IPCS International Programme on Chemical Safety

Environmental Health Criteria 83

DDT and its Derivatives – Environmental Aspects

Published under the joint sponsorship of the United Nations Environment Programme, the International Labour Organisation, and the World Health Organization

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The **International Programme on Chemical Safety (IPCS)** is a joint venture of the United Nations Environment Programme, the International Labour Organisation, and the World Health Organization. The main objective of the IPCS is to carry out and disseminate evaluations of the effects of chemicals on human health and the quality of the environment. Supporting activities include the development of epidemiological, experimental laboratory, and risk-assessment methods that could produce internationally comparable results, and the development of manpower in the field of toxicology. Other activities carried out by the IPCS include the development of know-how for coping with chemical accidents, coordination of laboratory testing and epidemiological studies, and promotion of research on the mechanisms of the biological action of chemicals.

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NOTE TO READERS OF THE CRITERIA DOCUMENTS

Every effort has been made to present information in the criteria documents as accurately as possible without unduly delaying their publication. In the interest of all users of the environmental health criteria documents, readers are kindly requested to communicate any errors that may have occurred to the Manager of the International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland, in order that they may be included in corrigenda, which will appear in subsequent volumes.

* * *

A detailed data profile and a legal file can be obtained from the International Register of Potentially Toxic Chemicals, Palais des Nations, 1211 Geneva 10, Switzerland (Telephone no. 988400 - 985850).

ENVIRONMENTAL HEALTH CRITERIA FOR DDT AND ITS DERIVATIVES - ENVIRONMENTAL ASPECTS

A WHO Task Group on Environmental Health Criteria for DDT and its Derivatives - Environmental Aspects met at the Institute of Terrestrial Ecology, Monks Wood, United Kingdom, from 14 to 18 December 1987. Dr I. Newton welcomed the participants on behalf of the host institution, and Dr M. Gilbert opened the meeting on behalf of the three co-sponsoring organizations of the IPCS (ILO/UNEP/WHO). The Task Group reviewed and revised the draft criteria document and made an evaluation of the risks for the environment from exposure to DDT and its derivatives.

The first draft of this document was prepared by Dr S. Dobson and Mr P.D. Howe, Institute of Terrestrial Ecology. Dr M. Gilbert and Dr P.G. Jenkins, both members of the IPCS Central Unit, were responsible for the overall scientific content and editing, respectively.

* * *

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INTRODUCTION

There is a fundamental difference in approach between the toxicologist and the ecotoxicologist concerning the appraisal of the potential threat posed by chemicals. The toxicologist, because his concern is with human health and welfare, is preoccupied with any adverse effects on individuals, whether or not they have ultimate effects on performance or survival. The ecotoxicologist, in contrast, is concerned primarily with the maintenance of population levels of organisms in the environment. In toxicity tests, he is interested in effects on the performance of individuals - in their reproduction and survival - only insofar as these might ultimately affect the population size. To him, minor biochemical and physiological effects of toxicants are irrelevant if they do not, in turn, affect reproduction, growth, or survival.

It is the aim of this document to take the ecotoxicologist's point of view and consider effects on populations of organisms in the environment. The risk to human health of the use of DDT was evaluated in Environmental Health Criteria 9: DDT and its Derivatives (WHO, 1979). This document did not consider effects on organisms in the environment, but did consider environmental levels of DDT likely to arise from recommended uses. No attempt has been made here to reassess the human health risk; the interested reader should refer to the original document, which contains the relevant literature in this area.

This document, although based on a thorough survey of the literature, is not intended to be exhaustive in the material included. In order to keep the document concise, only those data which were considered to be essential in the evaluation of the risk posed by DDT to the environment have been included.

The term bioaccumulation indicates that organisms take up chemicals to a greater concentration than that found in their environment or their food. 'Bioconcentration factor' is a quantitative way of expressing bioaccumulation: the ratio of the concentration of the chemical in the organism to the concentration of the chemical in the environment or food. Biomagnification refers, in this document, to the progressive accumulation of chemicals along a food chain.

1. SUMMARY AND CONCLUSIONS

1.1 Physical and Chemical Properties

DDT is an organochlorine insecticide which is a white crystalline solid, tasteless and almost odourless. Technical DDT, which is principally the *p,p'* isomer, has been formulated in almost every conceivable form.

1.2 Uptake, Accumulation, and Degradation

The physicochemical properties of DDT and its metabolites enable these compounds to be taken up readily by organisms. High lipid solubility and low water solubility lead to the retention of DDT and its stable metabolites in fatty tissue. The rates of accumulation into organisms vary with the species, with the duration and concentration of exposure, and with environmental conditions. The high retention of DDT metabolites means that toxic effects can occur in organisms remote in time and geographical area from the point of exposure.

These compounds are resistant to breakdown and are readily adsorbed to sediments and soils that can act both as sinks and as long-term sources of exposure (e.g., for soil organisms).

Organisms can accumulate these chemicals from the surrounding medium and from food. In aquatic organisms, uptake from the water is generally more important, whereas, in terrestrial fauna, food provides the major source.

In general, organisms at higher trophic levels tend to contain more DDT-type compounds than those at lower trophic levels.

Such compounds can be transported around the world in the bodies of migrant animals and in ocean and air currents.

1.3 Toxicity to Microorganisms

Aquatic microorganisms are more sensitive than terrestrial ones to DDT.

An environmental exposure concentration of 0.1 µg/litre can cause inhibition of growth and photosynthesis in green algae.

Repeated applications of DDT can lead to the development of tolerance in some microorganisms.

There is no information concerning the effects on species composition of microorganism communities. Therefore, it is difficult to extrapolate the relevance of single-culture studies to aquatic or terrestrial ecosystems. However, since microorganisms are basic in food chains, adverse effects on their populations would influence ecosystems. Thus, DDT and its metabolites should be regarded as a major environmental hazard.

1.4 Toxicity to Aquatic Invertebrates

Both the acute and long-term toxicities of DDT vary between species of aquatic invertebrates. Early developmental stages are more sensitive than adults to DDT. Long-term effects occur after exposure to concentrations ten to a hundred times lower than those causing short-term effects.

DDT is highly toxic, in acute exposure, to aquatic invertebrates at concentrations as low as 0.3 μg/litre. Toxic effects include impairment of reproduction and development, cardiovascular modifications, and neurological changes. Daphnia reproduction is adversely affected by DDT at 0.5 μg/litre.

The influence of environmental variables (such as temperature, water hardness, etc.) is documented but the mechanism is not fully understood. In contrast to the data on DDT, there is little information on the metabolites DDE or TDE. The reversibility of some effects, once exposure ceases, and the development of resistance have been reported.

1.5 Toxicity to Fish

DDT is highly toxic to fish; the 96-h LC_{50}s reported (static tests) range from 1.5 to 56 μg/litre (for largemouth bass and guppy, respectively). Smaller fish are more susceptible than larger ones of the same species. An increase in temperature decreases the toxicity of DDT to fish.

The behaviour of fish is influenced by DDT. Goldfish exposed to 1 μg/litre exhibit hyperactivity. Changes in the feeding of young fish are caused by DDT levels commonly found in nature, and effects on temperature preference have been reported.

Residue levels of > 2.4 mg/kg in eggs of the winter flounder result in abnormal embryos in the laboratory, and comparable residue levels have been found to relate to the death of lake trout fry in the wild.

Cellular respiration may be the main toxic target of DDT since there are reports of effects on ATPase.

The toxicity of TDE and DDE has been less studied than that of DDT. However, the data available on rainbow trout and bluegill sunfish show that TDE and DDE are both less toxic than DDT.

1.6 Toxicity to Amphibians

The toxicity of DDT and its metabolites to amphibians varies from species to species; although only a few data are available, amphibian larvae seem to be more sensitive than adults to DDT. TDE seems to be more toxic than DDT to amphibians, but there are no data available for DDE. All the studies reported have been static tests and, therefore, results should be treated with caution.

1.7 Toxicity to Terrestrial Invertebrates

There have been few reports on the effects of DDT and its metabolites on non-target terrestrial invertebrates.

Earthworms are insensitive to the acutely toxic effects of these compounds at levels higher than those likely to be found in the environment. The uptake of DDT by earthworms is related to the concentrations in soil and to the activity of the worms; seasonally greater activity increases uptake. Thus, although earthworms are unlikely to be seriously affected by DDT, they pose a major hazard to predators because of the residues they can tolerate.

Both DDT and DDE are classified as being relatively non-toxic to honey bees, with a topical LD_{50} of 27 μg/bee.

There are no reports on laboratory studies using DDE or TDE, in spite of the fact that these are major contaminants of soil.

1.8 Toxicity to Birds

DDT and its metabolites can lower the reproductive rate of birds by causing eggshell thinning (which leads to egg breakage) and by causing embryo deaths. However, different groups of birds vary greatly in their sensitivity to these chemicals; predatory birds are extremely sensitive and, in the wild, often show marked shell thinning, whilst gallinaceous birds are relatively insensitive. Because of the difficulties of breeding birds of prey in captivity, most of the experimental work has been done with insensitive species, which have often shown little or no shell thinning. The few studies on more sensitive species have shown shell thinning at levels similar to those found in the wild. The lowest dietary concentration of DDT reported to cause shell thinning experimentally was 0.6 mg/kg for the black duck. The mechanism of shell thinning is not fully understood.

