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DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE

STATEMENT

BY

JOAN Z. BERNSTEIN

Given by Chas. Monnett

GENERAL COUNSEL

DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE

BEFORE THE

COMMITTEE ON VETERANS' AFFAIRS

UNITED STATES SENATE

THURSDAY, FEBRUARY 21, 1980

Mr. Chairman and Members of the Committee:

I am Joan Z. Bernstein, General Counsel of Health, Education, and Welfare and Chair of the Interagency Work Group to Study the Possible Long-Term Health Effects of Phenoxy Herbicides and Contaminants. I appreciate this opportunity to appear before the Committee in my dual capacity to report on the Federal Government's current and planned efforts to study the possible long-term adverse health effects on humans of exposure to these chemical compounds.

Because of the Committee's special concern about health problems experienced by Vietnam veterans, I will review the status of HEW and work group efforts to study the effects on humans of phenoxy herbicides and dioxins, and will focus particularly on our examination of the phenoxy herbicide known as Agent Orange.

With me today are several members of the HEW scientific community who are very much involved in this effort. They are Dr. John Moore, Deputy Director of the National Toxicology Program; Dr. David Rall, Director of the National Institute of Environmental Health Sciences (NIEHS); Dr. John Froines, Acting Deputy Director of the National Institute for Occupational Safety and Health (NIOSH); and Dr. Patricia Honchar, Chief of the Dioxin Study and Registry at NIOSH. Dr. Froines is representing Dr. Anthony Robbins, the Director

of NIOSH. Dr. Moore is the Director of the Scientific Panel of the interagency work group and is being assisted in that endeavor by Drs. Rall and Robbins.

The subject under discussion today is surrounded by controversy and emotion. There is much that is already known about the effects of human exposure to phenoxy herbicides and dioxins, but much that remains in doubt. Accordingly, I believe that we at the Federal level must recognize and fulfill our responsibility to the American people for a thorough, objective, scientifically impeccable, and timely examination of this subject. We must complete such an examination and accounting for the Vietnam veterans, their families, and their offspring because we owe them nothing less. We must complete it, also, because we as a society must face the full impact on our physical environment of the chemicals we use. In the most literal sense, our claim to a healthful environment demands such action.

I believe the Chairman and Members of this Committee share my view. In this regard, I was gratified to read the Chairman's recent remarks on the Senate floor (as reported in the January 24, 1980, Congressional Record) concerning the need to avoid emotionalism and alarm, or the creation of false expectations, in connection with the Agent Orange studies. Secretary Harris, my colleagues from HEW and other agencies here today, and I all share your firm commitment to a full examination and a complete and accurate accounting

of the truth on this subject. We make this pledge both for the Vietnam veterans and others who have been working so hard to bring this matter to the country's attention, and for the public at large.

As most of you know, for many years chemical herbicides have been used widely throughout this country and the rest of the world for a variety of farming, forest management, and similar purposes. An important group are the phenoxy acid herbicides. Two of these, 2,4-D and 2,4,5-T, constitute Agent Orange, a herbicide that was widely used for forest defoliation and destruction of crops during the Vietnam conflict.

The chemical reactions that produce 2,4,5-T unavoidably contaminate it with trace amounts of a chemical referred to as TCDD (2,3,7,8-tetrachlorodibenzo-p-dioxin), which has been shown in laboratory studies to be one of the most toxic chemicals known. Although TCDD is but one of a family of dioxins, much of the concern as to the alleged health effects of Agent Orange and other dioxins has centered on this contaminant.

In addition to its use in Agent Orange, 2,4,5-T has been extensively applied in the United States. The Environmental Protection Agency temporarily banned major uses of 2,4,5-T in 1979 because of concern as to toxic human effects.

Hearings on whether permanently to ban 2,4,5-T are now in in progress. Herbicides using 2,4-D are still in wide current use.

The Department of Health, Education, and Welfare and a number of other governmental and private entities and individuals, here and abroad, have been concerned for some years about the potential long-term health effects of exposure to phenoxy acid herbicides and dioxin contaminants. Indeed, HEW has actively conducted or sponsored more than 50 studies relating to phenoxy acid herbicides, TCDD, and other dioxins for more than ten years. The results of this research represent much of our collective current medical and scientific knowledge on this subject.

In January, 1978, concern about the long-term health hazards of TCDD and other dioxins led to the Department's co-sponsoring, with the International Agency for Research on Cancer of the World Health Organization (WHO), the development of a report that assessed available knowledge on the effects of dioxins and future needs for information. Much of the current research in this field is designed to address the major recommendations developed at that meeting. Further, the Department established a group in the summer of 1979 to coordinate its research activities germane to the Agent Orange and dioxin issues.

From a government-wide perspective, during the past two years, the Administration has given increasing attention to the potential adverse human health effects resulting from exposure to the phenoxy herbicides and dioxins. Various Federal agencies have been involved in the collection of scientific information, the review and evaluation of existing animal and human exposure data on the toxicity of dioxins (especially TCDD), and the support of related research.

