on Environmental Assessment and Monitoring, SCOPE, ICSU, 1973

Member, Working Conference on Principles of Protocols for Evaluating Chemicals in the Environment, NAS/NRC, 1972-1973

Member, Panel on Hazardous Trace Substances, Office of Science and Technology/National Institute of Environmental Health Sciences, 1970-1973

MEMBERSHIP IN SOCIETIES

American Association for the Advancement of Science
American Institute of Biological Sciences
American Ornithologists' Union
British Ecological Society
British Ornithologists' Union
Cooper Ornithological Society
Ecological Society of America
New York Academy of Sciences
Society of American Naturalists
Society for the Study of Animal Behaviour
Society for the Study of Evolution

PUBLICATIONS--ECOLOGY AND ECOTOXICOLOGY

Dr. Nisbet is the author of numerous scientific publications in ecology and ecotoxicology, including more than 50 technical papers in scientific journals and a number of review papers and articles in books, symposia, and conference proceedings. A complete list of publications is available on request.

PUBLICATIONS--TOXIC CHEMICALS

In addition to papers on ecotoxicology, Dr. Nisbet is the author or coauthor of numerous scientific publications on the transport, fate, and effects of toxic chemicals in the environment. His principal publications are the following:

Nisbet, I.C.T. Carcinogenic risk assessment: An overview. Invited paper presented at a symposium on carcinogenic risk assessment. American Chemical Society, Washington, D.C. (September 1979)

Sarofim, A.F., Nisbet, I.C.T., Neely, B., Eschenroeder, A., Walsh, P.J., and Gilbertson, M. Exposure models. Chapter 4 in Proceedings of a Workshop on Exposure Assessment. Office of Research and Development, U.S. Environmental Protection Agency, Washington, D.C. (1980)

Committee on the Assessment of Polychlorinated Biphenyls in the Environment. Polychlorinated Biphenyls in the Environment. National Academy of Sciences/National Research Council, Washington, D.C. (1979)

Nisbet, I.C.T. Regulation of occupational carcinogens. In Proceedings of a Conference on Cancer in the Environment. Florida Audubon Society, Maitland, Fla. (1979)

Nisbet, I.C.T. Ranking chemicals for testing: A priority setting exercise under the Toxic Substances Control Act. In Proceedings of the Interagency Testing Committee Workshop, San Antonio, Texas (1979)

Nisbet, I.C.T. PCBs in Massachusetts and their implications for environmental health. In Proceedings of a Symposium on Drinking Water and Health. Massachusetts Rehabilitation Hospital/Massachusetts Audubon Society/U.S. Environmental Protection Agency, Boston, Mass. (1979)

Livingston, R.L., Matsumura, F., Nisbet, I.C.T., and Williams, G. Mirex, Kepone, and hexachlorocyclopentadiene: An environmental assessment. National Academy of Sciences/National Research Council, Washington, D.C. (1978)

Nisbet, I.C.T. Environmental transport and occurrence of PCBs in 1975. In Proceedings of the National Conference on Polychlorinated Biphenyls, Chicago, November 1975. Research Triangle Institute (for the U.S. Environmental Protection Agency), Research Triangle Park, N.C., pp 254-256 (1976)

Nisbet, I.C.T. Ecological effects. In Air Quality and Stationary Source Emission Control. Commission on Natural Resources, National Academy of Sciences/National Academy of Engineering, National Research Council, Washington, D.C., pp 170-187 (1975)

Nisbet, I.C.T. Sulfates and acidity in precipitation: Their relationship to emissions and regional transport of sulfur oxides. In Air Quality and Stationary Source Emission Control. Commission on Natural Resources, National Academy of Sciences/National Academy of Engineering, National Research Council, Washington, D.C., pp 276-312 (1975)

Tucker, E.S., Nisbet, I.C.T., Brooks, J.M., Hinson, M.O., Passino, D., Moolenaar, R., Spaulding, R., Risebrough, R.W., and Vermeulen, T. Synthetic organic chemicals. In Assessing Potential Ocean Pollutants: A Report of the Study Panel on Assessing Potential Ocean Pollutants to the Ocean Affairs Board. Commission on Natural Resources, National Research Council. National Academy of Sciences, Washington, D.C., pp 64-227 (1975)