1.9 Toxicity to non-laboratory Mammals

Experimental work suggests that some species, notably bats, may have been affected by DDT and its metabolites. Species which show marked seasonal cycles in fat content are most vulnerable, but few experimental studies on such species have been made. In contrast to the situation in birds, where the main effect of DDT is on reproduction, the main known effect in mammals is to increase the mortality of migrating adults. The lowest acute dose which kills American big brown bats is 20 mg/kg. Bats collected from the wild (and containing residues of DDE in fat) die after experimental starvation, which simulates loss of fat during migration.

2. PHYSICAL AND CHEMICAL PROPERTIES OF DDT AND RELATED COMPOUNDS

The term DDT is generally understood throughout the world and refers to *p,p'*-DDT (1,1'-[2,2,2-trichloroethylidine]-bis [4-chlorobenzene]). The compound's structure permits several different isomeric forms, such as *o,p'*-DDT (1-chloro-2-[2,2,2-trichloro-1-(4-chlorophenyl) ethyl] benzene). The term DDT is also applied to commercial products consisting predominantly of *p,p'*-DDT with smaller amounts of other compounds. A typical example of technical DDT had the following constituents: *p,p'*-DDT, 77.1%; *o,p'*-DDT, 14.9%; *p,p'*-TDE, 0.3%; *o,p'*-TDE, 0.1%; *p,p'*-DDE, 4%; *o,p'*-DDE, 0.1%; and unidentified products, 3.5%.

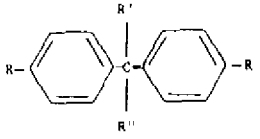
All isomers of the compound DDT are white, crystalline, tasteless, almost odourless solids, with the empirical formula $C_{14}H_9Cl_5$ and a relative molecular mass of 354.5. The melting range of *p,p'*-DDT is 108.5 to 109 °C and its vapour pressure is 2.53×10^{-5} Pa (1.9×10^{-7} mmHg) at 20 °C. DDT is soluble in organic solvents as follows (g/100 ml): benzene, 106; cyclohexanone, 100; chloroform, 96; petroleum solvents, 4-10; ethanol, 1.5. It is highly insoluble in water (solubility approximately 1 µg/litre) but very soluble in animal fats. The octanol-water partition coefficient (log k_{ow}) is 7.48

The chemical structure of some of the analogues of DDT is shown in Table 1. The structure of the *o,p'*- and *m,p'*-compounds can be inferred from those of the *p,p'*-isomers presented in the table. The table is confined to compounds that occur in commercial DDT, metabolites formed from them, and analogues that have had some use as insecticides. It must be emphasized that even the commercially-available insecticidal analogues have strikingly different properties. Especially remarkable is the slow metabolism and marked storage of DDT and its metabolite DDE and the rapid metabolism and negligible storage of methoxychlor.

Technical DDT has been formulated in almost every conceivable form including solutions in xylene or petroleum distillates, emulsifiable concentrates, water-wettable powders, granules, aerosols, smoke candles, charges for vaporizers and lotions. Aerosols and other household formulations are often combined with synergized pyrethroids.

This is a summary of part of the relevant section from Environmental Health Criteria 9: DDT and its Derivatives (WHO, 1979). Further details, including information on analysis, sources of pollution, and environmental distribution can be found in this document.

Table 1. Structure of *p,p'*-DDT and its analogues of the form:



(many of the compounds also exist as *o,p'*-isomers and other isomers)

Name DDT and its major metabolites	Chemical name	R	R'	R''
DDT	1,1'-(2,2,2-trichloroethylidene)-bis[4-chlorobenzene]	–Cl	–H	$-CCl_3$
DDE[a]	1,1'-(2,2-dichloroethenylidene)-bis[4-chlorobenzene]	–Cl	None	$=CCl_2$
TDE(DDD)[a,b]	1,1'-(2,2-dichloroethylidene)-bis[4-chlorobenzene]	–Cl	–H	$-CHCl_2$
DDMU[a]	1,1'-(2-chloroethenyldene)-bis[4-chlorobenzene]-	–Cl	None	=CHCl
DDMS[a]	1,1'-(2-chloroethylidene)-bis[4-chlorobenzene]	–Cl	–H	$-CH_2Cl$
DDNU[a]	1,1'-bis(4-chlorophenyl)ethylene	–Cl	None	$=CH_2$
DDOH[a]	2,2-bis(4-chlorophenyl)ethanol	–Cl	–H	$-CH_2OH$
DDA[a]	2,2-bis(4-chlorophenyl)-acetic acid	–Cl	–H	–C(O)OH
Some related insecticides				
Bulan®	2-nitro-1,1-bis-(4-chlorophenyl)butane	–Cl	–H	$-CHC_2H_5$ (NO$_2$)
Prolan®	2-nitro-1,1-bis-(4-chlorophenyl)propane	–Cl	–H	$-CHCH_2$ (NO$_2$)
DMC	4-chloro-α-(4-chlorophenyl)-α-(methyl)benzenemethanol	–Cl	–OH	$-CH_3$
dicocol (Kelthane®)	4-chloro-α-(4-chlorophenyl)-α-(trichloromethyl)benzenemethanol	–Cl	–OH	$-CCl_3$
chlorobenzilate[c]	ethyl 4-chloro-α-(4-chlorophenyl)-α-hydroxybenzeneacetate	–Cl	–OH	$-C(O)OC_2H_5$
chloropropopylate[c]	1-methylethyl 4-chloro-α-(4-chlorophenyl)-α-hydroxy-benzeneacetate	–Cl	–OH	$-C(O)OCH(CH_3)_2$
methoxychlor[c]	1,1'-(2,2,2-trichloroethylidene)-bis[4-methoxybenzene]	$-OCH_3$	–H	$-CCl_3$
Perthane®	1,1'-(2,2-dichloroethylidene)-bis[4-ethylbenzene]	$-C_2H_5$	–H	$-CHCl_2$
DFDT	1,1'-(2,2,2-trichloroethylidene)-bis[4-fluorobenzene]	–F	–H	$-CCl_3$

[a] Recognized metabolite of DDT in the rat.

[b] As an insecticide, this compound has the ISO approved name of TDE, and it has been sold under the name Rothane®; in metabolic studies the same compound has been referred to as DDD; as a drug, it is called mitotane.

[c] Common name approved by the International Organization for Standardization (ISO).

3. KINETICS, METABOLISM, BIOTRANSFORMATION, AND BIOACCUMULATION

Appraisal

The physicochemical properties of DDT and its metabolites enable these compounds to be taken up readily by organisms. The rates of accumulation vary with the species, with the duration and concentration of exposure, and with environmental conditions.

These compounds are resistant to breakdown and are readily adsorbed to sediments and soils, which can act both as sinks and as long-term sources of exposure (e.g., for soil organisms).

Organisms can accumulate these chemicals from the surrounding medium and from food. In aquatic organisms, uptake from the water is generally more important, whereas, in terrestrial fauna, food provides the major source.

In general, organisms at higher trophic levels tend to contain more DDT-type compounds than those at lower trophic levels.

Such compounds can be transported around the world in the bodies of migrant animals and in ocean and air currents.

Different organisms metabolise DDT via different pathways. Of the two initial metabolites, DDE is the more persistent, though not all organisms produce DDE from DDT. The alternative route of metabolism, via TDE leads to more rapid elimination (WHO, 1979). Much of the retained DDT and its metabolites are stored in lipid-rich tissues. Because there is an annual cycle in lipid storage and utilization in many organisms, there is also a related annual cyclic pattern in the handling of DDT.

3.1 Retention in Soils and Sediments and Plant Uptake

Shin et al. (1970) investigated the adsorption of DDT by soils of various different types and by isolated soil fractions. A sandy loam, a clay soil, and a highly organic muck were either used intact or had various components extracted before estimating their adsorptive capacity for the insecticide. Adsorption was least in the sandy loam and greatest in the muck (distribution coefficients [Kd] were in the ratio 1:10:80 for sandy loam, clay soil, and organic muck, respectively). All soils showed a strong adsorptive capacity for DDT. The adsorption of DDT was closely related to the organic matter content of the soils; progressive removal of lipids, resins, polysaccharides, polyuronides, and humic matter identified the organic fractions which bound the DDT. Humic material represents a major source of adsorptive capacity for DDT; the degree of sorption, however, is strongly connected with the degree of humification. Soil containing large amounts of humic material may not adsorb DDT as greatly as other soils where humification is more advanced. Wheatley (1965) estimated half-

times for the loss of DDT applied to soils. After surface application, 50% of DDT was lost within 16-20 days. The estimated time for the loss of 90% of surface-applied DDT was 1.5 to 2 years. With DDT mixed into the soil, 50% loss occurred in 5 to 8 years, and it was estimated that 90% of applied insecticide would be lost in 25-40 years.

Albone et al. (1972) investigated the capacity of river sediments, from the Severn Estuary, United Kingdom, to degrade DDT. *p,p'*-DDT (^{14}C-labelled) was applied to sediments either *in situ* on the mud flats or in the laboratory. Sediment movement in the area of the *in situ* study was sufficiently small to neither bury nor expose the incubation tubes set into the mud. Incubation *in situ* over 46 days led to very little metabolism of DDT in the sediments. Some *p,p'*-TDE was produced, but the ratio of DDT to TDE was 13 : 1 and 48 : 1 in two replicate experiments. There was no production of extractable polar products; metabolism beyond TDE did not occur. Incubation of the same sediments in the laboratory, over 21 days, led to much greater metabolism (ratios of 1 : 1.1 and 1 : 3.3, DDT to TDE, in replicate incubations) and the production of some unidentified, further breakdown products. Investigation of the microbial population of the sediment showed that some of the organisms were capable of degrading DDT; little metabolism appeared to take place *in situ*.

