REPORT OF THE DDT ADVISORY COMMITTEE*

TO

WILLIAM D. RUCKELSHAUS, ADMINISTRATOR
ENVIRONMENTAL PROTECTION AGENCY

September 9, 1971

*Established Under Provisions of Section 4.c. of the Federal Insecticide, Fungicide, and Rodenticide Act.

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DEPARTMENT OF PHARMACOLOGY AND TOXICOLOGY

September 9, 1971

Mr. William D. Ruckelshaus
Administrator
Environmental Protection Agency
Washington, D. C. 20460

Dear Mr. Ruckelshaus:

On behalf of the DDT Advisory Committee, I am pleased to submit the following report of our considerations of the scientific issues raised by DDT.

The Committtee hopes that the information in this report will be useful to you and your staff in evaluating the effects of this substance upon man and his environment.

If there is any additional information that either the committee or I may furnish you concerning the report or our considerations we will be pleased to supply them.

Sincerely yours,

James G. Hilton

James G. Hilton, Ph.D.
Chairman
DDT Advisory Committee

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Enc.

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INTRODUCTION

DDT (Dichloro-diphenyl-trichloroethane) was the subject of a decision announced by the United States Court of Appeals for the District of Columbia Circuit on January 7, 1971. This decision required the Environmental Protection Agency to take two steps: (1) to commence the administrative process for cancelling the registration of all products containing DDT; and (2) to consider whether the present information available to the Agency warranted the immediate suspension of the registration of all products containing this chemical. The Environmental Protection Agency responded to the first by issuing a cancellation notice, PR 71-1, for all products containing DDT on January 15, 1971. It responded to the second step requested by the court by issuing a position paper dated March 18, 1971, titled "REASONS UNDERLYING THE REGISTRATION DECISIONS CONCERNING PRODUCTS CONTAINING DDT, 2,4,5-T, ALDRIN AND DIELDRIN." In it, the Agency outlined its responsibilities under the Federal Insecticide, Fungicide, and Rodenticide Act and explained why suspension of DDT registrations was not considered necessary.

The administrative process for cancelling the remaining registrations for DDT began with the issuing of the cancellation notice of January 15, 1971. Objections to this notice were filed by a number of firms, most of which chose the option of requesting a public hearing. Montrose Chemical Corporation of California and the Crop King Company of Yakima, Washington, however, requested that the issues raised in the cancellation notice be referred to an advisory committee named from a list of nominees provided by the National Academy of Sciences in accordance with the Act. The DDT Advisory Committee was appointed by Mr. William D. Ruckelshaus, Administrator of the Agency, in a letter dated April 30, 1971.

Due to the nature of the factors involved in the issuance of the cancellation notice, the charge to the Committee was very broad. This charge stated in part: "The Committee is charged to consider all relevant scientific evidence concerning DDT, and to prepare a report and recommendations as to the scientific issues raised by the use of DDT." Based upon the breadth of the charge and the pressures of a fixed time deadline for its report, the Committee has elected to depend upon the two most comprehensive recent reports on DDT (Jensen Committee Report on Persistent Pesticides to Administrator, Agricultural Research Service, U.S. Department of Agriculture, May 27, 1969; and Mrak Commission Report of the Secretary's Commission on Pesticides and Their Relationship to Environmental Health, December, 1969) for the evaluation of much of the prior scientific information concerning this compound. This has allowed the Committee to concentrate its efforts upon obtaining and evaluating whatever new information has become available since these reports.

For the sake of convenience this report is divided into four major sections followed by the conclusions and recommendations of the Committee. In general, "DDT" is used to mean the combination of DDT and its metabolites. The first section deals with the current estimates of the quantities of DDT being used and the residues present in the various sections of the environment according to the most recent monitoring data available. The second section presents briefly a summary of the possible interferences with the analytical determination of DDT in environmental samples caused by the simultaneous presence of the polychlorinated biphenyl compounds. The third section presents the toxicity of DDT upon nontarget species with particular emphasis upon the toxicity to the mammalian species. The final section considers the present need for DDT.

USE AND RESIDUE ESTIMATES

U.S. Production History

The history of rapid expansion in the production of DDT and closely related compounds from 1944 to about 1960 and general decline since that date is well documented elsewhere and there is no need for detailed summary here.

Figure 1 presents the available data on production in the United States and usage in this country as estimated from the difference between total production and the stored and exported stocks reported for each year.

The rise was caused by growing recognition that DDT is an inexpensive, persistent, wide-spectrum insecticide. The declines have been attributed to developing resistance to DDT by many insect species, the introduction of effective replacement insecticide, and increasing concern about pesticides which are persistent and have a wide spectrum of effects on species.

U.S. use apparently declined from about 70,000,000 annually for 1956-1962 to about 30,000,000 pounds in 1968 and 1969. The Committee has received estimates for 1970 and 1971 which indicate continuing decline, but confirmed data are not yet available.

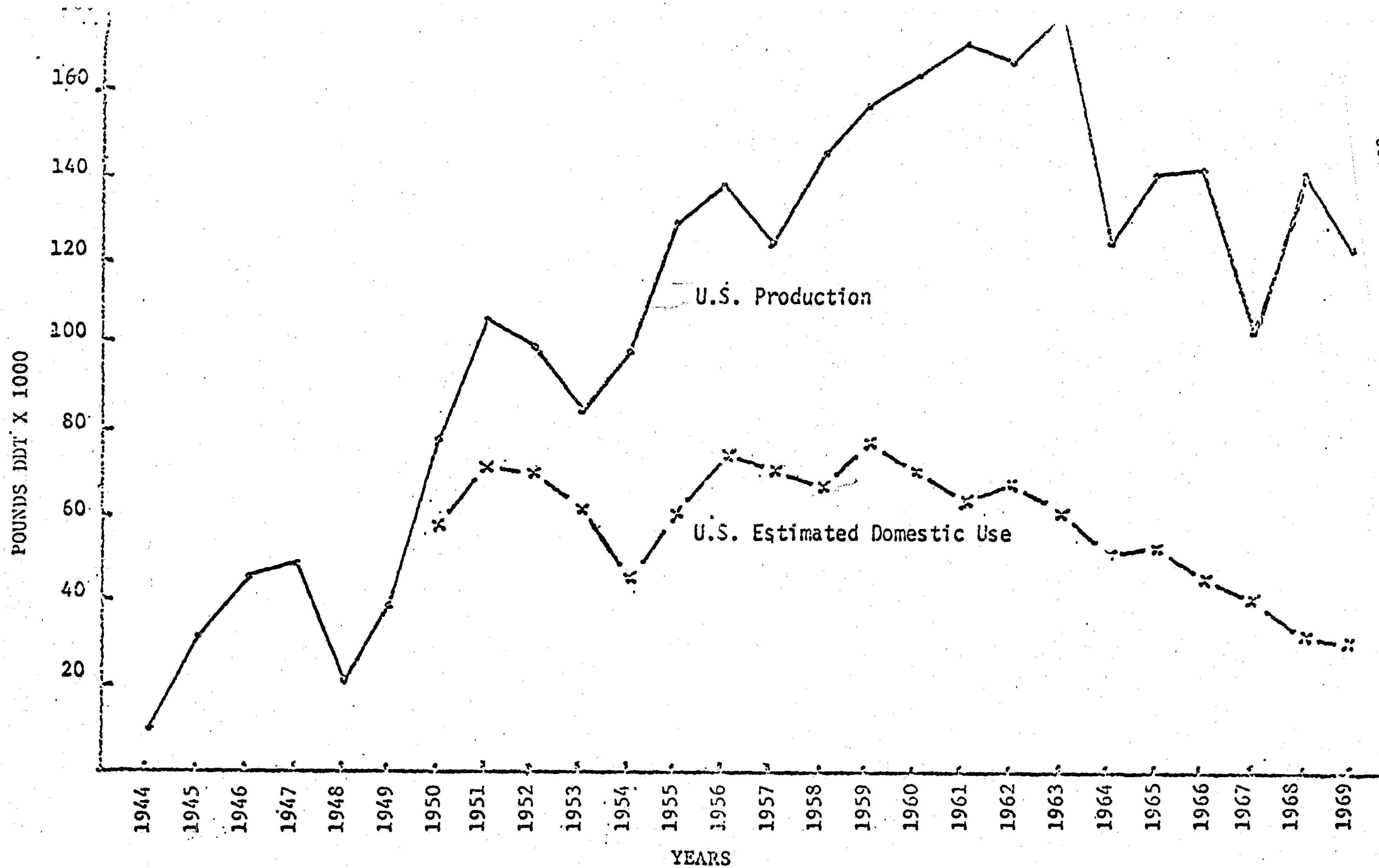

Figure 1. United States production and estimated usage of DDT. Data from Pesticides Review 1970; Stickel 1965; Goldberg, et al. 1971.