The Administration is supporting studies to be conducted by the Department of Defense, by the Veterans Administration, by the Center for Disease Control and the National Institutes of Health, both within HEW, and by other Federal agencies. In addition, members of the Domestic Policy Staff and the Office of Science and Technology Policy of the White House have reinforced the efforts of various agencies to conduct well-designed, valid, objective, and peer-reviewed laboratory and epidemiological studies concerning the potential toxic and adverse health effects of dioxins.

The Air Force has made a commitment to conduct a study of possible health effects in Air Force personnel who were involved in aerial herbicide missions in Vietnam (the RANCH HAND study). This commitment has led to the development of a protocol which has incorporated the recommendations of outside expert peer review groups. This revised protocol has been transmitted to a Committee of the Assembly of Life

Sciences of the National Academy of Sciences for their review. This study, to be elaborated on and discussed further by the Air Force, is one of several epidemiological studies which are being planned, currently in progress, or nearing completion.

On December 11, 1979, the President's Assistant for Domestic Affairs and Policy, Stuart Eizenstat, asked the Secretaries of Defense and Health, Education, and Welfare, and the Administrator of Veterans Affairs, to establish an interagency work group to facilitate, coordinate, and monitor agency studies of the possible long-term health effects of phenoxy herbicides and their contaminants. This work group, chaired by HEW, is charged with assuring that the protocols and methodology of current and proposed federally funded research and studies are scientifically sound. This interagency group also will ensure that all relevant research findings, whether publicly or privately financed, are promptly made available to the public and the Congress, in a comprehensible and comprehensive manner.

Although the formal work group held its first meeting on February 1, 1980, the real interagency effort began two years ago. Thus, the work group represents the formalization of a number of informal working relationships among the various agencies involved in dioxin studies rather than the starting point of such efforts.

This same concern about phenoxy herbicides and dioxins is clearly shared by the Congress and has resulted in the passage of legislation to spur adequate research and to assure its quality and objectivity. As you know, one of these bills, S. 2096, was disapproved by the President. It was the President's conviction that one provision of the bill encroached on functions vested by the Constitution in the Executive Branch and that the activities it required were already under way.

No doubt the members of this Committee and I could spend several interesting hours in debate over the separation of powers issues presented by the disapproval. However, rather than engage in such a dialogue, I would rather focus on the salient point of the veto message: the President's strong support of the effort to investigate the health effects of dioxin exposure and his commitment to continue and complete that investigation.

With that in mind, I'd like to discuss where we are and where I believe we are going in this investigation. HEW's own research over the past decade has encompassed a combination of laboratory investigations and studies of people who have been exposed to TCDD or phenoxy acid herbicides in their occupational environment or by accidental exposures.

Research with animals has indicated that TCDD, a dioxin contaminant in Agent Orange, is one of the most toxic agents known. These animal studies have already established that TCDD can cause cancer, birth defects and fetal toxicity when pregnant female animals are exposed, and can also cause depressions of the immunological systems and increased susceptibility to infectious agents.

Animal toxicity tests have served us well in reliably predicting toxic effects in man. Thus, the animal studies which show TCDD to be highly toxic are extremely important. Epidemiologic studies will help to define the full nature and expression of the toxicity of TCDD and other dioxin contaminants in man.

It is widely accepted, though obviously unfortunate, that occupational groups often are instructive populations in which to explore questions about the effect of a particular chemical or substance upon human health. Workplace exposures to particular materials are often well documented, and records are frequently available describing the work histories of industrial populations. Documented incidents of heavy exposure to dioxin due to industrial accidents have produced some information about its immediate effects in humans, but less is known about its long-term effects. In this setting, NIOSH has initiated an epidemiologic study designed to examine long-term effects of human exposure to TCDD.

NIOSH is assembling a registry of all workers in the United States who have been involved in making 2,4,5-T, one of the components of Agent Orange which is contaminated with TCDD. This study is designed to monitor the health of workers who have been exposed to dioxins. Because 2,4,5-T has been synthesized in this country since the 1940s by a number of industries, there may be a large enough group of workers who have been exposed to dioxin for a long enough period of time, to answer questions about the long-term effects of dioxins on humans. The study should assist in answering key questions about dioxins posed by Vietnam veterans and others.

Assembling the registry and determining how well it will answer questions or confirm animal toxicity results will take time. The first step, already completed, has been to ascertain which U.S. industries have ever made 2,4,5-T. Through confirmation of lists of suppliers and registrants of 2,4,5-T provided by the Air Force and the Environmental Protection Agency, a final list of the industries which have synthesized this material has been compiled. Contacting each industry to explain the NIOSH study and the information needed from them is under way.

Also in progress is the collection of worker records and other information from the industrial users. To determine precisely how long ago and for how long workers have been

exposed to 2,4,5-T, NIOSH must gather the work histories of the people involved. Together with detailed information about the exact process used to manufacture 2,4,5-T, this approach will allow the best determination of exposures which the workers have received. Additionally, any medical records which employers have maintained for their workers may provide more clues about the effects of exposure.

A critical step in this study will be tracing the health of workers exposed to 2,4,5-T. To do this, demographic information such as name, Social Security number, and last-known address for each individual must be obtained from the industry. Through Social Security records, a determination can be made of the vital status of each 2,4,5-T worker. For those no longer living, the cause of death will be determined through State death certificates.