3.2 Uptake and Accumulation by Organisms

The uptake and accumulation of DDT and its metabolites into organisms, as determined in controlled laboratory experiments, is summarized in Table 2. Results are expressed as bioconcentration factors (the ratio of the concentration of the compound in the organism to the concentration in the medium).

Concentration factors can be misleading with compounds such as DDT when exposure is high. The compound is readily taken up and retained at very low concentrations. At high concentrations, no more material can be taken up because a plateau has been reached. The only meaningful way to assess the capacity of organisms to take up and retain DDT is by looking over a wide range of exposure levels. The low concentration factor quoted in Table 2 for earthworms, for example, reflects the high exposure rather than a low capacity for uptake and retention of DDT, because concentration factors are simple ratios between "exposure" and final concentration in the organism.

Concentration factors for fish are generally higher than for their invertebrate prey (Table 2). It is now generally agreed that most of the DDT taken into aquatic organisms comes from the water rather than from their food (Moriarty, 1975). Again, the concentration factors can be misleading. Aquatic organisms take in a small proportion of ingested DDT. However, they retain a large proportion of the DDT which has been absorbed into the body from the food. There has been some controversy in the past over explanations for higher accumulations of DDT at higher trophic levels in aquatic systems. It now seems clear that this is not due primarily to biomagnification up food chains but

Table 2. Bioaccumulation of DDT[a]

Organism	Biomass (µg/ml)	Flow stat[b]	Organ	Temperature (°C)	Duration	Exposure (µg/litre)	Bioconcentration factor[c]	Reference
Bacteria								
Aerobacter aerogenes	100			22	24 h	1.2	3736	Johnson & Kennedy (1973)
Bacillus subtilis	130			22	24 h	0.676	4303	Johnson & Keneddy (1973)
Aerobacter aerogenes	25			22	4 h	0.64	10 639	Johnson & Keneddy (1973)
	200			22	4 h	0.64	1784	Johnson & Keneddy (1973)
Bacillus subtilis	43			22	4 h	0.64	13 880	Johnson & Keneddy (1973)
	348			22	4 h	0.64	1805	Johnson & Keneddy (1973)
Marine algae								
Cyclotella nana	17			23	2 h	0.7	37 600	Rice & Sikka (1973)
	8			23	2 h	0.7	58 100	Rice & Sikka (1973)
Isochrysis galbane	39			23	2 h	0.7	11 300	Rice & Sikka (1973)
	19			23	2 h	0.7	28 800	Rice & Sikka (1973)
Olisthodiscus luteus	108			23	2 h	0.7	4600	Rice & Sikka (1973)
	54			23	2 h	0.7	7000	Rice & Sikka (1973)
Amphidinium carteri	66			23	2 h	0.7	4300	Rice & Sikka (1973)
	33			23	2 h	0.7	9600	Rice & Sikka (1973)
Tetraselmis chuii	106			23	2 h	0.7	5200	Rice & Sikka (1973)
	53			23	2 h	0.7	6300	Rice & Sikka (1973)
Skeletonema costatum	29			23	2 h	0.7	31 900	Rice & Sikka (1973)
	15			23	2 h	0.7	38 400	Rice & Sikka (1973)
Diatom								
Cylindrotheca closterium					21 days	100	300	Keil & Priester (1969)

Table 2. (Contd).

Pond snail *(Physa 5 sp.)*	stat		6 days	3.0	6000	Reinbold et al. (1971)
Freshwater mussel *(Anodonta grandis)*	flow	20	3 weeks	0.62	3990[d]	Bedford & Zabik (1973)
Earthworm *(Lumbricus terrestris)*		10	4 weeks	17 000	0.47[d]	Davis (1971)
Water flea	stat	30	3 days	2.0	1330	Metcalf et al. (1973)
(Daphnia magna)	flow	21	3 days	0.08	114 100	Johnson et al. (1971)
Scud *(Gammarus fasciatus)*	flow	21	3 days	0.081	20 600	Johnson et al. (1971)
Glass shrimp *(Palaemonetes kadiakensis)*	flow	21	3 days	0.1	5000	Johnson et al. (1974)
Pink shrimp *(Penaeus duorarum)*	flow	8-15	13 days	0.14	1500	Nimmo et al. (1970)
Crayfish *(Orconectes nais)*	flow	21	3 days	0.08	2900	Johnson et al. (1971)
Mayfly larva *(Hexagenia bilineata)*	flow	21	3 days	0.052	32 600	Johnson et al. (1971)
Mayfly larva *(Siphlonurus sp.)*	flow	21	3 days	0.047	22 900	Johnson et al. (1971)

Table 2. (Contd).

Organism	Biomass (µg/ml)	Flow stat[b]	Organ	Temperature (°C)	Duration	Exposure (µg/litre)	Bioconcentration factor[c]	Reference
Dragonfly nymph *(Ischnura verticalis)*		flow		21	2 days	0.101	3500	Johnson et al. (1971)
Dragonfly nymph *(Libellula sp.)*		flow		21	2 days	0.079	910	Johnson et al. (1971)
Midge larva *(Chironomus sp.)*		flow		21	3 days	0.046	47 800	Johnson et al. (1971)
Mosquito larva *(Culex pipiens)*		flow		21	2 days	0.105	133 600	Johnson et al. (1971)
Mosquito larva *(Culex quinquifasciatus)*		stat		30	3 days	2.0	110[d]	Metcalf et al. (1973)
		stat		30	3 days	0.9	74[d]	Metcalf et al. (1973)
Mosquito fish *(Gambusia affinis)*		stat		30	3 days	2.0	344[d]	Metcalf et al. (1973)
		stat		30	3 days	0.9	217[d]	Metcalf et al. (1973)
Rainbow trout *(Salmo gairdneri)*		flow		5	12 weeks	0.176	21 363[d]	Reinert et al. (1974)
		flow		10	12 weeks	0.137	43 158[d]	Reinert et al. (1974)
		flow		15	12 weeks	0.133	51 355[d]	Reinert et al. (1974)
Brook trout *(Salvelinus fontinalis)*		flow		14	120 days	3 mg /kg diet	0.64[d]	Macek & Korn (1970)
		flow		14	120 days	0.003	8533[d]	Macek & Korn (1970)
Pinfish *(Lagodon rhomboides)*		flow			14 days	0.1	40 000[d]	Hansen & Wilson (1970)
		flow			14 days	1.0	11 020[d]	Hansen & Wilson (1970)

Table 2. (Contd).

Atlantic croaker	flow			14 days	0.1	12 500[d]	Hansen & Wilson (1970)
(Micropogon undulatus)	flow			14 days	1.0	12 170[d]	Hansen & Wilson (1970)
Fathead minnow	flow		24-25.5	14 days	45.6 mg/kg	1.17[d]	Jarvinen et al. (1977)
(Pimephales promelas)	flow		24-25.5	14 days	0.5	85 400[d]	Jarvinen et al. (1977)
	flow		24-25.5	14 days	2.0	69 100[d]	Jarvinen et al. (1977)
	flow		24-25.5	112 days	45.6 mg/kg	1.33[d]	Jarvinen et al. (1977)
	flow		24-25.5	112 days	0.5	93 200[d]	Jarvinen et al. (1977)
	flow		24-25.5	112 days	2.0	154 100[d]	Jarvinen et al. (1977)
Tilapia	stat			31 days	1.0	6800	Reinbold et al. (1971)
(Tilapia mossambica)				31 days	10	10 600	Reinbold et al. (1971)
Green sunfish	stat			31 days	1.0	3900	Reinbold et al. (1971)
(Lepomis cyanellus)				31 days	10	4020	Reinbold et al. (1971)
	stat		22	15 days	0.1-0.3	17 500[d]	Sanborn et al. (1975)
Chicken		eggs		8 weeks	0.1	1.87[d]	Foster et al. (1972)
		fat		8 weeks	0.1	5.8[d]	Foster et al. (1972)
Broiler hen		fat		6 weeks	1.0	10.3[d]	Kan et al. (1978)

Table 2. (Contd).

Organism	Biomass (µg/ml)	Flow stat[b]	Organ	Temperature (°C)	Duration	Exposure (µg/litre)	Bioconcentration factor[c]	Reference
White pelican *(Pelecanus erythrorhynchos)*			WB		10 weeks	72	11.9[d]	Greichus et al. (1975)
Double-crested cormorant *(Phalacrocorax a. auritus)*			WB		9 weeks	0.95	236.3[d]	Greichus & Hannon (1973)
American kestrel *(Falco sparverius)*			WB		11-16 months	2.8	103.9	Porter & Wiemeyer (1972)
Mule deer[e] *(Odocoileus heminonus)*			muscle		30 days oral	5 mg/day	122.8 µg /kg[d] residue	Watson et al. (1975)

a Unless specified otherwise, bioconcentration factors are based on whole body (WB) measurements.
b Stat = static conditions (water unchanged for duration of experiment); Flow = flow-through conditions (DDT concentration in water continuously maintained).
c Bioconcentration factor = concentration of DDT in organism/concentration of DDT in medium or food. Concentrations of DDT in organisms represents total DDT, i.e., DDT plus its stable metabolites, principally DDE. Bioconcentration factors calculated on a dry weight basis unless otherwise stated.
d Calculated on a wet weight basis.
e Oral dose (by capsule) given daily.

rather to a tendency for organisms at higher trophic levels to accumulate more DDT directly from the water.