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Environmental and Biological Load

In the following sections, we have summarized the most recent data available on DDT in the environment, so as to provide the means of determining whether the continuing decline in use has produced a decline in the concentration of DDT in the environment-soil, air, water, natural biota, human food, and man himself.

The levels in soil. Data on the buildup of DDT and other persistent pesticides in the environment were sketchy and frequently misleading during the first fifteen years of widespread use (1946-1960.)

The President's Science Advisory Committee Report of 1965 described what was known up to that time of the buildup of residues of DDT in soils with high pesticide use history as might have been expected, and they attributed this result to the beginning of a trend toward the use of less persistent pesticides. A subcommittee on soil contamination concluded, however, that the level of contamination was increasing, and that the problem of soil pollution by chemicals had grown to the point where it was a matter of immediate national concern. That Committee recommended strongly that there should be a program of monitoring by which the buildup of persistent materials in the environment could be documented as a basis for future steps toward improvement in environmental quality.

More recent discussions of DDT levels in soils have considered the diverse mechanisms by which this chemical leaves the soil to which it is applied, and returns to the soil at another location through precipitation, dust fall, and runoff (Edward 1966, Nash and Woolsen 1967, Risebrough et al. 1968, and Goldberg 1971). Because of the difficulty in measuring losses to the atmosphere, and the effect of agricultural treatment and weather on these exhanges, the early estimates of decomposition rates in soils and the concept of a "half-life" for DDT in the environment are in doubt.

The present evidence available to the Advisory Committee was a summary of 1969-1970 results of monitoring by the Environmental Quality Branch, Pesticides Regulation Division, EPA. While these results were for one and two years ago, they nevertheless represented the levels in soils a full 10 years after the peak levels in use of DDT. The 1969 results show that average levels for croplands are highest in the Southwestern, Southern and Eastern Seaboard States. All of these states average above 0.10 ppm, but ranged as high as 0.56 ppm. Urban and orchard soil samples were highest, with average values between one and two ppm, and individual samples as high as 52 ppm of DDT and its metabolites. Of six states sampled randomly in both 1968 and 1969, four showed an increase in DDT residue and two decreased in 1969. Because sampling sites were changed from year to year, the Committee does not consider that these results demonstrate a trend.

<u>The levels in air</u>. Recent research in the release of pesticides into the air, movement in aerial transport systems, and persistence in air was reviewed by the Mrak Commission in 1969, and no significant additional information has come to the attention of the Committee. Air transport is an important component in creating the worldwide patterns of distribution, but it is abundantly evident that much more evidence should be sought on the distribution, movements and significance of pesticides in air. Aerial application involves opportunities for introduction of pesticides into the air mass

and for export from the sites of application. Codistillation and evaporation may also introduce significant quantities and affect global distribution. The persistence of DDT and other materials in air masses is also highly pertinent and insufficiently understood. These and other fields of research require continued attention for optimal management of DDT and many other chemical materials. The relatively large quantities of DDT present in the biosphere offer unique opportunities for understanding these movements and the underlying mechanisms involved.

The levels in water. The solubility of DDT in water is reported to be 0.0012 ppm (Bowman et al. 1960). This means that observations of DDT in water at greater than 1.2 ppb should be viewed as attributable to contamination or other mechanisms. Residues of DDT much higher than this have been reported frequently in water, and in almost every case the contamination has been traced to a local application. Usually it is associated with heavy rainfall that washes a portion of the DDT applied into adjacent streams or lakes. Because of the low solubility the high levels of DDT do not persist in water, instead the DDT moves into the atmosphere or is taken up by sediments, living organisms and other particulate matter (Harrison et al. 1970).

Other studies are giving clearer understanding of the fluctations of DDT in water systems. Because of low solubility, the DDT tends to reach the surface of the water from which it enters the atmosphere. Very little information has been available as to the levels of DDT in the atmosphere during the period of high use in the 1950's but in the mid-1960's in England rainwater samples averaged 0.08 ppb, and during

1968-69 in Florida precipitation averaged 1 ppb (Tarrant and Tatton 1968, Yates et al. 1970). These results appear to vary widely depending on season, dust levels in the atmosphere and other factors.

The most recent data available to the Advisory Committee were the results of the U. S. Geological Survey water quality monitoring program. The results of these surveys indicate that DDT or its metabolites are frequently found in rivers monitored by the USGS at levels approaching the solubility of DDT (1.2 ppb), apparently because of the association between DDT and the fine particles carried by streams. These materials are deposited on stream and lake bottoms during periods of low flow, but undergo considerable re-suspension during storms or periods of peak flow. Storms such as hurricanes appear to be particularly important in the re-suspension of DDT-contaminated sediments in the coastal water of the United States.

These processes results in great variability in the levels of DDT observed in unfiltered water samples. Much of the time there is no detectable contamination by DDT or its metabolites, but at other times significant contamination is observed. Because the contamination originates from treated agricultural lands and from stream and marine bottom sediments, it appears that no decrease in water contamination can be expected until the levels in soils and sediments decrease.

The levels in natural food chains. The level of DDT in the food chains of native species, both on land and in the water resource systems, has been documented by detailed analyses of selected native species over the past 25 years. Both State and Federal agencies have been involved, with a greatly increased program over the past five years. There are now a number of examples of important fish and game resources that have reached a level of contamination that prevents distribution in interstate commerce under Food and Drug Administration regulations.

The high level of contamination in these species has been shown to result primarily from the process of biological concentration in natural ecosystems. Woodwell (1970) and Harrison et al. (1970) have described the role of the trophic structure and mechanisms of an ecosystem in transporting and concentrating DDT. Storage of DDT in each trophic level, transport from one trophic level to another, and the transformation of DDT into DDE or DDD by metabolism are all involved. In contrast to its near insolubility in water, DDT is highly soluble in the lipids of organic materials.

The combination of high solubility in lipids and high chemical stability allows "magnification" of DDT concentrations from organisms at the base of a food web to a higher trophic level fed on those in the levels below, and a substance like DDT, which is stored in the lipids and breaks down slowly, can accumulate to a high concentration in the higher trophic levels.

The review by Goldberg et al. (1971) indicates that in the open ocean, phytoplankton form the base of the food chain and may act as primary concentrators of the chlorinated hydrocarbons in the water. There is some evidence demonstrating inhibition of photosynthesis in single-cell marine plants by DDT (Wurster 1968) but the important fact here may not be the direct effect on plankton, but rather that plants serve as one of the vehicles for transferring DDT from the water to higher trophic levels. Another major pathway is through the utilization of DDT-contaminated dead organic matter in the sediments of water systems by benthic organisms.

The Goldberg committee concluded from an analysis of chlorinated hydrocarbons that marine fish are almost universally contaminated with chlorinated hydrocarbon residues. They note the effect of this contamination on the marine populations themselves, citing the speckled sea trout on the south Texas coast, in which DDT residues in the ripe eggs were about 8 ppm. Residues of 5 ppm in freshwater trout cause 100 percent failure in the development of sac fry or young fish, and they conclude that it is significant that extraordinarily few juvenile fish have been observed in certain coastal areas in recent years.