Ascertaining vital statistics and cause of death may require some time past the point when all records are accumulated from the industries. The final data analysis then will aim at determining, by total time of exposure, whether the mortality experience of these 2,4,5-T workers differs significantly in any way from that of the general population.

Because the records of 2,4,5-T workers are currently being collected, it is still not possible to say with certainty just how definitive results from the NIOSH registry will be. The ultimate value of the registry in answering

questions about health effects will depend on the number of workers registered, the adequacy of the records obtained from the industries, and the success of tracing these workers historically.

All of these activities are time consuming, but HEW believes that the NIOSH dioxin registry is a pursuit which holds promise for providing reliable information about the effects of exposure to dioxins on the workers who have been involved in the manufacture of 2,4,5-T, and on other groups such as Vietnam veterans exposed to Agent Orange. At a minimum, the registry should make possible an objective evaluation of morbidity and mortality patterns, including cancer incidence.

Another current occupational study involves a health examination of workers at a Nitro, West Virginia, plant that has been involved in the production of 2,4,5-T since the 1940s. Heavy exposure of some of these workers to TCDD occurred in 1949 from an industrial accident. Other studies involving workers exposed to 2,4,5-T and TCDD are under way in Arkansas and New York. Additionally, studies of workers exposed to other dioxins are under way in Illinois and Kentucky. Taken together, these studies represent one part of an overall effort to gather the data most relevant to the specific concern that Agent Orange exposure may have caused long-term adverse health effects in Vietnam veterans.

Another part of the scientific effort that is directly relevant to the veterans' concerns is the group of studies being conducted to ascertain whether TCDD, 2,4-D or 2,4,5-T produce genetic damage or induce alterations in males that may result in their fathering malformed offspring. This is especially important because research is clearly establishing that other members of the dioxin family of chemicals can produce toxic manifestations that are indistinguishable from those produced by TCDD. Studies of some occupationally exposed populations are consistent with these laboratory findings. Thus, what is learned about one dioxin is extremely important in adding to our knowledge about them all.

Animal toxicity studies have predicted and occupational studies have confirmed that skin lesions (chloracne) in humans are associated with TCDD exposure. There is also evidence of other toxic effects in humans, including: liver effects as indicated by enlargement and abnormalities in clinical tests of liver function; alterations in lipid (fat) metabolism; and, more recently, a modest decrease in the ability of peripheral nerves to transmit impulses.

Despite the great amount of insight that we already have, important gaps in our knowledge still exist. The symptoms that are known to be associated with dioxins or phenoxy acids often have not been shown to represent a unique disease pattern. Therefore, studies to determine

whether there is a relationship between these chemicals and a specific disease pattern in veterans exposed to them are imperative.

The interagency work group has appropriately begun by focusing on scientific information that is already available or under development about health effects in order to establish an action agenda for getting done that which remains undone. We must, however, recognize some of the problems involved in this scientific effort.

Despite all the current and contemplated research, it may be that although Agent Orange is the cause of some disease, the disease is also attributable to other agents. If so, the most that a study can tell us is that exposure to the chemical increases the disease's frequency. This limitation is especially acute in studying the effects of Agent Orange on the health of American troops in Vietnam. The time and concentration of their exposure is not known. Also, it is already known that the more serious illnesses claimed to be caused by phenoxy herbicides and dioxins can be caused by a variety of agents.

In the face of these problems, the work group has decided to set the following priorities for the gathering of information:

° First, to attempt to correlate the incidence of illness and disease among Vietnam veterans with their exposure in Vietnam to Agent Orange, in part by determining, insofar as practical, if Vietnam veterans as a class are as healthy as other relevant population groups.

° Second, to study the broader implications for public health in the United States and elsewhere raised by the continued use of substances containing dioxins.

The mission of the work group is essentially scientific. It may discover that members of the Armed Forces who served in Vietnam run a greater risk than other groups of contracting serious diseases. But it may also find that the origin of any such diseases is not peculiar to a given chemical or to the Vietnam experience.

° If these are the findings, they will not tell us at what elevation of risk a veteran's illness should be deemed service-connected, or if the United States should assume responsibility for compensating the Vietnam veteran or his survivors for illness should the increased risk be very small.

° They will not assist us in adjusting the equities between those Vietnam veterans and non-Vietnam veterans who contract similar ailments, or between veterans and other members of the public.

° Finally, they will give only tenuous guidance on the role that government should play in ameliorating the adverse consequences of dioxins to the health of the public at large.

I do not raise these difficult questions in order to answer them. I raise them because I am concerned that the intense public discussion to date about the design, objectivity and timeliness of research on this subject may be creating or contributing to an erroneous impression. Because of the controversy, many may have come to believe that once an optimal research agenda is established and carried out, the research results will provide definitive, incontrovertible scientific information about the health effects of phenoxy herbicides and their contaminants.