Terrestrial organisms do not live in a uniform medium surrounded by a relatively constant concentration of a chemical. Even soil organisms live in a medium with very variable concentrations of DDT or its metabolites at different levels of the soil profile or patchy distribution of the chemical. Some terrestrial organisms could be directly exposed to DDT during application of the insecticide, but most will be exposed to what remains of the DDT after application. Therefore, higher terrestrial organisms will accumulate DDT mostly from their food. The data in Table 2 are taken from controlled laboratory investigations. There is ample evidence from the field that DDT does accumulate in many organisms in different media. There is similarly evidence that the residues of DDT or its metabolites persist in organisms for long periods after exposure has ceased. The following should not be regarded as a comprehensive review of the literature on this subject, which is too large to be included. Rather, these are examples from different groups of organisms.

3.2.1 Plants

Fuhremann & Lichtenstein (1980) applied ^{14}C-labelled *p,p'*-DDT to loam or sandy soil (at 4 and 2 mg/kg, respectively) and grew oat plants on the treated soils for 13 days. At harvest, residues of DDT and its metabolites were analysed in soil and plant by scintillation counting, thin layer chromatography, and GLC. Of the total applied DDT, 95.7% was recovered from loam soil and 88.6% from sandy soil. Almost all of the DDT present was extractable in organic solvent (only 2.8%, for loam, and 0.7%, for sand, was present in a water-bound form), indicating little or no metabolism of the compound except to persistent organically extractable residues. DDE was detected in both soils, accounting for 3.4% of the total extracted in loam soil and 2.2% in sand. Other metabolites, including *o,p'*-DDT, TDE, and dicofol were recovered in very small quantities. Very little DDT (and none of its metabolites) was detected in oat roots grown on loam, amounting to 0.2% of the total DDT applied. The uptake was greater (4.6%) in roots of oats grown on sand, but the uptake of labelled carbon into plant tops, from both soils, was so low that it could not be analysed.

DDT was not translocated into the foliage of alfalfa when applied to the soil (Ware, 1968; Ware et al., 1970) or into soybeans (Eden & Arthur, 1965). Harris & Sans (1967) found only trace amounts of DDT or its metabolites in the storage roots of carrots, radishes, and turnips after growing the plants in soils containing up to 14 mg DDT/kg.

3.2.2 Microorganisms

The uptake and accumulation of DDT from the culture medium by microorganisms has been reviewed by Lal & Saxena (1982). All of the

microorganisms studied showed some capacity to take up DDT from their growth medium, but the relative amount taken up varied greatly from species to species. Many species took up more than 90% of the DDT when exposed to concentrations ranging from 1 to 1000 μg/litre, whereas a few species took in only 0.5% of the available DDT. The concentration factors (i.e., the concentration within the organism expressed as a ratio against the concentration in the medium) for DDT were variable but always high (Table 2).

3.2.3 Aquatic invertebrates

Concentration factors are also variable in aquatic invertebrates. In all cases there is considerable uptake and retention of the DDT, though often as DDE or other metabolites rather than as the parent compound. The main point of interest is the ability of aquatic organisms to take up large amounts of the compound, over time, from water where DDT is present at very low concentrations, and to retain it.

Risebrough et al. (1976) measured DDT in sea water and in mussels (*Mytilus* sp.) from San Fransisco Bay and the French Mediterranean coast. Concentration factors varied between 40 000 and 690 000 for DDT and between 45 000 and 310 000 for DDE.

Eberhardt et al. (1971) applied radioactively labelled DDT, at a rate of 220 g/ha, to a freshwater marsh and followed the distribution of the compound and its metabolites. Concentration factors in ten species of plants varied between 5500 and 84 000. Various invertebrates showed high concentration factors: ramshorn snail (*Planorbidae*), 4700; backswimmer (*Notonectidae*), 10 000; crayfish (*Orconectes immunis*), 22 000; bloodworm (*Tendipes*), 25 000; and red leech (*Erpobdella punctata*), 47 000. Reporting earlier on the same study, over 15 months, Meeks (1968) showed that plants and invertebrates accumulated DDT to a maximum mainly within the first week after treatment, whereas vertebrates required longer to attain maximum residues. Residues of DDT in the surface water and suspended particles had fallen below detectable levels within 1 month. Residues in sediments stabilized at about 0.3 mg/kg after 9 months.

3.2.4 Fish

The uptake of DDT from water is affected by the size of the fish; smaller fish take up relatively more DDT from water than larger specimens of the same species. A range in weight of mosquitofish between 70 and 1000 mg led to a four-fold difference between the smallest and largest fish in DDT uptake from water over 48 h (Murphy, 1971).

A rise in temperature results in increased uptake of DDT by fish (Reinert et al., 1974). Rainbow trout were exposed to a single water concentration of DDT (nominally 330 ng/litre) at temperatures of 5, 10, or 15 °C; the actual concentrations of DDT in water varied with

temperature and were measured at 176, 137, and 133 ng/litre, respectively, for 5, 10, and 15 °C. Whole body residues of DDT (total) after 12 weeks exposure were 3.8, 5.9, and 6.8 mg/kg for the three temperatures, respectively. Expressing the results as bioconcentration factors to allow for the differences in dissolved DDT showed a similar, clear increase in the relative amount of DDT taken up and retained (Reinert et al., 1974).

Increasing salinity decreases DDT uptake significantly, but has no effect on the uptake of DDE or TDE by fish (Murphy, 1970). Increasing the salinity from 0.15‰ to 10‰ decreased DDT uptake over 24 h from 22% of the dose to 18% (body residues decreased from 658 to 329 ng). There was a further significant decrease in uptake when the salinity was increased to 15‰ (Murphy, 1970)

Fish accumulate DDT from food in a dose-dependent manner. When Macek et al. (1970) fed rainbow trout on diets containing 0.2 or 1.0 mg DDT/kg, the fish retained more than 90% of the dietary intake of DDT (measured as total DDT) over the 90-day exposure period. The authors estimated the time required for the elimination of 50% of accumulated DDT to be 160 (± 18) days. When Warlen et al. (1977) fed Atlantic menhaden on a diet containing ^{14}C-labelled DDT at three dose levels, the fish assimilated and retained between 17% and 27% of the cumulative dose from food containing 0.58, 9.0, or 93 µg/kg. There was a straight-line relationship between exposure time and body burden of total DDT, with no tendency for residues to reach a plateau within the 45 days of feeding with DDT. At the end of the feeding period, the fish had accumulated DDT or its metabolites, to levels of approximately 1.1, 11, and 110 µg/kg for the three doses respectively. The biological half-time of DDT in the fish was estimated to be 428, 64, and 137 days, for groups exposed to 0.58, 9.0, or 93 µg/kg diet, respectively.

3.2.5 Terrestrial invertebrates

Relatively low concentration factors have been reported for terrestrial molluscs by Dindal & Wurtzinger (1971), who also reviewed the earlier literature. However, low concentration factors, derived from short-term studies, can be misleading for these organisms because of the high persistence of DDT in soil. Residues of DDT were as high as 40 mg/kg and, therefore, molluscs represent a source of DDT which will be concentrated by organisms which eat them. The same is true for earthworms, which also show low concentration factors (Davis, 1971; Edwards & Jeffs, 1974). Gish & Hughes (1982) investigated residues of DDT and other pesticides in earthworms for 2 years following application. They showed that body residue levels were cyclic, with higher levels of DDT and its metabolites occuring between late spring and early autumn and lower levels from late autumn to early spring. Peak high levels occurred in May and low levels in January, coinciding with the seasonal high and low activity periods of earthworms. These changing residue levels presumably indicate that DDT is retained in

soil and that earthworms contain more of the residual metabolites when they are processing more soil through the gut.

3.2.6 Birds

Laboratory studies on birds have shown them capable of accumulating DDT from food, yielding high concentration factors (Table 2).

The accumulation of DDT and its metabolites in birds in the field has been regularly and extensively reviewed (Moore, 1965; Moriarty, 1975; Newton, 1979). The results of an analysis of a long-term sampling programme of birds in the United Kingdom (Cooke et al., 1982) confirm many of the early theories. Birds with the highest residues of DDT or its metabolites were either terrestrial predators feeding on other birds or aquatic predators feeding on fish. Thus, residues of DDE in the liver of the peregrine falcon, with birds as its principal dietary component, averaged 7.56 mg/kg, whereas for the rough-legged buzzard, with mammals as the principal dietary component, mean DDE levels were 0.05 mg/kg over a period extending from the early 1960s to the late 1970s.

There are marked geographical differences throughout the United Kingdom, related to usage patterns of DDT (Cooke et al., 1982), and also marked seasonal changes in residues. These seasonal changes appear to relate more to physiological changes in body composition, which occur with climatic and breeding seasons, than to the environmental availability of pollutants. Some species, e.g., heron, barn owl, and kingfisher, showed a decline in DDE residues with time, but others, e.g., sparrowhawk, kestrel, and great-crested grebe, did not, levels in 1977 being similar to those in 1963. Eventually residues of DDT in wildlife decline with time after a ban is imposed on the use of the pesticide. However, the highly persistent nature of DDE means that significant residues will continue to be found for a considerable period. The situation in the United Kingdom and the USA appears to be broadly similar (O'Shea & Ludke, 1979).