The situation of freshwater ecosystems is probably best illustrated by the results of studies published by the Wisconsin Conservation Department (Kleinert <u>et al</u>. 1967, Poff and Degourse 1970). Surveys were initiated in 1965, and results are available through 1969. These authors conclude that the 1965 and 1966 surveys demonstrate a widespread and significant level of contamination in inland fisheries with DDT, and that residues in fishes from certain Wisconsin waters had already reached levels harmful to fish. The 1970 report by Poff and Degourse has more data for larger and older fish, which show more severe contamination. It is difficult to say that the higher levels reported by them are entirely due to a continuing buildup in DDT level, but it appears that concentrations in Lake Michigan Fish were about the same in 1969 as in 1965.

The economic effects by 1970 were worse because the Food and Drug Administration had set a tolerance of 5 ppm of DDT in fish products marketed for interstate shipment. A paper by Lueschow and Winter (1970) concludes that "The Food and Drug Administration limitation of 5 parts per million of DDT in fish which may be sold in interstate commerce has virtually eliminated the commercial market for Lake Michigan fish." They note that based on the 1966 catch estimates for the entire Great Lakes, approximately 42 percent of the commercial catch would be unacceptable for interstate commerce.

The environmental load of DDT in terrestrial ecosystems is most evident in certain species of birds. Insects, other soil invertebrates, and aquatic life serve as the food of these terrestrial bird species. The effects have been recorded through studies of museum eggshells, showing that thinning has occurred since the mid-1940's in a wide range of species. Where shell thinning has occurred, the populations have usually declined (Ratcliffe 1967, Hickey and Anderson 1968). The decline in populations of the peregrine falcon and bald eagle have both been linked with substantial evidence to the buildup in DDT or its metabolites in their tissues. In addition to many such observations of coincidence between these pesticides and reduced bird populations, there are now reports of well-designed experiments which show that DDT and related compounds can cause the observed physiological and behavioral effects (Porter and Wiemeyer 1969, Heath et al. 1969).

The levels in market-basket foods. The Food and Drug Administration has maintained strict control of pesticide residues in food throughout the period of increasing pesticide use. Monitoring of DDT in foods was initiated much earlier than the monitoring of DDT in other sectors of the environment, and as a result the change in levels of contamination of foods is well documented.

The most up-to-date data provided to the Committee were those from a forthcoming publication titled, "Pesticide Residue Levels in Foods in the United States from July 1, 1963, to June 30, 1969," by R. E. Duggan, G. Q. Lipscomb, E. L. Cox, R. E. Heatwole, and R. C. Kling. Their report shows that the level of DDT and its metabolites in total-diet samples reached a peak in 1966, Table 1. The higher level of contamination at that time is believed due to the use of DDT in proximity to forage crops intended for beef and dairy product production.

Table 1. DDT Levels in Market-Basket Diet (in milligrams/day)

FDA Data

	1965	1966	1967	1968	1969	1970
DDT + DDE	0.031	0.041	0.026	0.019	0.016	0.015
DDE		0.028	0.017	0.015	0.011	0.010

Pesticide regulations were modified in 1966 and a drop in DDT compounds in the FDA total-diet samples was observed by 1967. Continued decline in contamination by DDT was apparent each year from 1967 to 1970, but in smaller steps each year. These data suggest that the levels in food are now more closely in equilibrium with the levels of DDT in the environment and thus cannot be expected to change quickly as the result of new restrictions in the use of DDT.

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The results of the FDA survey over the same period have also shown a continuing rise in the percentage of diet samples showing a detectable level of DDT. During the period when the concentration of DDT in food was declining, 1967-1970, the percent of samples showing detectable levels of DDT increased from 38% to 56%. Over the same period the average daily intake of DDT compounds declined from 20 percent of the FAO-WHO acceptable daily intake (0.05 mg/kg of body weight per day) to 10 percent.

The levels in man. The concentration of DDT in man is highly variable from one region to another of the country, and from one economic class to another within regions. As a result, it has been extremely difficult to generalize as to the mean level of chlorinated hydrocarbons in man without a concerted large-scale sampling program. Beginning in 1967, a sampling program large enough to overcome these difficulties and provide meaningful data was initiated.

The Committee was provided with the results of this Human Monitoring Survey (HMS) by the Environmental Protection Agency covering the period from 1967 to 1970. The results are summarized in Table 2. A change in technique during 1968 poses some difficulty for direct comparisons, but a pattern of peak contamination in 1968 is suggested. Other data from the HMS show that the ratio of DDE to DDE + DDT has increased from 0.60 to 0.80 in the past 15 years. Probably because DDE is more persistent, the levels of DDE from the HMS do not show a decline in the 1970 results. Thus, the apparent sequence for DDT compounds appears to be made up of a significant decline in DDT (possibly related to the decline of DDT in food), and a continuing slow increase in the levels of DDE, the primary breakdown product of DDT.

Table 2. Mean Levels of Selected Chlorinated Hydrocarbon Pesticide Residues in Adipose of the General Population (ppm)

Dr. Ann Yobs

Human Monitoring Survey

Year	1967	1968	1968	1969	1970 1/	1971
# Samples	722	3300	3237	3264	2626	
Method	(non-cleanup)	(non-cleanup)	(Mod. 2/ MOG)	(Mod. MOG)	(Mod. MOG)	
pp DDT	1.28	1.52	1.53	1.20	1.15	
pp DDE	4.22	5.28	4.08	4.02	4.12	
Total DDT equivalent	6.22	7.60	6.26	5.81	5.97	

1/ Incomplete data 6/6/71

2/ MOG=Modified Mills, Onley, Gaither cleanup procedure used

The Committee also reviewed the results of studies on DDT-derived material in human milk summarized by the HMS. Unfortunately, no recent data were available for the United States. Data from ten years ago indicated levels in human milk that would lead to a daily intake by a nursing infant at, or in excess of, the acceptable intake recommended by FAO-WHO. In view of the reduced intake of DDT by adults in the United States, it is unfortunate that no more recent data are available on the quantities of DDT and metabolites in human milk.

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Projections of the expected environmental load. Any survey of recent trends in the levels of a contaminant such as DDT and its breakdown products must give attention to the expected levels over the next 5, 10, or 20 years. Several methods are possible. One can utilize a direct plot of the data available of the levels in air, water, soil, food and human tissue and consider the possibility of a continuation from the trends evident in the plotted data points. Due to the great variability in data on DDT levels in the environment and the short period of observations, it is difficult to observe any significant trend at this time.

A second procedure would be to consider the recent monitoring data in the light of everything that is known about the processes of DDT redistribution, the concentration in the environment, and the rate of breakdown to nontoxic compounds. Relatively little information is available as to the rate of turnover of the DDT pool in sediments and in soils, or the rate of breakdown of DDT in aquatic environments. In spite of these deficiencies, our understanding of movement of DDT in the environment allows us to view the variability in reported levels as not due to any decline in environmental load but as due to random movement of the DDT stemming from major upsets in weather, erosion, and riverflow. The quantity of DDT involved in this movement may be traced to the relatively heavy use of DDT in the past 20 years. Although the proportion of the contamination made up by DDT itself probably will show an appreciable decline in the next few years, the combined contamination by DDT and the more stable DDE seems likely to show only a modest decline.

A third approach to making projections of the DDT contamination is to use a systems analysis to provide simulation of DDT levels over the recent period of monitoring and continuing for up to 50 years in the future. Such an analysis has been done for the Committee by Drs. O'Neill and Burke of the Oak Ridge National Laboratory and their results are included as Appendix D. The advantage of a systems model is that each pool of DDT in the environment, and the rate at which it feeds or is fed by other pools, is considered simultaneously in making projections of the anticipated DDT load. The rates of exchange in the environment have had to be estimated from the changes in concentration apparent in the monitoring program.

Of the three approaches to estimating the expected rate of decontamination, the systems study offers the most potential, but it also identifies most clearly the deficiencies in our monitoring program and in our understanding of DDT in the environment. We believe that a re-examination of the projections obtained by O'Neill and Burke should be undertaken regularly as new data become available. Within the limits of the data available at this time, however, the systems study demonstrates that only a slight reduction in environmental contamination can be expected within the next decade. It also shows quantitatively the impact of different levels of use of DDT in the next few years. Although the continued use of relatively small quantities of DDT will not seriously affect the slow decline in environmental load during the next 10 years, it will seriously affect the expected reduction of DDT in the environment over the longer period of two generations.