Nisbet, I.C.T., Fleischer, M., Sarofim, A.F., Fassett, D.W., Hammond, P., Shacklette, H.T., and Epstein, S. Environmental impact of cadmium: A review by the Panel on Hazardous Trace Substances. Environ. Health Perspect. May:253-323 (1974)

Nisbet, I.C.T., Levin, S., and Chen, C. Simulated systems. In Principles for Evaluating Chemicals in the Environment: A Report of the Committee for the Working Conference on Principles of Protocols for Evaluating Chemicals in the Environment. Environmental Studies Board, National Academy of Sciences/National Academy of Engineering, and Committee on Toxicology, National Research Council. Washington, D.C., pp 231-239 (1973)

Nisbet, I.C.T., Hammond, P.B., and Sarofim, A.F. Polychlorinated biphenyls: Environmental impact. Environ. Res. 5:249-362 (1972)

Nisbet, I.C.T., and Sarofim, A.F. Rates and routes of transport of PCBs in the environment. Environ. Health Perspect. April: 21-38 (1972)

CONSULTING ACTIVITIES (including regulatory experience)

1980-present Council on Environmental Quality (CEQ) (reproductive hazards). Dr. Nisbet is directing a team of Clement scientists and consultants in an assessment of the importance of toxic chemicals as causes of reproductive failure in the human population. This work involves the critical review of health statistics, epidemiological and toxicological data, information on exposure to toxic chemicals, and regulatory options.

1980-present U.S. Environmental Protection Agency (EPA) (air carcinogen policy). Dr. Nisbet is the senior member of a Clement team providing technical assistance to EPA in the development of a generic policy for regulating carcinogenic air pollutants. This requires an analysis of scientific data relevant to the identification and regulation of carcinogens and specifying unresolved scientific issues.

1979-present	Occupational Safety and Health Administration (OSHA) (cancer policy), J-9-P-9-0196. Dr. Nisbet is one of a team of Clement scientists that is reviewing data on toxicity and exposure for a large number of chemicals. The Clement group is helping OSHA to prepare a candidate list of occupational carcinogens and to establish priorities for regulation.
1979	EPA (pesticide reviews), WA78-B317. Clement is performing a critical review of data on the toxicology and pathology of 85 pesticides. Dr. Nisbet is leader of the Risk Assessment Team and Chairperson of the Quality Control Board that provides scientific oversight for the entire project. As Team Leader, he has prepared reports on risk assessment methodology and on a risk assessment for hexachlorobenzene (HCB).
1979	EPA (pesticide monitoring), 68-01-5095. Dr. Nisbet directed a team of Clement scientists that reviewed data on 2,4,5-T, 2,4,5-TCP, and TCDD and prepared extensive critical reports on these chemicals. This work included assessment of environmental chemistry and fate, toxicology, environmental and occupational exposure, and risk assessment, including estimation of carcinogenic risks.
1979	OSHA (benzene). Dr. Nisbet provided technical assistance to OSHA in analyzing scientific evidence on risks of cancer posed by occupational exposure to benzene for use in briefs for the Supreme Court.
1979-present	EPA (PCBs). Dr. Nisbet is studying human exposure to PCBs from fish in Lake Michigan and is assessing potential risks resulting from this exposure.
1976-1980	OSHA (cancer policy), J-9-F-7-099. Dr. Nisbet directed a Clement team of scientists that provided technical assistance to OSHA throughout the development of a cancer policy, including review of underlying data, drafting technical sections of the preamble to the proposed regulation, locating witnesses, assisting with preparation of testimony, serving as expert witness,

analyzing comments presented by industry and other parties, examining witnesses at the public hearing, analyzing the record, and drafting technical sections of the preamble to the final regulation.

1978 — U.S. Congress Office of Technology Assessment (priority setting for chemicals in food), OTA-C-78-372. Dr. Nisbet served as a member of a team of scientists that reviewed methods of setting priorities and prepared a detailed report recommending methods to be used in establishing priorities for monitoring potential food contaminants.