3.2.7 Mammals

DDT is taken up by, and retained in, wild mammals. The degree of uptake and retention varies with the species. In a study following a single application of DDT to a forest to control spruce budworm at a rate of 0.89 kg/ha, Dimond & Sherburne (1969) and Sherburne & Dimond (1969) reported residues of DDT and its metabolites in mammals over 9 years. Herbivorous mice, voles, and hares contained less DDT than carnivorous mink and insectivorous shrews. In herbivores, residues approached pre-treatment levels after 6-7 years, whereas residues were still significantly higher in shrews and mink than in the same species taken from untreated areas 9 years after the single treatment with DDT. In these species, the authors calculate that it would take at least 15 years for residues to reach background levels. They regard the high residue levels in mammals at higher trophic levels as deriving

principally from DDT retained in the soil, since there is little long-term retention on vegetation.

In a 3-year study, after treating a field ecosystem with ^{36}Cl-ring-labelled DDT at a dose rate of 0.92 kg/ha, Forsyth & Peterle (1973) measured DDT residues in various tissues of two species of shrew. The highest residue (135 mg/kg) occurred in fat, compared with 10, 10, and 4 mg/kg in liver, muscle, and brain, respectively. Shrews of the species *Blarina brevicauda* released into treated areas accumulated DDT to the same degree as resident shrews within 15-20 days of exposure. Equilibrium between intake and excretion of DDT occurred within approximately 30 days in muscle, liver, and brain and within 40 days in fat. The second species of shrew *(Sorex cinereus)* accumulated residue levels of DDT during the following 2 years which were successively greater than levels present in the first year, indicating that DDT was increasing in availability to this species with the passage of time. The levels of DDT in muscle were not influenced by sex but were influenced by breeding condition. Male shrews with scrotal testes and lactating females developed lower levels of DDT in muscle and viscera than did males with abdominal testes or non-lactating females.

Benson & Smith (1972) measured levels of DDT and its metabolites in deer exposed to DDT used for spruce budworm control, and found that, in the year of spraying, there was up to 20 mg/kg in fat. Males had considerably higher levels of DDT than females. Fawns also had higher levels than their mothers, though this was from a small sample. The majority of the residues consisted of *p,p'*-DDT, with almost insignificant levels of DDE. Five years later, the residue levels in males were still higher than those in females, though these had fallen to about 1% of original levels. Most of the deer population was 3 years old or less, and so the figures for 5 years after spraying represent DDT ingested from the environment and not from direct exposure.

Some, though very little, DDT was detected in black bears by Benson et al. (1974). There was no evidence that the area had been directly sprayed with DDT. This study illustrates that there is a general environmental contamination with DDT, which can be accumulated by mammals, though to a small degree, without direct application of the material to their habitat.

4. TOXICITY TO MICROORGANISMS

Appraisal

Aquatic microorganisms are more sensitive than terrestrial ones to DDT.

An environmental exposure concentration of 0.1 µg/litre can cause inhibition of growth and photosynthesis in green algae.

Repeated applications of DDT can lead to tolerance in some microorganisms.

There is no information on effects concerning the species composition of microorganism communities. Therefore, it is difficult to extrapolate the relevance of single-culture studies to aquatic or terrestrial ecosystems. However, since microorganisms are basic in food chains, adverse effects on their populations would influence ecosystems. Thus, DDT and its metabolites should be regarded as a major environmental hazard.

Studies cited in this section will be restricted to those effects produced by low concentrations of DDT. Some studies still use DDT at concentrations above its water solubility. Reviews of other effects of DDT and its analogues, at higher concentrations, on cell division and several biochemical parameters have been produced by Luard (1973) and Lal & Saxena (1979).

4.1 Bacteria and Cyanobacteria (Blue-green Algae)

Ledford & Chen (1969) cultured bacteria isolated from surface-ripened cheese with 0.5 mg DDT/litre or 0.5 mg DDE/litre, but found no effect on growth.

At a concentration of 10 µg/litre in the culture medium, DDT stimulated the growth of the bacterium *Escherichia coli* (Keil et al., 1972). Yields of cultures exposed to 100 µg/litre did not differ from controls. There was no effect of DDT on denitrification (conversion of nitrate to nitrite) at a concentration of 100 mg/kg in soil and, similarly, no effect on this process when carried out by a bacterial culture (Bollag & Henninger, 1976). DDT at up to 22 kg/ha did not affect the numbers of soil bacteria in outdoor-treated plots (Bollen et al., 1954), and five annual applications of DDT to a sandy loam soil did not significantly affect the numbers of soil bacteria (Martin, 1966).

Concerning cyanobacteria (blue-green algae), Goulding & Ellis (1981) found no effect on the growth of *Anabaena variabilis* at a DDT concentration of 1 µg/litre. Batterton et al. (1972) suggested that DDT reduced the tolerance of *Anocystis nidulans* to sodium chloride. The organism is resistant to salt and to DDT, at concentrations up to 8000 mg/litre, but not to combinations of the two stressors.

4.2 Freshwater Microorganisms

Lee et al. (1976) showed that DDT inhibited photosynthesis in the green alga *Selenastrum capricornutum* at concentrations between 3.6 and 36 µg/litre, inhibition increasing with time of exposure.

Two different species of green algae were shown to be resistant to DDT and its metabolites, DDE and TDE, at concentrations up to 1000 mg/litre in culture. *Scenedesmus* and *Dunaliella* revealed rates of photosynthetic uptake of ^{14}C-labelled CO_2 similar to those of controls (Luard, 1973). Considerable variation exists between species of microorganisms concerning the effect of DDT and its analogues; resistance to DDT is not restricted to one taxonomic group, either freshwater or marine (Luard, 1973). The source of the resistance is unclear. The two species studied show very different characteristics; *Dunaliella* has no cell wall, whereas *Scenedesmus* has a complex cell wall. Since both show resistance to DDT, it is unlikely that the chemical is excluded from the cell by the cell wall. Cell membranes and chloroplast membranes are an alternative barrier to DDT uptake and effect. It is not known how these structures might be involved in DDT resistance; studies with isolated chloroplasts suggest that there is no barrier to DDT uptake there.

Cole & Plapp (1974) found inhibition of growth and photosynthesis of the green alga *Chlorella pyrenoidosa* with DDT at 1 µg/litre in the medium. However, inhibition was inversely related to the number of cells in the culture. With high cell counts, there was no inhibition of either growth or photosynthesis with DDT present at up to 1 mg/litre. Inhibition only occurred at low cell densities in culture.

Goulding & Ellis (1981) found that the green alga *Chlorella fusca* was affected by DDT at 0.1 µg/litre. The amount of inhibition of growth varied with time and with the method of assessing the result. Cell numbers were maximally affected (75% inhibition) after 72 hours, and after 200 hours cell numbers had reached control levels. When growth was assessed by chlorophyll content or biovolume, the initial inhibition was more marked and cultures were only equivalent to controls after 480 hours. The apparent anomaly is explained by reductions in cell size in response to DDT.

Christie (1969) reported no effect of DDT on the growth of *Chlorella* and attributed this to the ability of the organism to metabolize the compound.

Lal & Saxena (1980) reported that DDT did not affect growth and DNA synthesis in the ciliate *Stylonychia notophora* at concentrations of 1 mg/litre or less.

4.3 Marine Microorganisms

MacFarlane et al. (1972) showed that DDT, at concentrations between 9.4 and 1000 µg/litre, reduced photosynthetic carbon fixation and the cell content of chlorophyll *a* relative to controls in a marine diatom *Nitzschia delicatissima*, over a 24-h period.

The diatom was cultured with DDT under four different light intensities. The insecticide had the greatest effect at the highest light intensity, where carbon fixation was reduced by 94% in water containing 100 μg DDT/litre. At higher DDT concentrations, there was no further reduction in either carbon fixation or chlorophyll content.

The photosynthesis of several species of marine phytoplankton has been found to be inhibited by DDT at concentrations of 100 μg/litre or less (Wurster, 1968). Four different species showed increasing inhibition up to DDT concentrations of 100 μg/litre, but no greater effect at higher concentrations. A green alga, *Pyramimonas*, was affected by DDT only at concentrations higher than 10 μg/litre. The other three species, a diatom, a coccolithophore, and a dinoflagellate were affected at DDT concentrations between 1 and 10 μg/litre. In a similar study (Menzel et al., 1970) four different species of marine phytoplankton were studied. Inhibition of photosynthesis, where it occurred, followed a similar dose-response relationship. For three species (*Skeletonema costatum*, a diatom; *Coccolithus huxleyi*, a coccolithophorid; and *Cyclotella nana*, a second diatom) inhibition began between 1 and 10 μg DDT/litre and reached a maximum at 100 μg/litre. The other organism, a green flagellate *Dunaliella tertiolecta*, was unaffected by DDT at concentrations up to 1 mg/litre, the highest exposure tested.

The marine dinoflagellate *Exuviella baltica* showed significant inhibition of growth after exposure to DDT at concentrations as low as 0.1 μg/litre (Powers et al., 1979).