ANALYTICAL INTERFERENCE WITH THE DETERMINATION OF DDT BY POLYCHLORINATED BIPHENYLS IN THE ENVIRONMENT

A number of polychlorinated biphenyls (PCBs) have been commercially available in the U.S. since 1930. Currently PCBs are being marketed by Monsanto Chemical Company under the trade name of Aroclor with the percentage chlorine designated by the last two digits of their four digit identification number. The first two digits indicate the type; for example, the 1200 series for the biphenyls which are the most common. In the environment, PCBs behave like DDT and many other organochlorine pesticides. PCBs are very stable, resist degradation, are insoluble in water and highly soluble in lipids. It is inevitable, therefore, that PCBs would be concentrated in biological systems since they possess all the characteristics associated with DDT and its metabolites. PCBs are extracted and detected by the same techniques employed for the organochlorine pesticides and consequently residue chemists have had to develop adequate analytical procedures to separate them from associated chlorinated pesticides, prior to quantification.

Prior to 1967, PCBs were either misidentified as specific pesticides such as DDT or viewed as unidentified compounds, possibly unknown pesticidal metabolites. PCBs were previously evident as extraneous, unidentified peaks in GLC chromatograms of extracts of marine fish and birds, until identified in 1966 by Jensen and by Widmark in 1967 and Holmes et al. (1967). It was not until a year or two later that federal monitoring and surveillance sample analysis began to include any routine screening for PCBs, in addition to the usual screening for chlorinated pesticides. The timelag between use and the detection of

PCBs in the environment can be attributed to accumulative concentration over the years and/or recent sophistication in analytical techniques and instrumentation.

The presence of PCBs, DDT and DDT-like moieties in environmental samples presents the residue chemist with both a qualitative and quantitive analytical problem. Because of the similiarity of retention times of gas chromatographic peaks, concentration of certain PCBs can interfere with the accurate determination of p,p'-DDT, p,p'-DDD and p,p'-DDE depending on the type of GLC column employed. One of the most common PCBs found in the U.S. environment, Arcolor 1254, interferes with the GLC peaks associated with p,p'DDT and p,p'DDD and p,p'DDE. Another common PCB, Aroclor 1260, interferes the least with peaks associated with p,p'DDE. The seriousness of the PCB interference or bias in DDT, DDD, and DDE quantifications per se by gas chromatography is dependent on several interrelated factors such as the polarity of the gas chromatographic column packing, the percentage of chlorine in the particular Aroclor being chromatographed, and the concentration and ratio of PCB and DDT and metabolites in the extract.

Considering the most common Aroclors found in the environment, such as 1254 and 1260, unless the ratio for PCB:DDT concentration in the sample is greater than 2:1, the PCB bias to accurate quantifications of DDT and its metabolities by electron capture gas chromatography will be relatively negligible. The PCB bias will interfere and can become serious if the ratio of PBC:DDT concentration in the environmental sample approaches 5:1 or higher. One reason for this is that electron capture detector usually employed in the gas chromatography is considerably less sensitive to the PCB mixture than to individual DDT compounds on a comparative weight basis.

The analytical problems associated with separating PCBs from DDT, DDD and DDE have largely been overcome in the past few years. It cannot be said that the extraction and isolation recovery of PCBs is absolutely quantitative but it can approach 80-90% if adequate preliminary procedures are carefully followed. Separation of PCBs by column chromatography using Florisil has been reported for several pesticides by Reynolds (1969), but p,p'-DDE is eluted along with the PCBs. Armour and Burke (1970) have developed a separation of the DDT analogs and PCBs by employing a silicic acid column. Identification of these interfering PCBs by combined gas chromatography - mass spectrometry has been reported by Widmark (1967) and more recently by Bagley et al. (1970) using a thin-layer chromatography preliminary separation followed by GLC-mass spectrometry. Separation and identification of DDT analogs in the presence of PCBs by two dimensional TLC has recently been proposed by Westfall and Fehringer (1970).

Based on current data, results obtained from environmental monitoring and market surveillance sampling of various foods and other published data, it appears that the most serious chronic PCB contamination is in fish and fish-eating birds. Apparently PBCs are widely distributed among marine birds which are the terminal carnivors of a complex mesh of food chains in the sea. Concentrations of DDT and PCB in marine birds tend to be an order of magnitude higher than in marine fish according to Risebrough et al. (1968). Occasional acute PCB contamination occurs in various areas of our environment leaving the

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mistaken impression that serious PCB contamination is a universal problem. Most of the acute residues of PCBs found to date can be attributed usually to inadvertent or accidental industrial causes. A recent example of acute PCB contamination in poultry was traced to contaminated fish meal resulting from unnoticed leakage of PCBs in the sterlizing vats. The PCB contamination found currently in fruits, vegetables, and in most samples of milk, cheese and eggs appear to be relatively insignificant, at least as to posing a serious analytical bias to DDT or DDE determinations of these samples.

Undoubtedly, prior to recent cognizance by the chemists of possible PCB interference, some of the peaks or portions of them could have been erroneously attributed to DDT, DDD or DDE presence in the sample, particularly if adequate conformation procedures were not followed. In many environmental samples, the seriousness of the PCB bias on the DDT quantification is dependent on the significance of the DDT concentrations in the sample. According to Risebrough et al. (1969), although p,p' DDE is the most abundant of the DDT compounds in the environment, there appears to be no significant PCB interference in DDE quantifications. Consequently, he concludes that total DDT residues in the past, before the extent of PCB interference was known, would not be greatly changed after correction for this interference.

In summary and based on the rather limited knowledge currently available, it would appear that generally the PCB bias to the analytical determination of DDT, DDD or DDE has been and continues to be insignificant in most foods, feeds and environmental samples. An exception, where it appears there may sometimes be serious analytical bias due to the presence of high ratios of PCB:DDT, would be fish and fish-eating birds and any associated byproducts.

Recent reports of PCB content in human adipose tissues indicated that the PCB problem is not of widespread concern. Undoubtedly isolated cases of abnormally high PCB levels will continue to be reported but these are considered to be the exception rather than the rule.

The degree of carcinogenicity of the various common environmental PCBs remains to be elucidated as well as a more complete evaluation of their relative toxicity to mammals. There is limited information that toxicity is associated with the percentage of chlorine but generally the Aroclors are less toxic to mammals than DDT and its metabolites.

TOXICOLOGY

Over a period exceeding 25 years numerous careful studies have been conducted on the toxicity of DDT in experimental animals, in domestic animals, and in man. The information available on the mammalian toxicity of DDT exceeds that available for most other pesticides and for many of the most widely used drugs.

Acute Toxicity. The acute toxicity of DDT to mammals is low. Animal experimentation conducted over 20 years ago established that the median lethal dose of DDT by the oral route in mg/kg is 150-250 for mice and rats, 150-300 for cats and dogs, 300-500 for guinea pigs and rabbits, over 200 for monkeys, over 300 for cows and horses and 1000 for sheep and goats (J.Amer. Med. Assoc., 1951). All subsequent experimentation and use experience has confirmed the early finding of low mammalian toxicity of DDT. A remarkably small number of cases of acute DDT poisoning have occurred in man and there is no well-documented case of fatal uncomplicated DDT poisoning. This situation is in marked contrast to the high acute toxicity of potential substitutes for DDT including organophosphorus insecticides.

The pharmacological effects of oral doses of DDT in man have been studied. There are some differences in the doses reported to produce various effects but the types of changes and their duration were the same in all studies. The lowest oral doses of DDT reported to produce effects in man were those used by Velbinger (1947). In that study, oral doses of 250 or 500 mg per man in suspension or oil solution produced no effect except a variable, slight disturbance of the sensitivity of the mouth. Doses of 750 or 1000 mg in oil solution led to disturbances of the sensitivity of the lower part of the face, uncertainty of gait, malaise, hypersensitivity to contact, cool moist skin but no changes in reflexes. Discomfort reached a peak in about 6 hours. A dose of 1500 mg in oil solution produced prickling of the tongue beginning about 2.5 hours after ingestion. Disturbance of equilibrium, dizziness, confusion and tremors of the extremities gradually increased. A peak reaction characterized by malaise, headache, fatigue, and delayed vomiting was reached about 10 hours after ingestion and recovery was almost complete in 24 hours.