I believe this is an unfortunate view because even the best effort of which our scientists are capable may not produce such conclusive results. In short, we may be left, after the research is done, with many of the same social policy issues we face today. Nevertheless, we believe the research being carried out or planned is important and valuable. We hope it will help all of us formulate a fair and humane social policy. But it will not and cannot by itself answer questions that seem to us to be fundamentally ones of broad social policy that both the Administration and the Congress must soon confront.

The timetable for a definitive report by the work group and the development and review of its scientific findings will be established within the relatively near future. In the coming months, as the work group holds additional meetings, we will keep this Committee apprised of current or planned research. We will also try to keep you and the public fully informed on our progress at each stage along the way.

In that regard, I have attached to this statement, and ask that it be considered a part of my testimony, a copy of the work group's first report to Stuart Eizenstat. The report provides additional details on a number of points I have discussed briefly and explores many additional and related features of the overall effort. We will be happy to answer any questions the Committee may have. Thank you.

Statement of U.S. Environmental Protection Agency
Before the Committee on Veterans' Affairs
United States Senate
Concerning Agent Orange
February 21, 1980

We appreciate the interest of your Committee in learning about current activities of Federal agencies as they relate to concerns of veterans or members of the U.S. Armed Forces who may have been exposed to Agent Orange and who believe that they may have been injured by their exposure. As Members of the Committee know, Agent Orange was used during the Viet Nam conflict by the military. Although its two active ingredients, 2,4-D and 2,4,5-T, are also contained in herbicide products approved for certain uses in this country, Agent Orange itself was not required to be evaluated or approved under domestic pesticide regulatory law.

Before discussing the rather complex regulatory history of 2,4,5-T, and EPA's actions early last year to remove major uses from the market, we would like to give those Members who may not be familiar with our pesticide responsibilities some background information so that our actions can be evaluated in the context of our legal mandate.

The Environmental Protection Agency conducts a comprehensive regulatory program for pesticides, including herbicides, under authority of the Federal Insecticide, Fungicide, and Rodenticide Act, as amended. The objective of FIFRA is to ensure that pesticides will not "cause unreasonable adverse effects on the environment," which the Act defines as "any unreasonable effects on man or the environment, taking into account the economic, social and environmental costs and benefits of using any pesticide." To further this objective Congress has placed a number of regulatory tools at EPA's disposal.

First, FIFRA is a licensing law. Pesticides may enter commerce only after they are approved or "registered" following an evaluation against statutory risk/benefit standards. As I will explain in more detail later, the Administrator may take action to terminate any approval whenever it appears to him, on the basis of new information, or a reevaluation of information, that the pesticide no longer meets the statutory standard. These decisions are made on a use-by-use basis, since the risks and benefits of a pesticide vary considerably from one use to another.

FIFRA is also a use control law. Special precautions and instructions may be imposed such as requirements that applicators wear protective clothing, or restriction of use to trained and certified applicators which can mitigate risks and at the same time permit use and the attainment of benefits. These instructions, warnings and prohibitions are incorporated into product labeling, which may not be altered or removed. Comprehensive amendments to FIFRA enacted in 1972 made the use of a pesticide "inconsistent with" its approved labeling a crime, thereby providing some measure of assurance that uses are limited to those which have been evaluated and found not to pose unreasonable risks when all prohibitions, restrictions and precautions are observed. Penalties for pesticide misuse are substantially higher for persons who apply pesticides for hire than for private citizens or farmers.

FIFRA embodies the philosophy that those who would benefit by government approval of a pesticide product should bear the burden of proof that their product will not pose unreasonable risks.

This allocation of burden of proof applies both when initial marketing approval is sought and in any proceeding initiated by the Administrator to interrupt or terminate registration (suspend or cancel). Licensing decisions are usually based on tests furnished by an applicant for registration, which must be performed in accordance with testing guidelines prescribed by EPA. Current requirements for testing of pesticides for which major uses are proposed can be satisfied only through the expenditure of several millions of dollars and up to four years of laboratory and field testing.

Pesticide registration test standards have not, however, always been as rigorous as they are today. Advances in testing methodology, and heightened awareness of the potential chronic health effects of long-term low-level exposure to chemicals which have come only within the past decade, have brought about major changes. Therefore, many products that are on the market today were subjected to risk evaluations at the time of first approval, which are plainly inadequate by contemporary standards. Congress directed in 1972 that EPA should reevaluate its licensing decisions, and those of its predecessor in pesticide regulation, the U.S. Department of Agriculture, through a process called reregistration. At the same time, FIFRA provides that manufacturers must be given time sufficient to conduct tests to satisfy any new requirements.

We hope this lengthy discussion is useful to the Subcommittee. It is especially important for the Subcommittee to understand that the fact that the government has once approved a pesticide for domestic use does not mean that EPA can be confident today that its use can continue without unreasonable adverse effects. Moreover, the basis for pesticide approval

has for many years been risk/benefit balancing, and registration therefore should not be confused with a finding by the government that absolute safety is assured.

The toxicity of 2,4,5-T and its TCDD contaminant became a focus of regulatory concern even before EPA assumed responsibility for pesticide regulation in December 1970. Investigations of allegations that the military uses of Agent Orange could have severe deleterious human health effects prompted the U.S. Department of Agriculture to suspend uses of 2,4,5-T in waterbodies, on food crops, and around the home in April and May 1970. Of these suspensions only one, use on rice, was contested by the manufacturers of the herbicide.