4.4 Soil Microorganisms

TDE had no significant effects on growth and reproduction of soil amoebae except at concentrations higher than 1 mg/litre (Prescott & Olson, 1972). Populations of protozoa in garden soil were reduced by DDT at a concentration of 5 mg/kg (MacRae & Vinckx, 1973). Numbers were still significantly reduced after 3 months.

4.5 Fungi

Two aquatic and one terrestrial fungi showed stimulated growth in response to DDT present at concentrations of between 2 and 60 μg/litre of growth medium (Hodkinson & Dalton, 1973)

5. TOXICITY TO AQUATIC ORGANISMS

DDT and its derivatives are highly toxic to aquatic organisms; water concentrations of a few micrograms per litre are sufficient to kill a large proportion of populations of aquatic organisms in acute or short-term exposure. In addition to its high short-term toxicity, DDT also has long-term sublethal effects on aquatic organisms. Many physiological and behavioural parameters have been reported to be affected by the insecticide. This toxicity, coupled with its high capacity for bioconcentration and biomagnification, means that DDT presents a major hazard to aquatic organisms.

5.1 Aquatic Invertebrates

Appraisal

Both the acute and long-term toxicities of DDT vary between species of aquatic invertebrates. Early developmental stages are more sensitive than adults to DDT. Long-term effects occur after exposure to concentrations ten to a hundred times lower than those causing short-term effects.

DDT is highly toxic, in acute exposure, to aquatic invertebrate, at concentrations as low as 0.3 µg/litre. Toxic effects include impairment of reproduction and development, cardiovascular modifications, and neurological changes. Daphnia reproduction is adversely affected by DDT at 0.5 µg DDT/litre.

The influence of environmental variables (such as temperature, water hardness, etc.) is documented but the mechanism is not fully understood. In contrast to the data on DDT, there is less information on the metabolites DDE or TDE. The reversibility of some effects once exposure ceases has been reported, as well as the development of resistance.

5.1.1 Short-term and long-term toxicity

The short-term toxicity to aquatic invertebrates is summarized in Table 3.

Most aquatic invertebrates are killed by low water concentrations of DDT and its metabolites, though the majority of the published data is on DDT itself. Six invertebrate species studied by Macek & Sanders (1970) showed 96-h LC_{50} values ranging from 1.8 to 54.0 µg/ litre. Adult molluscs are relatively resistant to DDT and the compound has been used to control crustacean pests on oyster beds (Loosanoff, 1959). However, the larval stages of molluscs are affected by DDT; clam larvae showed 90% mortality after exposure to DDT at 0.05 mg/litre (Calabrese, 1972). Molluscs exhibit effects on shell growth at low DDT concentrations. Tubifex worms are resistant to DDT; 3 mg/litre did not kill any *Tubifex tubifex* (Naqvi & Ferguson, 1968). Many aquatic crustaceans

yield LC_{50} values less than 1 µg/litre. Muirhead-Thomson (1973) showed that predator invertebrates, such as dragonfly nymphs, were more tolerant of DDT than prey organisms. Since the prey organisms are also food for fish, the balance of aquatic ecosystems could be changed by very low levels of DDT. Lowe (1965) reported that juvenile blue crabs *(Callinectes sapidus)*, exposed to 0.25 µg DDT/litre for 9 months, grew and moulted normally; there were no apparent sublethal effects. However, exposure to 5 µg DDT/litre killed all crabs.

The metabolite TDE has been studied in parallel tests with the parent compound in some organisms. There is no consistent relationship between the toxicity of the two compounds. TDE is considerably less toxic to stonefly larvae than DDT, by a factor of about 100 (Sanders & Cope, 1968). However, for other freshwater organisms TDE may have similar, lower, or greater toxicity according to the organism and duration of test (Table 3). For most marine invertebrates, DDT is most toxic, followed by DDE and TDE (data from Mayer, 1987).

5.1.2 Physiological effects on aquatic invertebrates

Butler (1964) demonstrated a 50% reduction in shell growth in young eastern oysters exposed for 96-h to DDT at 14 µg/litre. Roberts (1975) showed that DDT at 50 µg/litre reduced the amplitude of ventricular contractions in the isolated heart of the bivalve *Mya arenaria* within 4 minutes. At higher concentrations, DDT stopped heart contractions altogether. Recovery, even of the arrested heart, was rapid after the immediate replacement of the DDT solution with clean sea water.

Kouyoumjian & Uglow (1974) found that for the planarian worm *Polycelis felina*, TDE was most toxic and DDT least toxic, with DDE showing intermediate toxicity. Sublethal effects of DDT and TDE were demonstrated. DDT reduced the rate of asexual fission. Both DDT and TDE were shown to reduce the righting time of animals turned onto their backs. This was presumed to be a nervous system effect.

Maki & Johnson (1975) report 50% reduction in three parameters of reproduction in the water flea *Daphnia magna* at 0.5 µg/litre, for total young produced, at 0.61 µg/litre for average brood size, and at 0.75 µg/litre for percentage of days reproducing.

In vitro effects on gill ATPases of two species of crab have been reported (Jowett et al., 1978; Neufeld & Pritchard, 1979). There is a transitory effect in vivo on gill ATPases and, thereby, an effect on plasma osmolarity. However, this osmoregulatory effect soon disappears (Pritchard & Neufeld, 1979). Leffler (1975) reported metabolic rate elevation, decreased muscular coordination, inhibition of autotomy reflex, and reduced carapace thickness/width ratio in juvenile crabs exposed to DDT. Osmoregulation was not affected. The DDT was given in the food of the crabs at a concentration of 0.8 mg/kg. DDT has been found to accelerate limb regeneration and the onset of the next moult in fiddler crabs (Weis & Mantel, 1976). The authors suggest that the effect is on the central nervous system, with DDT causing changes in neurosecretory activity.

Table 3. Toxicity of DDT and its derivatives to invertebrates

Organism[f]	Flow stat[a]	Temp (°C)	Salinity °/oo	Compound	Parameter	Water concentration (µg/litre)	Reference
Estuarine and marine invertebrates							
Eastern oyster (juv.)	flow	30	23	DDT[d]	96-h EC_{50}[j]	9	Mayer (1987)
(Crassostrea virginica)	flow	12	25	DDE[d]	96-h EC_{50}[j]	14	Mayer (1987)
	flow	20	30	TDE[d]	96-h EC_{50}[j]	25	Mayer (1987)
Shrimp	stat	20	sea water	DDT[d]	96-h LC_{50}	0.4	McLeese & Metcalfe
(Crangon septemspinosa)	stat	10	sea water + sediment	DDT[d]	96-h LC_{50}	31	(1980)
Mysid shrimp (adult) *(Mysidopsis bahia)*	stat	25	23	DDT[d]	96-h LC_{50}	0.45 (0.39-0.52)	Mayer (1987)
Pink shrimp (juv.)	flow	24	28	DDT[d]	48-h LC_{50}	0.6	Mayer (1987)
(Penaeus duorarum)	flow	16	31	TDE[d]	48-h LC_{50}	2.4	Mayer (1987)
White shrimp (juv.) *(Penaeus setiferus)*	flow	27	28	DDT[d]	24-h LC_{50}	0.7	Mayer (1987)
Grass shrimp (juv.) *(Palaemonetes pugio)*	flow	27	28	DDT[d]	24-h LC_{50}	0.8	Mayer (1987)
Brown shrimp (juv.)	flow	28	17-27	DDE[d]	24-h LC_{50}	52	Butler (1964)
(Penaeus aztecus)	flow	28	17-27	DDE[d]	48-h LC_{50}	28	Butler (1964)

Table 3. (Contd).

Organism	Flow Stat[a]	Temp (°C)	Alkalinity[c]	Hardness[c]	pH	Compound	Parameter	Water concentration (µg/litre)	Reference
Freshwater invertebrates									
Water flea	stat	20	192	138	8.2-8.5	DDT[d]	48-h LC_{50}	1.1 (1.0-1.3)	Randall et al. (1979)
(*Daphnia magna*)	stat	15		44	7.1	DDT[d]	48-h LC_{50}	4.7 (2.8-5.6)	Mayer & Ellersieck (1986)
	stat	20	192	138	8.2-8.5	DDT[e]	48-h LC_{50}	1.7 (1.5-1.8)	Randall et al. (1979)
	stat[b]	24		320-340	7.6	DDT (99%)	14-day LC_{50}	0.67 (0.65-0.69)	Maki & Johnson
	stat[b]	24		320-340	7.6	DDT (99%)	14-day EC_{50}[g]	0.5 (0.48-0.52)	(1975)
	stat[b]	24		320-340	7.6	DDT (99%)	14-day EC_{50}[h]	0.61 (0.58-0.64)	Maki & Johnson
	stat[b]	24		320-340	7.6	DDT (99%)	14-day EC_{50}[i]	0.75 (0.71-0.79)	(1975)
	stat	10		44	7.1	TDE[d]	48-h LC_{50}	9.1	Mayer & Ellersieck (1986)
	stat	21		44	7.1	TDE[d]	48-h LC_{50}	8.9	
reared in soft water ($CaCO_3$: 50 mg/litre)	stat	20.5		250	7.8-8.2	DDT	24-h LC_{50}	510 (230-1120)	Berglind & Dave (1984)
	stat	20.5		250	7.8-8.2	DDT	48-h LC_{50}	1.1 (0.89-1.7)	
	stat	20.5		250	8.4-8.5	DDT	24-h LC_{50}	98 (75-127)	Berglind & Dave (1984)
	stat	20.5		250	8.4-8.5	DDT	48-h LC_{50}	1.3 (1.1-1.5)	
reared in hard water ($CaCO_3$: 300 mg/litre)	stat	20.5		250	7.8-8.2	DDT	24-h LC_{50}	71 (41-130)	Berglind & Dave (1984)
	stat	20.5		250	7.8-8.2	DDT	48-h LC_{50}	0.68 (0.46-1.0)	
	stat	20.5		250	8.4-8.5	DDT	24-h LC_{50}	42 (32-56)	Berglind & Dave (1984)
	stat	20.5		250	8.4-8.5	DDT	48-h LC_{50}	0.5 (0.41-0.61)	
	stat	20.5		50	7.8-8.2	DDT	24-h LC_{50}	0.99 (0.66-1.49)	
Water flea (*Daphnia pulex*)	stat	15		44	7.1	DDT[d]	48-h LC_{50}	0.36 (0.28-0.47)	Mayer & Ellersieck (1986)

Table 3. (Contd).