Chronic toxicity. Various studies on the chronic toxicity of DDT have been conducted. It should be noted that the parameters measured in toxicity studies done 20 years ago were fewer than those that are considered today.

Long-term exposure to low levels of DDT produces histological changes predominantly in the liver. All investigators who have studied the chronic toxicity of DDT to rats have observed the same changes although the doses required to elicit the effects were not exactly the same in all studies. In 1950 Laug et al., reported that rats fed 5 ppm of DDT for 6 months showed detectable liver changes. Ortega et al., (1956) fed levels of DDT from 5 ppm to 400 ppm to rats for 6 months and followed recovery in some animals for 12 months. The lowest dietary level that produced liver changes (hypertrophy, inclusion bodies, and cytoplasmic granulation) was 15 ppm in males but higher doses were required in females. The liver changes were reversible after withdrawal of the diet. Monkeys fed dietary levels of 5, 50, 200 and 5000 ppm of DDT for a prolonged period showed no liver pathology up to 200 ppm.

The most informative chronic studies on man were those done by Hayes and associates (1956, 1971) in which known doses of DDT were fed for prolonged periods. In these studies some subjects received doses as high as 35 mg per day for 21.5 months and some of them were observed for 5 years after completion of the feeding period. The level fed to these men was 535 times the average normal intake and no clinical or laboratory evidence of an adverse effect was observed. Other studies have been conducted on man with prolonged and intensive exposure to DDT in manufacturing plants (Laws et al., 1967). In the most recent study of this type 35 men with 11 to 19 years of exposure in a DDT manufacturing plant showed no clinical or laboratory effects attributable to exposure to DDT even though the average daily intake was estimated from storage and excretion data to be 17.5 to 18 mg per man per day as compared with an average of 0.04 mg per man for the general population. The chronic toxicity studies on DDT have provided no indication that the insecticide is unsafe for humans when used in accordance with commonly recognized practice. The chronic toxicity tests on man have not been extensive enough with respect to both the numbers of individuals and duration of follow-up to contribute information concerning possible carcinogenic effects.

Carcinogenicity of DDT. The question of the potential carcinogenicity of DDT has been studied in a number of laboratories. Fitzhugh and Nelson reported in 1947 that DDT fed in high doses to rats causes a slight increase in hepatic cell tumors. A statistically significant increase in hepatomas was noted in the Bionetics Study (Innes et al. 1969) in two strains of mice in both sexes. Weisburger et al. (1965) reported that the frequency of hepatomas after N-2-fluorenylacetamine was increased as the latent period decreased after DDT feeding. Tarján and Kemény (1969) reported multigeneration studies in mice fed 3 ppm DDT. A generalized increase in the frequency of tumors in the F_2 and following generations was noticed. Multigeneration studies in mice, in progress, sponsored through the International Agency for Research on Cancer both at Lyon and Milan, show a statiscally significant increased incidence of hepatomas - although the studies have not been completed and final conclusions cannot be drawn.

Studies by Halver (1967) have shown that DDT does cause hepatic cell tumor in trout at relatively low doses. Some studies, generally of a duration too short to detect any but strong carcinogens, have not shown a carcinogenic effect of DDT. (Ortega, 1956, Ottoboni, 1969).

It seems clear, therefore, that DDT is capable of causing hepatomas in rodents and further that there is some evidence of carcinogenicity with respect to other sites.

The significance of hepatoma induction is unclear. In the IARC Lyon Study, two of the hepatomas showed matastases to the lungs, both in animals given DDT. With other hepatomas after transplantations to other mice tumor growth did not occur. Many experts in carcinogenesis feel that hepatoma induction is essentially equivalent to carcinogenesis, while others feel that hepatomas are reversible lesions. The demonstrated ability of DDT to stimulate the hepatic endoplasmic reticulum and microsomal mixed function oxidase activity and to cause an increase in liver weight may be involved. Carbon tetrachloride, chloroform, and brombenzine show increased liver toxicity after stimulation of microsomal oxidase activity. One expression of this toxicity might be hepatoma production.

The evidence to date clearly shows that DDT induces hepatomas and suggests it may be carcinogenic.

The implications of this finding for man must be drawn with care. Considerable uncertainty exists with respect to the ability to extrapolate effects seen in small numbers of laboratory animals at high doses to large numbers of humans exposed to low doses. If one accepts that an eventual human health hazard is a possibility, it must be recognized that very little can be done at this time. The world burden of DDT is so high compared to the current annual use in the U.S., that instant as opposed to a rapidly phased cessation of DDT usage would probably make no significant difference in human exposure levels. Expanded use of DDT is contraindicated.

There is no evidence from human epidemiological studies to shed light on the possible human carcinogenicity of DDT. Occupational exposure studies (Laws, 1967) have been too limited in sample size and duration to follow-up to detect a moderate or weakly carcinogenic action of DDT. Long-term studies of occupational exposure of DDT are urgently needed.

Mutagenicity of DDT. Few data exist in the literature concerning the potential mutagenicity of DDT and its derivatives. At high doses, it can cause C. mitosis (colchicine-like effects) in dividing plant cells. A recent report suggests DDE may be associated with chromosome rearrangements in wild Drosophila, but the association was quite indirect being related to DDE levels in fat bodies of local frogs. Unpublished data appear to be contradictory. In general, acute high doses were used. There exists an urgent need for extended dose experiments with a proper dose range. It is impossible to state whether DDT is or is not mutagenic in mammalian systems.

Effects of DDT on reproduction in mammals. The conventional three-generation reproduction study on rats that is used to evaluate drugs and pesticides teratogenicity has recently been done with DDT fed at levels of 0, 20, and 200 ppm (Ottoboni, 1969). There are no teratogenic effects and fertility and viability of the young were not affected. Another aspect of this study relates to the influence of DDT consumed in the milk during the suckling period. There were clearly no effects from feeding 20 ppm of DDT to the mothers on the survival of young to the weaning age. In another study DDT fed to mice at 7 ppm produced a slight significant reduction in the number of liters per pair in one strain of mice but not in another strain (Ware and Good, 1967). In one recent study doses of DDT of 1 mg/kg and higher doses were given to rats daily by the intraperitoneal route for 21 days beginning at 24 hours after birth (Fahim et al., 1970). These high doses caused mortality and a decreased growth rate.

Toxicity of DDT to birds. Evaluation of the toxicity of DDT to various bird species has not kept pace over the last 25 years with mammalian toxicity studies on DDT and other pesticides. It is only recently that standard toxicological procedures for mammalian toxicity measurements have begun to be applied to selected species of birds. The needs in this area were summarized in 1968 by L. Stickel who indicated that interpretations concerning potentially dangerous effects of organochlorine pesticides require experimental studies designed for that purpose and aimed at finding diagnostic methods for identification of lethal and sublethal toxic limits. The same author recommended that species differences should be determined in the absence of extraneous physiological and environmental stresses. Thus it is being recognized that the important concept of dose-response relationships and species differences have an important bearing on DDT poisoning in birds. It has long been recognized in mammalian toxicology that analysis of tissues for toxic substances has limited value in established cause and effect relationships for intoxication by chemical agents. It is only the presence of abnormally high levels of toxicants consistent with levels known to be associated with lethality and the absence of other toxic substance that can justify a conclusion that a certain chemical agent is responsible for mortality. Tissue levels that have been found justify a suspicion that bird populations have been affected by DDT but the quantitative toxicological data needed to support a definite conclusion are still meager. The apparent relationships between increased mortality of wildlife and DDT spraying programs has been discussed by Dustman and Stickel (1969).

One example of the type of study that is extremely valuable in understanding the effects of DDT on survival and reproduction is the one recently reported by Heath et al., (1969) in which mallard ducks were fed various levels of DDT, DDE, and DDD. The results of this study showed that DDE at levels of 10 ppm and 40 ppm severely impared reproduction success, caused eggshell thinning, and increased embryo mortality. Survival of hatchlings was not affected. DDD did not impair reproductive success. DDT induced thinning of shells and reduced duckling survival only when a level of 25 ppm was fed. None of the treatments induced crippling among hatchlings. This type of information is valuable because it shows the higher sensitivity of this species to DDT and DDE than is observed in mammals and, on the other hand, it shows that dietary levels far in excess of those in food of species that are protected by established tolerances are required to produce injury in this bird species. Many additional studies are needed not only with individual pesticides but also with combinations of environmental chemicals and DDT.