1977-1978 — National Institute for Occupational Safety and Health (NIOSH), 21-77006-0000. Dr. Nisbet directed a small team of Clement scientists that prepared complete critical reviews of the toxicology of DDT and aldrin/dieldrin. He was the principal author of two extensive reports that were published by NIOSH in 1978 as Special Occupational Hazard Reviews.

1976-1977 — EPA (heptachlor/chlordane), WA7-1319-A. Dr. Nisbet prepared a critical analysis of data on human exposure to heptachlor/chlordane and their metabolites. This analysis was used by the Carcinogen Assessment Group as the basis for its carcinogenic risk assessments. Dr. Nisbet testified as an expert witness in public hearings on the cancellation of these two pesticides.

1977-1978 — National Science Foundation/Council on Environmental Quality (TSCA Interagency Testing Committee), NSF ENV-77-15417, EQ8AC013. Dr. Nisbet led a team of Clement scientists that provided technical assistance to the Interagency Testing Committee in listing, screening, evaluating, and prioritizing chemicals for testing under Section 4(e) of TSCA. This work was described in the Committee's Initial Report to the Administrator of EPA (42 FR 55026).

1976-1977 — EPA (chlorobenzilate, safrole, and BAAM), 76-B258, WA-7-1304-A. Dr. Nisbet was director of a small Clement team that prepared detailed analyses, including statistical analyses, of carcinogenesis bioassays of these three pesticides.

1976	EPA (effluent standards for PCBs). Dr. Nisbet reviewed data on the chemistry, environmental behavior, and toxicity of polychlorinated biphenyls for the EPA Office of Water and Hazardous Materials. He was the sole author of EPA's Criteria Document for PCBs and the principal scientific consultant to EPA during public hearings on proposed effluent standards under Section 307a of the Federal Water Pollution Control Act. In this position, he testified as an expert witness, assisted in the preparation of expert testimony and of the cross-examination of opposing witnesses, analyzed the record of the hearing, and drafted proposed findings of fact. These findings were incorporated by the Administrator into his opinion and later upheld by the U.S. Court of Appeals.
1975-1976	EPA (heptachlor/chlordane). Dr. Nisbet was principal scientific consultant to the EPA Office of the General Counsel in hearings on the suspension of heptachlor/chlordane. He assisted in the preparation of witnesses' direct testimony, the cross-examination of opposing witnesses, and the rebuttal testimony. He was the principal author of the analytical sections of EPA's post-hearing brief.
1974	EPA (aldrin/dieldrin suspension). Dr. Nisbet was principal scientific consultant to the Office of the General Counsel in hearings on the suspension of aldrin/dieldrin. His responsibilities were the same as those described above.
1973-1974	Environmental Defense Fund (EDF) (aldrin/dieldrin cancellation). Dr. Nisbet was principal scientific consultant to EDF in hearings before EPA on the cancellation of aldrin/dieldrin. His responsibilities were similar to those described above, and he was principal author of the analytical sections of two EDF briefs.
1974	EDF (DDT cancellation). Dr. Nisbet was principal consultant to EDF in hearings before EPA on the cancellation of DDT. His responsibilities were similar to those described above.

In addition to the work for government agencies summarized above, Dr. Nisbet has consulted for a number of private clients on toxic chemical regulation, especially in the areas of exposure assessment and carcinogenic risk assessment. This work has included:

- Participation in an in-depth review of the environmental chemistry and toxicology of a new pesticide and the potential risks posed by it
- Calculation of potential human exposure to this pesticide and carcinogenic risks posed by several proposed uses
- Calculation of potential carcinogenic risks associated with use of a hair-dye component
- Tabulation of several hundred chemicals reliably reported to be carcinogenic, together with data on human exposure to these chemicals and numerical risk estimates for 18 chemicals presently unregulated
- Assessment of potential risks posed by a mutagenic flame retardant
- Assessment of possible risks associated with contamination of water supplies by a chlorinated solvent
- Critical review of toxicological data and structure-activity analysis and identification of potential hazards for a class of chemicals used by a major industrial concern
- Critical review of toxicological data and structure-activity analysis for a class of chemicals manufactured by members of a major trade association
- Assessment of potential human exposure and risks resulting from the presence of a toxic contaminant in a manufactured product
- Assessment of potential human exposure and risks likely to result from use of a registered pesticide on a new crop