Water flea (*Simocephalus serrulatus*)	stat	15		44	7.1	DDT[d]	48-h LC_{50}	2.5 (1.9-3.3)	Mayer & Ellersieck (1986)
	stat	21		44	7.1	DDT[d]	48-h LC_{50}	2.8 (2.3-3.5)	
	stat	15		44	7.1	TDE[d]	48-h LC_{50}	3.2 (2.3-4.4)	Mayer & Ellersieck (1986)
	stat	21		44	7.1	TDE[d]	48-h LC_{50}	4.5 (3.1-6.6)	
Scud (*Gammarus fasciatus*)	stat	21	35	44	7.1	TDE[d]	24-h LC_{50}	4.6 (3.6-5.8)	Sanders (1972)
	stat	21	35	44	7.1	TDE[d]	96-h LC_{50}	0.6 (0.05-1.2)	Sanders (1972)
	stat	21	35	44	7.1	DDT[d]	24-h LC_{50}	15 (9.0-20)	Sanders (1972)
	stat	21	35	44	7.1	DDT[d]	96-h LC_{50}	3.2 (1.8-5.6)	Sanders (1972)
	stat	21	260	272	7.4	TDE[d]	24-h LC_{50}	3.2 (2.1-4.3)	Sanders (1972)
	stat	21	260	272	7.4	TDE[d]	96-h LC_{50}	0.86 (0.42-1.3)	Sanders (1972)
	stat	21	260	272	7.4	DDT[d]	24-h LC_{50}	4.2 (1.8-5.6)	Sanders (1972)
	stat	21	260	272	7.4	DDT[d]	48-h LC_{50}	3.1	Sanders (1972)
	stat	21	260	272	7.4	DDT[d]	96-h LC_{50}	1.8 (1.0-3.1)	Sanders (1972)
	stat	21	260	272	7.4	DDT[d]	120-h LC_{50}	0.32	Sanders (1972)
	flow	18-21	260	272	7.4	DDT[d]	24-h LC_{50}	1.1	Sanders (1972)
	flow	18-21	260	272	7.4	DDT[d]	48-h LC_{50}	1.0	Sanders (1972)
	flow	18-21	260	272	7.4	DDT[d]	96-h LC_{50}	0.8	Sanders (1972)
	flow	18-21	260	272	7.4	DDT[d]	120-h LC_{50}	0.6	Sanders (1972)
Scud (*Gammarus lacustris*)	stat	21		44	7.1	DDT[d]	24-h LC_{50}	4.7 (3.2-7.0)	Mayer & Ellersieck (1986)
	stat	21		44	7.1	DDT[d]	96-h LC_{50}	1.0 (0.68-1.5)	
	stat	15				DDT[e]	96-h LC_{50}	9.0	Gaufin et al. (1965)
Glass shrimp (*Palaemonetes kadiakensis*)	stat	21	260	272	7.4	DDT[d]	24-h LC_{50}	6.8 (6.2-7.5)	Sanders (1972)
	stat	21	260	272	7.4	DDT[d]	48-h LC_{50}	4.7	Sanders (1972)
	stat	21	260	272	7.4	DDT[d]	96-h LC_{50}	2.3 (1.3-4.9)	Sanders (1972)
	stat	21	260	272	7.4	DDT[d]	120-h LC_{50}	1.0	Sanders (1972)
	stat	21	260	272	7.4	TDE[d]	24-h LC_{50}	11 (8.4-16)	Sanders (1972)
	flow	18-21	260	272	7.4	TDE[d]	96-h LC_{50}	0.68 (0.47-1.1)	Sanders (1972)
	flow	18-21	260	272	7.4	DDT[d]	24-h LC_{50}	9.4	Sanders (1972)
	flow	18-21	260	272	7.4	DDT[d]	48-h LC_{50}	7.7	Sanders (1972)
	flow	18-21	260	272	7.4	DDT[d]	96-h LC_{50}	3.5	Sanders (1972)
	flow	18-21	260	272	7.4	DDT[d]	120-h LC_{50}	1.3	Sanders (1972)

Table 3. (Contd).

Organism	Flow Stat[a]	Temp (°C)	Alkalinity[c]	Hardness[c]	pH	Compound	Parameter	Water concentration (µg/litre)	Reference
Crayfish *(Orconectes nais)*									
mature	stat	21	260		7.4	DDT[d]	24-h LC_{50}	1100 (1000-1400)	Sanders (1972)
	stat	21	260		7.4	DDT[d]	96-h LC_{50}	100 (80-120)	Sanders (1972)
1 day old - 15g	stat	21	260		7.4	DDT[d]	24-h LC_{50}	1.4 (1.1-4.2)	Sanders (1972)
	stat	21	260		7.4	DDT[d]	96-h LC_{50}	0.3 (0.18-0.5)	Sanders (1972)
1 week old - 20g	stat	21	260		7.4	DDT[d]	24-h LC_{50}	1.0 (0.6-5.0)	Sanders (1972)
	stat	21	260		7.4	DDT[d]	96-h LC_{50}	0.18 (0.12-0.3)	Sanders (1972)
2 weeks old - 23g	stat	21	260		7.4	DDT[d]	24-h LC_{50}	1.2 (0.9-5.5)	Sanders (1972)
	stat	21	260		7.4	DDT[d]	96-h LC_{50}	0.2 (0.16-1.1)	Sanders (1972)
3 weeks old - 30g	stat	21	260		7.4	DDT[d]	24-h LC_{50}	1.0 (0.6-5.0)	Sanders (1972)
	stat	21	260		7.4	DDT[d]	96-h LC_{50}	0.24 (0.1-0.6)	Sanders (1972)
5 weeks old - 50g	stat	21	260		7.4	DDT[d]	24-h LC_{50}	3.2 (1.8-8.0)	Sanders (1972)
	stat	21	260		7.4	DDT[d]	96-h LC_{50}	0.9 (0.7-1.4)	Sanders (1972)
8 weeks old - 500g	stat	21	260		7.4	DDT[d]	24-h LC_{50}	45 (40-52)	Sanders (1972)
	stat	21	260		7.4	DDT[d]	96-h LC_{50}	28 (24-36)	Sanders (1972)
10 weeks old - 1200g	stat	21	260		7.4	DDT[d]	24-h LC_{50}	50 (48-56)	Sanders (1972)
	stat	21	260		7.4	DDT[d]	96-h LC_{50}	30 (26-42)	Sanders (1972)
Sowbug (isopod)	stat	21	35		7.1	DDT[d]	24-h LC_{50}	8.7 (4.9-13.0)	Sanders (1972)
(Asellus brevicaudus)	stat	21	35		7.1	DDT[d]	96-h LC_{50}	4.0 (1.2-6.5)	Sanders (1972)
	stat	21	35		7.1	TDE[d]	24-h LC_{50}	18 (14-25)	Sanders (1972)
	stat	21	35		7.1	TDE[d]	96-h LC_{50}	10 (7.0-14)	Sanders (1972)
Caddis fly (nymph) *(Hydropsyche californica)*	stat	10.5-12				DDT[e]	96-h LC_{50}	48	Gaufin et al. (1965)
Caddis fly (nymph) *(Arctopsyche grandis)*	stat	10.5-12				DDT[e]	96-h LC_{50}	175	Gaufin et al. (1965)

Table 3. (Contd).