Toxicity of DDT to fish. The available data on fish demonstrate clearly that many species are highly susceptible to DDT. The high susceptibility of fish and the hazards from injury to their food chain by DDT have been known at least since 1944 (Ginsburg, 1945). Thus the high toxicity of DDT to goldfish was described in 1944 (Ellis et al.) and deaths of young fish in waters sprayed with DDT were reported in 1946 (Pierlou). Work of a quantitative nature on the effects of DDT on fish has been in progress for many years and it was perhaps greatly stimulated by the early use of fish as a species for the bioassay of DDT. Controlled studies of the effects of many pesticides on fish and shellfish have been conducted by the U.S. Department of the Interior, Bureau of Commerical Fisheries. (Now: Gulf Breeze Marine Laboratory, EPA). The development of a monitoring system using oysters which accumulate residues above 0.01 ppb of DDT from test solutions and store it at magnifications ranging from 15 to 70 thousand times was an important advance in determining the contamination of water sources. The work that has been accomplished in this area leaves no doubt that the levels of DDT of importance for survival of aquatic species are far below those of concern to mammalian species. There is sufficient toxicological information on DDT in aquatic species to indicate that reduction and prevention of contamination of water sources is a problem of major concern.

Biochemical Effects on DDT. DDT is capable of modifying the activity of hepatic microsomal enzymes that catalyze the biotransformation of many drugs and other chemicals usually to less active compounds. DDT produces this effect by inducing synthesis of these enzymes. The consequent increase in endoplasmic reticulum explains the hypertrophy of the liver that has long been an established effect of sufficiently high doses of DDT. Very few

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quantitative studies have been done on dietary levels of DDT required to produce enzyme induction. In the male rat the minimum dietary level that causes significant enzyme induction by DDT is 1 ppm. Female rats are less sensitive to enzyme induction by DDT which correlates with failure to observe morphological changes at the same dose in female rats as in males.

A study of 18 workers with an average of 14.4 years of exposure to DDT in a manufacturing plant showed evidence of enzyme induction. However, even at this relatively high intake, the values, although significantly different, mostly fall within the normal range (Poland et al., 1970).

Recent evidence suggests that DDT can inhibit MG++ ATPase in rabbit brain (Koch, 1969) and Na K ATPase in teleost GI tract (Janicki and Kinter, 1971) and can form charge transfer complexes with nerve components (Narahashi and Haas, 1967). DDT also causes a decrease or abolition of the potential difference and short circuits current across the isolated toad bladder wall (Sides, 1971). These observations may provide a beginning insight into the acute and subacute CNS toxicity of DDT and since Na K ATPase is important in maintaining salt and water balance in telesots they may similarly provide the basis for an explanation of the very high toxicity of DDT for teleosts.

Other recent reports describe an effect of DDT on the thyroid function of birds (Jeffries and French, 1969). Whether this effect, or the effect of DDT on the hepatic mixed function oxidases plays a role in the eggshell thinning action of DDT is uncertain.

Interactions involving mechanisms other than enzyme induction are not known for DDT except for the antagonistic action of barbiturates against the central nervous system action of DDT. This antagonism constitutes the basis for the antidotal action of phenobarbital in acute poisoning by DDT. It should be noted, however, that systematic studies on possible additive or synergistic effects have not been conducted on DDT with other environmental chemicals such as PCB's.

NEEDS

There is little question from man's viewpoint relative to the protection of his health and welfare and to the production of food and fiber that his broad-ranging competition with insects requires measures for the control of their depredation. Although there are many types of useful insect control practices, it was essentially concluded in the most recent scientific analyses (Reports of the Jensen Committee and Mrak Commission, 1969), that, for the immediate and perhaps even foreseeable future, insect control will depend to a major extent on chemical insecticides, particularly as directed toward integrated and supervised programs of insect pest management. Encouraging progress is continuing on the development of non-insecticidal control techniques, but actual achievement of their broad-scale applications to insect control problems remains for the future.

The specific question of whether DDT should or should not be available for use as a component of the presently available chemical control resources is difficult. The cumulative scientific evidence concerning the persistant nature and toxicological actions of DDT have been summarized and analyzed above. It is obvious that there are still a number of questions in which conclusive evidence is not complete, even though DDT has been characterized as the most broadly studied organic chemical ever produced. Present scientific evidence permits useful conclusion on many, but not all, important questions.

The production and use of DDT in the United States has been steadily decreasing for a variety of reasons since the peak period of

1960 and 1963. The various types of registered uses have been drastically reduced with recent cancellations of registrations for use on tobacco and shade trees, in and around the home, and in aquatic environments (except those essential to the control of disease vectors as determined by Public Health Officials) in 1969 and for other uses on a wide variety of crops, animals, and products in 1970. Of the comparatively small number of remaining uses, approximately 70% is used for cotton insect control. Other extensive uses are for control of insects on soybeans and peanuts.

There is little difficulty on humanitarian grounds in appraising the justification for the continued usefulness and cost-benefit ratio of DDT in such programs as the World Health Organization (WHO) malaria control program. Even the most dedicated proponents of banning DDT appear at this time to exclude this program from their recommendations. This apparently results from the acceptance of the results of the program in terms of conservation of human lives and alleviation of misery, the many factors justifying the choice of DDT as the agent of use, and the apparent low potential of this use to nontarget environmental contamination. There is considerably more difficulty in evaluating the necessity for other uses.

The Committee received information on essential uses of DDT for continued registration presented by the various States and evaluated by a Special Review Group on DDT Registration, advisory to the Secretary of Agriculture. It has not been possible in the time available for the Committee to address itself to the specifics of justification for each registration recommended for continued use, and the range of variables for each is such that it is virtually impossible to formulate responsible general criteria. The impression of the Committee is that the recommendations of the States as presented show considerable variation in the extent of scientific evidence on which they were based. The primary justification offered for the recommended continuations appears to be that no effective substitutes for DDT are available, but this actually derives from a broad variety of reasons. Alternative insecticides may present a more acutely toxic hazard to both man and other nontarget organisms. They are likely to be less residual, requiring more frequent applications. Because of the relatively low cost of DDT formulations, alternative insecticides are almost certain to be more expensive. The substitution of less fully evaluated materials than DDT may supplant the known problems of DDT with other unknown problems of more serious consequences. Perhaps the most discouraging and least permittable reason is that potential alternatives are not registered for such uses. The development of satisfactory alternatives presents a complex problem which may be further complicated by the continued use of DDT tending to act as a deterrent to their development. One obvious solution is to retain the cancellation of all DDT registrations, thus forcing the further development and registration of alternatives. But what then of the interim, when no alternatives are available? The Committee recognized the significant reductions in domestic use which have already occurred and their potential implications. The Committee was particularly interested in assessing evidence for indications of decreases in environmental contamination during the period of decreasing use. Although the evidence and projections from it are not indisputably clear, they strongly suggest that residues of DDT have begun to decrease and that residues of derived materials as represented by DDE appear to have peaked. These suggest that registrations and use of DDT should continue to be redueed with the ultimate goal of virtual elimination of any signficant additions to the environment. Thus, the registration of DDT cancelled in PR 71-1, and the essential uses as receommended by the various States should be reevaluated, and any continued uses of DDT should be based on detailed scientific review, evaluations, and justifications by appropriate panels of qualified entomologists and then by independent panels of scientist knowledgeable in toxicological and environmental sciences. Additionally, provisions should be made for continuing review so that new scientific research and evidence may be taken into account in evaluating and justifying the continuing essentiality of uses relative to assuring the further elimination of nonessential uses as rapidly as possible. The Committee is cognizant that changes in existing laws concerned with such matters are currently in discussion stages and might be expected to facilitate the courses of action discussed above.