All registrants were advised of these actions and two of the 2,4,5-T registrants, Dow Chemical and Hercules, exercised their right under the version of FIFRA then in effect to petition for referral of the cancellation of the rice use to an Advisory Committee. A nine-member Advisory Committee of scientists was then appointed to consider all relevant facts, submit a report and recommendations regarding registration of certain uses of 2,4,5-T and state the reasons or bases for these recommendations. Their report was submitted to the Administrator of EPA on May 7, 1971.

The Committee recommended that use of 2,4,5-T be permitted in forestry, range land, and rights-of-way, providing that a limit of 0.1 ppm of contamination with TCDD be set for all future production of 2,4,5-T; that 2,4,5-T be applied not more than once a year at any one site; and that 2,4,5-T be applied with proper caution so that it will not contaminate other areas where it may come into contact with humans. The Committee

also recommended that this action be reviewed again when existing deficiencies in information about possible magnification of TCDD in the food chain were rectified by specific research.

In July 1972 the Dow Chemical Company, a major producer of 2,4,5-T, obtained an injunction against further cancellation hearings, which was later overturned by the U.S. Circuit Court of Appeals for the Eighth Circuit. On July 20, 1973, EPA issued a notice of intent to hold a hearing to determine whether to cancel the remaining uses of 2,4,5-T under the 1972 revisions to the FIFRA cancellation proceedings. However, on June 24, 1974 EPA withdrew from the proceedings in order to obtain better TCDD monitoring data.

The state of our knowledge of 2,4,5-T was more limited in the sixties and early seventies than it is today. Indeed, it was more limited than the information available to EPA on other pesticides which were candidates for regulation. The lack of a detection methodology precise enough to find TCDD in environmental samples, human tissues, or market basket surveys at levels we now know to be present raised the question of whether exposure could occur at all. Secondly, the use of animal data to predict effects in humans was not so well accepted as it is today.

Regulatory agencies with responsibility to protect public health rely on carefully controlled animal experiments of varying duration and design to estimate risks of chronic hazards and acute effects. Of course, ethical considerations, as well as the practical impossibilty of isolating an experimental population from all potentially harmful substances during an investigation which may require many years, do not permit human experiments for chronic effects. While confirmatory epidemiological data is useful in reaching regulatory decisions the expense and time associated

with gathering epidemiological data limit its usefulness. Further, the many difficulties in investigative design, data collection, and data validity which are commonly encountered in epidemiology create a strong bias in the direction of false negative results. These false negatives, in turn, limit the value of such studies for regulatory decisionmaking. Most health and safety regulatory laws proceed from the philosophy that potential harm which can be averted without unreasonable economic consequences should be averted, even if it is not certain that harm will otherwise occur.

Regulators and academics are not the only scientists who recognize the value of properly designed animal experimentation. Manufacturers routinely conduct long-term animal feeding studies in order to demonstrate that their products do not cause chronic effects. While use of animal testing is born out of practical necessity, such tests have been shown to have reliable predictive value (virtually all known human carcinogens are also carcinogens in test animals.)

One of the principle reasons for EPA's decision to terminate the 2,4,5-T cancellation proceeding in 1974 was our concern about the absence of exposure data to combine with the well established evidence of extreme teratogenic, fetotoxic, and carcinogenic toxicity of 2,4,5-T or TCDD.

In July 1975 EPA promulgated new procedures designed to make easier our work in reaching conclusions on pesticides which had been identified as being "suspect" of causing serious adverse effects. We felt that the new approach, described as "Rebuttable Presumption Against Registration", or RPAR, would complement the statutory mechanisms for pesticide review which, because of their adjudicatory nature, tend to make it difficult for some interested parties to participate. Also, RPAR was expected to

offer advantages in collecting additional toxicity or benefits data needed to reach sound public policy decisions, where there were obvious deficiencies in the existing data base in spite of years of official and unofficial concern about possible health effects.

On April 27, 1978 EPA issued a Notice of Rebuttable Presumption for 2,4,5-T, and a related dioxin-contaminated herbicide, Silvex. This document summarized the extensive toxicity testing which had been undertaken for these chemicals and TCDD by manufacturers, academic researchers, and the government. We encouraged the public to supplement this information with further scientific evidence concerning risks, and with economic analyses of the impact of cancellation for the various uses of the herbicides. We received thousands of submissions. Among these was a carefully presented account of what appeared to a member of the lay public who contacted us to be an unusual incidence of miscarriage in an area of Oregon where forest use of 2,4,5-t and Silvex is an annual occurrence. After interviews with the women who had experienced the miscarriages, EPA decided that our epidemiologists should investigate records of hospitalization for miscarriage. In the first weeks of 1979 EPA found a statistically significant increase in miscarriage frequency in areas of 2,4,5-T use in forestry which correlated in time with spray operations. It is important to note here that we did not claim that the study proved a cause and effect relationship between miscarriage and the spraying. Rather, we concluded that the correlation which existed was consistent with what one would expect based upon the available animal data and if exposure was occurring; and that the study therefore suggested evidence of the previously undiscovered human exposure link. This evidence became available literally on the eve of the large scale spring herbicide

treatments that are conducted annually in commercial forestry.