CONCLUSIONS

From the data available to the Committee on the use of DDT and its impact upon the environment, upon man, and upon other nontarget species the Committee has reached the following conclusions on pertinent scientific issues.

1. The quantities, uses, and acreage receiving DDT in the United States have declined rapidly and continuously within the past ten years, but the quantities of DDT (and its metabolites) detectable in water and soil have not markedly decreased.

2. DDT and its metabolites have spread from their sites of application throughout the global biosphere. The routes and mechanisms of movement are only partially understood, but include atmospheric transport, surface runoff, re-suspension from sediments, and the biological food chain. Large quantities of DDT are accumulating in the estuaries and oceans.

3. DDT and its metabolites persist for years, concentrate in some nontarget organisms, display a variety of adverse biochemical and physiological effects, threaten to reduce or eliminate some nontarget species, and lower the marketability of valuable fish and shellfish. Evidence from a limited number of adequate studies and a large number of less critical and interpretable observations convinces us of present or probable damage to some molluscs, crustaceans, fish, and birds.

4. While forecasts of the prospective decline in DDT contamination of the environment are difficult and necessarily tentative the evidence available indicates that present contamination of food

and native biota is principally attributable to a large existing environmental pool that is recycling at low concentrations and which seems likely to decrease relatively slow over a number of decades.

5. There has been slight but significant reduction in the amount of DDT present in the foods ingested by man although the frequency of low-level contamination has increased. There has been no significant reduction in the body burden of DDT and its metabolites carried by man.

6. DDT has a very low acute toxicity to man and his domestic animals, and exposure to high doses for short periods of time does not appear to cause any irreversible damage.

7. Prolonged exposure to relatively high doses of DDT has been demonstrated to be tumoragenic and possibly carcinogenic in rodents. With our present knowledge and information, these data cannot be directly extrapolated to man and his domestic animals. Based upon the current intake and storage levels of DDT and its metabolites in these species, the probability of tumoragenesis and carcinogenesis is low.

8. The evidence that DDT may exert a mutagenic effect in certain situations is incomplete and conflicting at the present time. There is no evidence that DDT exerts a teratogenic effect.

9. The polychlorinated biphenyl compounds, because of their analytical similarily to DDT, have in the past been confused with DDT and some of its metabolites. These substances are also widespread environmental pollutants and could have been responsible for some of the reports of the wide distribution of DDT in the environment.

RECOMMENDATIONS

1. Reduce the use of DDT in the U. S. at the accelerated rate of the past few years with the goal of virtual elimination of any significant additions to the environment.
2. Encourage effective development and registration of alternative insecticides or insect control methods capable of replacing DDT so as to accelerate the reduction of DDT recommended above.
3. Provide for review of any continued uses of DDT for scientific basis and justifications by qualified entomologists and then by independent scientists knowledgeable in the toxicological and environmental areas.
4. Create, by 1973, an appropriate panel of experts to review and analyze new evidence on the remaining scientific questions posed by the presence of DDT and its metabolites in the environment, and provide a mechanism for the review of the new information about DDT at regular intervals.
5. Review throughly the PCB compounds and their environmental distribution, hazards, and interrelation with DDT.*
6. Provide for continued availability of DDT for specific uses essential to the Public Health control of disease-bearing insect vectors until satisfactory alternatives are developed.

* As this report was being completed, information was received that a task force will be coordinated through the Office of Science and Technology and the Council on Environmental Quality to investigate polychlorinated biphenyls in food and other components.

THE IMMINENCY OF HAZARDS

The Federal Insecticide, Fungicide, and Rodenticide Act empowers the Administrator of the Environmental Protection Agency to suspend immediately the registration of an economic poison (pesticide) whenever he determines that "such action is necessary to prevent an imminent hazard to the public." The Committee has been specifically requested to provide its expert, independent judgement on the issue of whether products containing DDT constitute "an imminent hazard to the public." We recognize the signal importance of a decision on this question, but consider it to be outside of our primary charge "to deal with scientific issues." Therefore, we provide a separate statement of our opinion of hazard.

The definition of "imminent hazard" is important in reaching such a judgement. The Committee has used the concept stated as follows in the position statement of 18 March 1971 by the Environmental Protection Agency titled "Reasons Underlying the Registration Decisions Concerning Products Containing DDT, 2,4,5-T, Aldrin and Dieldrin."

> "....This Agency will find that an imminent hazard to the public exists when the evidence is sufficient to show that continued registration of an economic poison poses a significant threat of danger to health, or otherwise creates a hazardous situation to the public, that should be corrected immediately to prevent serious injury and which cannot be permitted to continue during the pendency of administrative proceedings. An "imminent hazard" may be declared at any point in a chain of events which may ultimately result in harm to the public. It is not necessary that the final anticipated injury actually have occurred prior to a determination that an "imminent hazard" exists. In this connection, significant injury

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or potential injury to plants or animals alone could justify a finding of imminent hazard to the public from the use of an economic poison...."

Using this concept of "imminent hazard", the Committee agreed that:

A. The present reported annual usage level of DDT does not present an imminent hazard to human health in terms of individual bodily functions and safety.

B. DDT and its derivatives are serious environmental pollutants and present a substantial threat to the quality of the human environment through widespread damage to some nontarget organisms. There is, therefore, an imminent hazard to human welfare in terms of maintaining healthy desirable flora and fauna in man's environment.

Although the Committee has agreed that DDT represents an imminent hazard to human welfare because of the quantities of this substance currently present in the environment, it believes that either immediate suspension or rapid and continuous decrease in the use of DDT will achieve essentially the same results.

Respectfully submitted,

James G. Hilton, Ph.D.

APPENDIX A

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APPENDIX B

PERSONS APPEARING BEFORE THE COMMITTEE

First Meeting

June 7 & 8, 1971

Mr. Harold G. Alford, Pesticides Regulation Division, Environmental Protection Agency

Dr. R. R. Bates, National Cancer Institute, National Institutes of Health

Mr. C. B. Fielding, Office of General Counsel, Environmental Protection Agency

Dr. O. Garth Fitzhugh, Office of Pesticides Programs, Environmental Protection Agency

Dr. Wayland J. Hayes, Jr., Vanderbilt University

Dr. William E. Hazeltine, Butte County (California) Mosquito Abatement District

Dr. Clarence Hoffman, Agricultural Research Service, United States Department of Agriculture

Dr. C. R. Jordan, University of Georgia

Dr. Thomas H. Jukes, University of California, Berkeley

Dr. Edward R. Laws, Jr., Johns Hopkins University

Dr. Griffith E. Quinby, Wenatchee, Washington (spoke at the request of Crop King)

Mr. Samuel Rotrosen, Montrose Chemical Corporation

Mr. Max Sobelman, Montrose Chemical Corporation

Dr. Fred H. Tschirley, Office of Secretary, United States Department of Agriculture

Mr. P. W. Whiteaker, Pesticides Regulation Division, Environmental Protection Agency

Dr. Charles F. Wurster, Jr., University of New York at Stoneybrook, representing Environmental Defense Fund

Dr. David Young, Mississippi State University

Second Meeting

June 24 & 25, 1971

Mr. Harold G. Alford, Pesticides Regulation Division, Environmental Protection Agency

Dr. Phillip A. Butler, Gulf Breeze Marine Laboratory, Environmental Protection Agency

Dr. Fred DeSerres, Biology Division, Oak Ridge National Laboratory

Mr. Reo E. Duggan, Office of Compliance, Food and Drug Administration

Mr. Herman Feltz, Water Resources Division, U.S. Geological Survey

Dr. William M. Upholt, Office of Pesticides Programs, Environmental Protection Agency

Dr. G. B. Wiersma, Office of Pesticides Programs, Environmental Protection Agency

Dr. Ann Yobs, Division of Community Studies, Environmental Protection Agency

Third Meeting

July 21, 22, & 23, 1971

Mr. Harold G. Alford, Pesticides Regulation Division, Environmental Protection Agency

Mr. Lowell E. Miller, Office of Pesticides Programs, Environmental Protection Agency

Mr. Charles L. Smith, Pesticides Regulation Division, Environmental Protection Agency

APPENDIX C

EXHIBITS FURNISHED THE COMMITTEE

1. Charge to DDT Advisory Committee

2. PR Notice 70-19, Cancellation of Registration of Certain DDT Products, August 18, 1970

3. PR Notice 71-1, Cancellation of Registration Under the Federal Insecticide, Fungicide, and Rodenticide Act of Products Containing DDT, January 15, 1971.

4. PR Notice 71-5, Cancellation of Registration of Dichloro Diphenyl Dichloroethane (TDE), March 18, 1971.

5. Petition from I. T. Fisk, Counsel for Crop King Company, to U.S. Department of Agriculture, September 28, 1970.

6. Petition from R. L. Ackerley, Counsel to Montrose Chemical Corp., to William D. Ruckelshaus, February 18, 1971.

7. Petition from I. T. Fisk, Counsel for Crop King Company, to Environmental Protection Agency, February 16, 1971.

8. Reasons underlying the registration decisions concerning products containing DDT, 2,4,5-T, Aldrin and Dieldrin, Environmental Protection Agency, March 18, 1971.

9. Environmental Defense Fund, Incorporated, et al. Petitioners v. William D. Ruckelshaus, Administrator, Environmental Protection Agency, Respondent. Petition for Review of an Order of the Secretary of Agriculture, Docket 23,813, January 7, 1971.

10. List of References on DDT

    A. Mammalian Toxicity and Human Exposure

    B. Mammalian and Avian

    C. Aquatic and Marine

APPENDIX D

A SIMPLE SYSTEMS MODEL FOR DDT AND DDE MOVEMENT IN THE HUMAN FOOD-CHAIN

R.V. O'Neill[1]
and
O.W. Burke[2]

Abstract of a Report submitted to the Advisory Committee on DDT[3]

At the request of the DDT Advisory Committee, a brief study has been carried out to develop a simple model of DDT and DDE movement in the food chain supporting man. The objective is to examine the potential of a systems model for estimating anticipated DDT load in humans and in the environment under various options of DDT usage and application. The model was developed from the data in Table 1 supplied by the Committee, but also includes the ecolo-ical understanding of environmental transports and biological flux processes developed from recent ecosystem modeling research. However, the model is designed for a specific purpose and readers are cautioned against drawing implications from it that are not warranted by the data or the model at this time.

The limited data period constituted the first basic constraint on the model. The second important constraint was the

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[1] Ecological Sciences Division ORNL

[2] Instrumentation and Control Division, ORNL

[3] Carried out and supported in part through the Deciduous Forest Biome Study of the U.S. International Biological Program.

brief period of time available to design the model; derive values for parameters, and utilize the model to examine prospective future concentrations of DDT in the environment.

It appears that an adequate model of DDT movement should consider DDT and DDE separately for the environment, for food, and for man, resulting in a total of six state variables and six differential equations. Ideally, the DDT in food supply should be divided into a portion which has residual pesticide by direct application, with subsequent losses at a rapid rate, and a portion which receives a continuing low level contamination from an environmental pool. This division is dictated by an analysis of the data which shows that after cessation of DDT applications near forage in 1966, the system has become dominated by an environmental pool with a slow turnover rate.

Because of the limited data on DDE, the model actually developed was necessarily simple. The model could not be solved with an explicit environmental pool, because data on this pool were completely lacking. Also, to reduce the number of parameters to be fitted from the data, it was necessary to consider DDT plus DDE together in food and man. This allows a model with only five unknowns, but incurs the disadvantage of not being able to distinguish the dynamics of the two forms.

Concern about the statistical reproducibility of the data has resulted in further limitations in the model. Questions arise with regard to the 1968, 1969, and 1970 data on human adipose tissue. The authors have had to assume that these data fluctuations in DDT and DDE levels were due to sampling errors, and that the true response curve is a slow reduction in DDT and DDE from a peak in 1968. This assumption is partly based on the fact that to postulate a recent upturn in the level of pesticides in man we would require mechanisms that we do not know and cannot support on biological grounds. If our assumption, that an initial downturn is evident from the 1967-1970 data, is not supported, then our model is in serious error, and our forecasts underestimate prospective concentrations in man. The authors view the decision to assume that the data are variable, and to make conservative estimates of concentrations in man, as the most important single assumption of our model.

The model adopted was expressed as a system of three differential equations where one expressed the fraction of the food contaminated by direct spraying, another the portion of the food which receives DDT from the environmental pool (both in response to a forcing function of DDT usage), and the third described concentration in human adipose tissue. The parameters in each equation are $\underline{a_i}$, expressing the rate of uptake in relation to the magnitude of the source, and $\underline{b_i}$, elimination constants. Intake by man, therefore, is proportional to the sum of the first two elements, since the food concentration is the result of both sources of contamination. The model was first implemented on an analog computer, programmed so that the values of the parameters could be changed through a wide range of values, and the model behavior compared to the data points. Values of the parameters were changed until the model successfully mimicked the data for

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1965-1970. The function representing DDT input to the system was taken from the data on DDT usage, and was assumed to be linear. The final model generated values for food concentrations and human load that matched the data available very faithfully. A series of simulations then was performed on the digital computer to examine changes in DDT and DDE concentrations in man under various assumptions about DDT application. A series of six runs was made for a period of fifty years, four of which are summarized in Table 2. In the first case (column 1), the model was used to extrapolate concentrations in human adipose tissue with DDT usage continuing to decrease at the present rate of about seven million pounds a year. The assumption of linear decrease implied an anticipated application of 5,000,000 pounds in 1972 dropping to zero in 1973. The model predicts that the level in man will continue to decline, reaching 1 ppm by 2002, but that fifty years after ceasing DDT usage, there would still be measurable quantities of DDT plus DDE in man. The second column shows the result of ceasing DDT usage in 1972 (i.e., a zero application). The differences between columns 1 and 2 are detectable only in the second decimal place. The third example shows the results when DDT usage is maintained at five million pounds per year after 1972. In this instance, the levels in man would continue to decline, but would be maintained at a significantly higher level in the long run, remaining above 1 ppm after fifty years. The fourth column describes the reponse to continue DDT usage at the level of 1966. After the initial decline, apparently due to improved application practices, and the slowness of buildup in the environmental pool, the concentrations in man begin to rise by 1978, eventually reaching some asymptotic value above 6.7 ppm.

In evaluating these results, the limitations of the model structure, the data, period, and the data reliability must all be considered. The predicted long-term concentrations should not be viewed with as much confidence as the general response patterns over time and the relative concentrations under the various treatments.

Table 1. DATA UTILIZED FOR MODELING DDT AND DDE MOVEMENT THROUGH THE HUMAN FOOD CHAIN; THE DATA WERE SUPPLIED BY THE ADVISORY COMMITTEE FROM THE RESULTS OF CURRENT MONITORING PROGRAMS IN U. S. GOVERNMENT AGENCIES.

Year	DDT Usage (10^6 lbs.)	DDT + DDE in market-place diet (mg./day)	DDT + DDE in human adipose tissue (ppm)
1965	53	.031	
1966	46	.040	
1967	40	.026	4.65
1968	33	.019	5.61
1969		.016	5.22
1970		.015	5.27

Table. 2. DDT + CONCENTRATIONS IN HUMAN ADIPOSE TISSUE PREDICTED BY THE MODEL FOR VARIOUS ASSUMPTIONS ABOUT DDT USAGE.

Year	Continued reduction of DDT usage at present rate.	Zero future usage of DDT	DDT usage maintained at 5 x 10^6 lbs./year	DDT usage maintained at 1966 levels
1970	5.14	5.14	5.14	5.30
1974	4.18	4.13	4.20	5.08
1978	3.41	3.35	3.53	5.32
1982	2.78	2.73	3.02	5.60
1986	2.27	2.23	2.60	5.84
1990	1.86	1.82	2.25	6.03
1994	1.52	1.49	1.98	6.20
1998	1.24	1.21	1.75	6.32
2002	1.01	0.99	1.56	6.43
2006	0.82	0.81	1.41	6.52
2010	0.67	0.66	1.29	6.59
2014	0.55	0.54	1.18	6.65
2018	0.45	0.44	1.10	6.70
2022	0.37	0.36	1.03	6.73