On February 28, 1979, EPA took emergency action to halt forest spray operations and other major uses of 2,4,5-T and silvex. The emergency action withstood almost immediate challenge in the U.S. District Court for the Eastern District of Michigan. Following the Court's ruling, the Dow Chemical Company and other registrants withdrew from EPA's administrative suspension hearing. This hearing opportunity is accorded to registrants by the statute as an expedited mechanism through which to present evidence as a basis for modifying the suspension order.

Suspension under FIFRA is analagous to a temporary restraining order. It is based on a finding that the risks of continued use during the period required to complete a cancellation hearing outweigh the benefits that would be foregone during that period (historically, 1-3 years). The cancellation hearing is the mechanism by which evidence is adduced and tested concerning the totality of risks and benefits resulting from use of the pesticide over its life. The consolidated hearings on whether all uses of 2,4,5-T and Silvex, a related herbicide, should be finally cancelled are expected to begin next month. Attached to this statement are the suspension and cancellation notices issued by EPA, as well as the Agency's pretrial brief on the risks of 2,4,5-T and Silvex which was recently filed with the Administrative law judge.

Before closing we should mention that information on the risks of 2,4-D, the other constituent of Agent Orange, is undergoing an intensive evaluation to determine the significance of studies of its reprodutive and inheritable (mutagenic) effects. We recognize that 2,4-D use may increase since 2,4,5-T is unavailable for many of its former uses and for that reason an early decision on whether the risks of 2,4-D

warrants issuance of an RPAR notice or some other regulatory action is desirable. Although theoretical chemists believe that one dioxin isomer (2,7dichlorodioxin) could be formed during the manufacture of 2,4-D, no dioxins have been found during years of study.

We hope that this account of EPA's regulatory actions under FIFRA will compliment the extensive testimony you have received from other agencies who are investigating exposure to phenoxy herbicides with a view toward developing appropriate public policy where that exposure may have occured due to military service. EPA is an observer to the inter-agency work group established last December, and in that capacity will share with the work group information which we develop or which comes to our attention in the conduct of our duties under FIFRA which may be of value in its efforts.

Thank you.

April 29, 1980

2,4-D FACT SHEET

I. Background

2,4-D is one of the most widely used herbicides in the United States. There are approximately 1,500 products containing 2,4-D registered with EPA, and more than 70 million pounds of the active ingredient are distributed annually. The term "2,4-D" refers to the phenoxy herbicide 2,4-dichlorophenoxy acetic acid and its 35 derivative salt and ester forms. 2,4-D is used to control broadleaf weeds in a variety of places including home lawns, cereal and grain crops, commercial areas, commercial turf, rights-of-way, and forests.

Public concern about the potential adverse health effects of 2,4-D has intensified since the emergency suspension of 2,4,5-T and Silvex in March 1979. This concern stems primarily from 1) the chemical similarity of 2,4-D and 2,4,5-T as phenoxy herbicides, and 2) the question of 2,4-D dioxin-contamination, especially contamination with tetrachlorodioxin, a manufacturing contaminant in 2,4,5-T, which causes cancer and miscarriages. Due to the chemical similarity of 2,4-D and 2,4,5-T, the public has expressed concern about the potential for cancer and miscarriages from the use of 2,4-D. There is also concern because the controversial military defoliant Agent Orange, used in Viet Nam, was composed of 2,4,5-T and 2,4-D. Agent Orange was never registered by EPA for civilian use in the United States. Its use in Viet Nam by the U.S. military has resulted in claims of adverse health effects to American military personnel. The Veterans Administration is studying these claims.

Prompted by these concerns and EPA's need to resolve the questions surrounding the use of 2,4-D, the Agency initiated a review of the available information on the potential health effects of 2,4-D. This review was conducted in part to determine if the herbicide should be reviewed under the RPAR process (Rebuttable Presumption Against Registration) or if another regulatory action was appropriate.

II. Agency Review and Conclusions

Based on the results of this review, EPA has concluded that a) the presently available information on the potential adverse health effects of 2,4-D does not support a regulatory action to remove 2,4-D products from the market; b) information from scientifically valid studies does not indicate that the continued use of 2,4-D poses an imminent hazard or unreasonable adverse effect when used according to label precautions and direction for use; and c) the Agency should act quickly and vigorously to obtain better toxicological information on 2,4-D.

These conclusions are based on these following considerations:

1. There is no evidence available at this time that indicates 2,4-D contains any form of dioxin. This includes the tetrachloro-dioxin (TCDD), which is a manufacturing contaminant of 2,4,5-T and causes cancer and miscarriages.

TCDD is not theoretically expected to be found in 2,4-D. The manufacturing processes and starting chemicals from which 2,4-D and 2,4,5-T are made are not the same. Although other much less toxic dioxins are theoretically possible in 2,4-D, they have not been found despite thorough chemical analyses.

2. Because products containing 2,4-D have been registered for use since the 1940's, most of the scientific data submitted to support the product registrations now on the market were developed many years ago. While some of these studies are scientifically valid, many others do not meet today's standards for scientific testing. As a result, there are significant information gaps in several areas including cancer-potential, reproductive effects, neurotoxicity, and metabolism in animals.

3. The studies most pertinent to the question of tumor-causing potential (oncogenicity) of 2,4-D were considered inadequate and inconclusive. No valid conclusions could be drawn one way or another from the data.

4. Animal tests conducted on the potential reproductive effects of 2,4-D show that, unlike 2,4,5-T with its contaminant TCDD, severe life-threatening effects were generally absent from 2,4-D treatments at moderate or high doses. However, new tests will need to be conducted at lower doses to clearly establish no-effects levels. In comparison, TCDD, which is present in 2,4,5-T and not in 2,4-D, produces serious life-threatening effects on the fetus at minute doses including the lowest dose tested in many studies.

5. The scientific evidence available at this time does not indicate the potential human exposure is sufficient to result in human health effects.

6. The most vigorous authority available to EPA under the pesticide law to fill information needs is a new section of FIFRA (Federal Insecticide Fungicide Rodenticide Act) passed in 1978. This provision, known as 3(c)(2)(B), allows EPA to request any additional data from pesticide registrants that is considered necessary to maintain the registration of existing products. The Agency can immediately require the

manufacturers to develop the data where gaps exist. The registrants have 90 days to show that they are complying. Their product registrations may be summarily suspended if they fail to meet the Agency's conditions. No other action could obtain this information any faster. EPA is putting the data requirements into final form and they will be issued to the registrants after review by our Scientific Advisory Panel. These scientific experts will review and comment on the data requirements to assure that they will provide the information EPA needs to more definitively answer the questions on potential health effects of 2,4-D.

7. Based on a review of the toxicology data (see section IV below), and a review of the risks of other pesticide chemicals now undergoing regulatory action, the Agency believes that the risks of several other pesticides are higher and better documented than those associated with 2,4-D. To put the review of these other higher priority chemicals aside in order to devote EPA resources to taking action against 2,4-D would not, in the Agency's opinion, best serve the public interest.

III. Additional Actions

In addition to requiring several important studies of the manufacturers on 2,4-D, EPA will also:

1. Conduct several tests on reproductive effects (through our Office of Research and Development) of several derivatives of 2,4-D in order to quickly get new information and have a good basis for comparison with the company-produced data.

2. Continue its ongoing review of forest pest control practices. This review will evaluate all chemical and non-chemical controls to identify the most environmentally protective ways to control forest pests. The Agency believes that a piecemeal approach to forest chemical regulation only leads to confusion, both to the industry and to the public. Unless we review the whole range of possible controls, examining one chemical at a time only gives rise to questions about the chemicals which would be used to replace those examined and prohibited from use.

3. Review all new data as it comes in to determine if a change in our regulatory posture is warranted. This includes evaluating the results of new animal tests as well as looking into reported incidents involving human exposure to the chemical.

4. Continue to support field tests to measure exposure to 2,4-D during the present growing season.

5. EPA is informing the Inter-Agency Work Group, established by the White House to study the possible long-term effects of Agent Orange, of the actions being taken. EPA will also share its scientific findings with this committee.

IV. Toxicology Background

The potential hazard of a chemical is usually measured in laboratory animal tests. Animals are given doses of a chemical over a specific time period. Scientists attempt to derive from most of these tests a "no observable effect level" (NOEL) -- the dose level below the dosage where effects are first observed. From the animal tests and NOEL's, the potential effects on humans and other animals can be estimated. A set of brief definitions is provided below to permit better understanding of the subsequent discussion of toxicological findings.

A. General terms

1. Acute oral toxicity (LD50) - this test determines the dose level which produces death in half the test animals after a single oral dose (short-term test). Used to predict the near-term toxicity of the chemical immediately upon contact with people or other non-target animals.

2. Chronic feeding tests - animals are fed for most their life span (usually greater than 18 months in rodents) in order to determine the dose level which shows no toxic effect in test animals. This is the test from which the NOEL is (usually) derived.

3. Oncogenicity testing - animals fed relatively large doses of the test chemical for their life span (usually 18 months to 2 years in rodents) to try to induce tumors. These tests are used to predict whether the chemical may pose a cancer hazard.

4. Reproductive testing - these tests evaluate the effects of the chemical on the fertility of both the male and female parents by exposing the animals for a period of time before breeding. The tests also measure the possible effects of the chemical on the pregnant female and the fetuses through several generations. (The test with rodents through 3 generations runs approximately 14 months.)

5. Teratology testing - these tests evaluate the effects of the chemical on fetuses by exposing pregnant females during the short period of time that the fetus is most susceptible to congenital malformation. Teratogenic effects include cleft palate, central nervous system deformities, eye and limb deformities, and internal organ malfunction. These are considered to be life-threatening effects that put the animal at a disadvantage for surviving in its environment.

6. Fetotoxicity - fetotoxic effects can be seen in either the reproduction or teratology tests. Toxicity may be seen in the extreme form as fetal death or as less severe problems, such as delayed formation of bones, reduced body weights at birth, or edema (abnormal fluid accumulation in the tissue). Most fetotoxic effects appear to be reversible once exposure to the test chemical is curtailed. Therefore most fetotoxic effects are considered to be less serious than teratogenic effects, with the exception of fetal death.

8. Summary of Toxicology Review

Most of the data in EPA files on the potential health effects of 2,4-D are centered on the acid form, even though there are many derivatives, such as salts and esters. This is because the many forms of 2,4-D metabolize to the acid form in the environment and in the body. The discussion of animal data below, therefore, concerns the acid form of 2,4-D unless otherwise noted.

1. Acute toxicity - low to moderate. The potential for immediate poisonings from contact with the chemical is unlikely.

2. Neurotoxicity - There is little definitive information on the possible neurological effects of 2,4-D. In several reported cases of impaired nerve function, it was not known if the individuals were peculiarly sensitive to that type of effect or were exposed to other toxic materials.

3. Reproductive effects (effects on the unborn) - Tests have been conducted on rats, mice and hamsters to evaluate the possible reproductive effects of 2,4-D. 2,4-D causes some of the less serious fetotoxic effects, such as edema (swelling of tissues) at the lower dose levels tested, and causes life-threatening birth defects (skeletal malformations) and cleft palates only at the very high levels tested.

Based on available animal studies, EPA estimates that the level of exposure in a "worst case" situation (eg. a person standing directly under a spray plane) would be 500 to 1000 times less than the dose level that might cause an effect.

Much of the data available to judge these effects was generated by old study protocols, has deficiencies in the test methods, and needs clarification by further study.

EPA also reviewed summaries of tests conducted in Russia which state that some derivatives of 2,4-D produced adverse effects on unborn animal fetuses at much lower levels than indicated by the data in EPA's files. These summaries could not be used in the Agency's review because the identity of the test material, and its impurities, was unclear, and because there were no numerical data to back up the summary conclusions. In some cases tests need to be done on specific derivatives of 2,4-D.

4. Oncogenicity (potential for causing tumors) - Several rodent studies have been conducted to date. The tests were conducted a decade ago and are considered to be inadequte and inconclusive by today's scientific standards. New studies on rodents are needed.

5. Mutagenicity (inheritable effects) - The vast majority of the mutagenicity studies conducted on 2,4-D are negative. However, there are three positive studies. Taken as a group, the results of the studies can best be described as inconsistent and inconclusive. A new series of tests being conducted by the Department of Health, Education, and Welfare will be reviewed by EPA when they are completed.

6. Epidemiology - No epidemiological studies of human health effects from 2,4-D exposure have been completed. However, EPA is currenlty investigating reports about alleged adverse effects from potential chemical exposure in several parts of the country. EPA will be looking at the results of those studies and will decide in the near future about additional field work.

V. Exposure to 2,4-D

There are at least three ways that the average citizens might come into contact with 2,4-D - through the diet, during home use, and drift of the herbicice from nearby use.

a) Diet

The EPA has set tolerances for residues of 2,4-D in various food crops. The Food and Drug Administration (FDA) routinely samples a variety of foods (the Market Basket Survey) which FDA considers to be representative of the average American diet. Samples are analyzed for pesticide residues. During the period of 1974 to 1977, no 2,4-D residues were found in any of the products in the Market Basket Survey. However, during the 1965 to 1977 period, a variety of other food products were analyzed under other surveys in which about 1.1% were positive for 2,4-D in very minute quantities that were well below EPA's tolerance (allowable residue) levels.

b) Home use

There are currently a number of registered home-use products which contain 2,4-D in a variety of formulations. Exposure to the herbicide in home-use situations will depend to some extent on the specific formulation used. If care is exercised by the homeowner in adhering to the directions for use and precautionary statement on the label, exposure to 2,4-D should be low.

c) Drift

"Drift", the airborne transport of pesticide materials to a non-target area, is a common source of exposure. Sometimes, a pesticide will drift during application, depending on climatic conditions (temperature, wind speed), type of formulation used, terrain (forests, mountains), and type of application method used (aerial, ground spray). Several States have imposed restrictions on 2,4-D use in order to cut down on drift potential.

Once on the ground or target crop, the herbicide may become airborne again by the process of vaporization. This particular type of drift has been the subject of intensive research by the producers of 2,4-D. Since the introduction of less volatile forms of the herbicide over the last few years, this kind of drift has become much less extensive.

VI. Environmental Persistence

2,4-D is not a persistent pesticide. Breakdown of the herbicide begins almost immediately after application at a rate dependent on several environmental factors such as temperature, humidity and medium (air, soil, crop, water). The rate of loss (commonly referred to as the half-life) is a measure of the time required for half of the substance to be degraded or lost.

On sprayed vegetables, the half-life varies from 1-3 weeks depending on geographic location, climatic conditions, vegetation type, application technique and formulation used.

In soil, the half-life varies from several days to 2 weeks, depending on acidity, soil type and amount of rain.

In water, the half-life varies from a few days to several months depending on factors such as oxygen concentration, acidity, light intesity, water temperature and formulation used.