May fly (nymph) *(Ephemerella grandis)*	stat	8.8-10		DDT[e]	96-h LC_{50}		25	Gaufin et al. (1965)
Stonefly (naiad) *(Acroneuria pacifica)*	stat	11-12		DDT[e]	96-h LC_{50}		320	Gaufin et al. (1965)
Stonefly (naiad)	stat	11-12		DDT[e]	96-h LC_{50}		1800	Gaufin et al. (1965)
(Pteronarcys californica)	stat	15.5	35		DDT	24-h LC_{50}	41 (27-62)	Sanders & Cope (1968)
	stat	15.5	35		DDT	48-h LC_{50}	19 (14-27)	
	stat	15.5	35		DDT	96-h LC_{50}	7 (4.9-9.9)	
	stat	15.5	35		TDE	24-h LC_{50}	3000 (2100-4300)	Sanders & Cope (1968)
	stat	15.5	35		TDE	48-h LC_{50}	1100 (800-1500)	
	stat	15.5	35		TDE	96-h LC_{50}	380 (280-520)	
Stonefly (naiad) *(Pteronarcella badia)*	stat	15.5	35		DDT	24-h LC_{50}	12 (8.8-16)	Sanders & Cope (1968)
	stat	15.5	35		DDT	48-h LC_{50}	9 (7-11)	
	stat	15.5	35		DDT	96-h LC_{50}	1.9 (1.3-2.7)	
Stonefly (naiad) *(Claasenia sabulosa)*	stat	15.5	35		DDT	24-h LC_{50}	16 (12-20)	Sanders & Cope (1968)
	stat	15.5	35		DDT	48-h LC_{50}	6.4 (4.9-8.3)	
	stat	15.5	35		DDT	96-h LC_{50}	3.5 (2.9-4.2)	

[a] Stat = static conditions (water unchanged for duration of test); Flow = flow-through conditions (DDT concentration in water continuously maintained).
[b] Static conditions but test solution renewed every 24 h.
[c] Alkalinity and hardness expressed as mg $CaCO_3$/litre.
[d] Technical grade (99%).
[e] Emulsifiable concentrate (25% active ingredient).
[f] Juv. = juvenile.
[g] Value based on total number of young produced.
[h] Value based on average brood size.
[i] Value based on % days reproducing.
[j] Effect on shell growth.

Eggs of the Chironomid midge, contaminated with DDE by exposure of the female during ovarian development, failed to hatch as many adults as uncontaminated eggs. DDE in the water had less of an effect than DDE contamination within the eggs obtained from the female. The females had been maintained in water containing 30 µg DDE/litre; eggs were kept in water containing 20 µg DDE/litre (Derr & Zabik, 1972).

Crayfish populations exposed over long periods to DDT develop some tolerance to the insecticide (Albaugh, 1972). In 48-h tests, LC_{50} values for the crayfish *Procambarus clarkii* were 3.0 (2.5-3.6) µg/litre for the unexposed population, and 7.2 (5.8-8.8) µg/litre for the exposed population (95% confidence limits in parentheses). Naqvi & Ferguson (1968) demonstrated the development of tolerance to DDT after exposure to the insecticide, in a wide variety of aquatic invertebrates, including cyclopoid copepods, tubifex worms, and pond snails. These tolerant populations occurred in the Mississippi delta in areas of cotton cultivation.

5.2 Fish

Appraisal

DDT is highly toxic to fish; the 96-h LC_{50}s reported (static tests) range from 1.5 to 56 µg/litre (for largemouth bass and guppy, respectively). Smaller fish are more susceptible than larger ones of the same species. An increase in temperature decreases the toxicity of DDT to fish.

The behaviour of fish is influenced by DDT. Goldfish exposed to 1 µg/litre exhibit hyperactivity. Changes in the feeding of young fish are caused by DDT levels commonly found in nature, and effects on temperature preference have been reported.

Residue levels of > 2.4 mg/kg in eggs of the winter flounder result in abnormal embryos in the laboratory, and comparable residue levels have been found to relate to the death of lake trout fry in the wild.

Cellular respiration may be the main toxic target of DDT since there are reports of effects on ATPase.

The toxicity of TDE and DDE has been less studied than that of DDT. However, the data available show that TDE and DDE are both less toxic than DDT.

The exact mode of action of DDT in fish remains unclear. There have been many different suggestions to explain both lethal and sublethal effects. Most of these are primarily the result of effects on membranes. DDT is very soluble in lipid and, therefore, dissolves in the lipid component of membranes. It may interfere both with membrane function and with many enzyme systems that are located on membranes. It has been shown experimentally to interfere with the normal function of so many systems that a primary action of DDT is difficult to determine.

5.2.1 Short-term and long-term direct toxicity to fish

The short-term toxicity of DDT to fish is summarized in Table 4.

The relatively few studies on TDE (Gardner, 1973; Korn & Earnest, 1974; Mayer & Ellersieck, 1986; Mayer, 1987) show it to be less toxic than DDT, in the same test system, by factors of 5-10. The still fewer studies on DDE indicate a similarly lowered toxicity relative to the parent compound (Mayer & Ellersieck, 1986; Mayer, 1987). Whilst there is some variation between species, DDT has proved highly toxic to all fish tested; static 24-h LC_{50} values range from 2.1 µg/litre for the largemouth bass (Mayer & Ellersieck, 1986) to 180 µg/litre for the goldfish (Henderson et al., 1959). For 96-h tests, LC_{50} values range from 1.5 µg/litre for largemouth bass (Mayer & Ellersieck, 1986) to 56 µg/litre for the guppy (Henderson et al., 1959). Several authors have stated that DDT toxicity varies somewhat with temperature and water hardness.

Buhler et al. (1969) studied the long-term effects, over 95 days, of feeding DDT-contaminated diets to juvenile chinook and coho salmon. The DDT was dissolved in corn-oil and then incorporated into a semi-synthetic diet. Fish were fed until they stopped actively taking the slowly sinking food. Pure *p,p'*-DDT was slightly more toxic to juvenile salmon than the technical product, and chinook salmon were 2 to 3 times more sensitive to the same dose of DDT in the diet than coho salmon. Size was an important factor in the toxicity of DDT, smaller fish being more susceptible than larger ones. The authors estimated, by extrapolation, a 90-day LD_{50} value of 27.5 µg/kg per day for chinook and 64 µg/kg per day for coho salmon juveniles. In fish exposed to higher doses of DDT, pre-death symptoms were marginal. Some increased agitation and slight photophobia were reported. Fish exposed to low doses of DDT took longer to die, and other symptoms were noted. Many individuals developed ulceration of the nasal area. This spread over the head and in some cases eyes were lost. Pathological examination showed a specific and severe kidney lesion; this was limited to one short section of the distal convoluted tubule, which eventually degenerated almost completely. The authors suggested this as the main lethal lesion in the fish.

In a later study (Buhler & Shanks, 1970), the same authors showed that median survival time was directly proportional to body weight in young coho salmon fed technical DDT. Fish were all given a diet containing 200 mg DDT/kg and food consumption was monitored for each group of fish. The main effect of body size on DDT lethality was related to the intake of the chemical by the fish; smaller fish ate more of the contaminated diet and consequently received the greatest dose in mg/kg bodyweight terms. However, even after correcting for dosage received, the smaller fish were more susceptible than larger ones. The authors suggested that the lower lipid content of smaller fish might have accounted for the remaining difference. Twelve groups of 100 fish ranged in weight (average for each group) from 3 to 15 g. Total DDT intake ranged from 0.4 to 3 mg/fish; daily intake was higher

in the smaller fish at 3 mg/kg per day, falling to 1.3 mg/kg per day for the largest. The estimated LC_{50} ranged from 95 mg/kg for the smallest to 135 mg/kg for the largest fish, and median survival time increased from 30 days for the smallest fish to 106 days for the largest.

Crawford & Guarino (1976) exposed killifish *(Fundulus heteroclitus)* to a twice-repeated schedule of 24 h in water containing DDT at a concentration of 0.1 mg/litre and 24 h in clean water. At this exposure level, there was a delay in the rate of development of fertilized eggs but no apparent effect on the hatched fry. Fertilization of killifish eggs was diminished when insemination was carried out in sea water containing DDT at 0.1 mg/litre. Mortality at a late stage of embryo development has been reported for a variety of salmonids and related to egg residues of DDT (Allison et al., 1964, for cutthroat trout; Burdick et al., 1964, for lake trout; Macek, 1968, for brook trout; and Johnson & Pecor, 1969, for coho salmon).

Smith & Cole (1973) reported effects on embryos developing from eggs laid by adult winter flounder *(Pseudopleuronectes americanus)* that were exposed to 2 µg DDT/litre for various times and, therefore, accumulated different residue levels in the eggs. These residue levels varied from 1.15 to 3.70 mg DDT/kg and from 0.07 to 0.4 mg DDE/kg. Embryos showed abnormal gastrulation and a high incidence (mean 39%) of vertebral deformities. Bone erosion and haemorrhaging at the vertebral junctures were often associated with the vertebral deformities.

Halter & Johnson (1974) report that DDT is toxic to the early life-stages of coho salmon. Mean survival times were considerably reduced by water concentrations of DDT greater than 0.5 µg/litre.

5.2.2 Sublethal behavioural effects on fish

Hansen (1969) and Hansen et al. (1972) investigated the avoidance of DDT by sheepshead minnows and mosquitofish in a 'Y'-shaped avoidance maze. Although there was some statistically significant avoidance of DDT when fish were given the choice between DDT and clean water, this only occurred at concentrations of the insecticide above the 24-h LC_{50}. Fish of both species, when given the choice between DDT at 0.1 and 0.01 mg/litre, chose the higher concentration of the chemical. This suggests that the perception of DDT is poor and that fish could not reliably avoid DDT in water at toxic concentrations.

Olofsson & Lindahl (1979) administered either 0.5 or 1.0 mg DDT/kg body weight to cod by oral intubation. There was a significant effect, at the higher dose but not the lower one, on the ability of the fish to compensate its posture to cope with a rotating tube in which it was swimming.

Hansen (1972) allowed mosquitofish to select a desired salinity in a fluvarium with a salinity gradient. Fish selected a higher salinity than controls when exposed to DDT, but only at exposure levels which caused some mortality. The author suggested that DDT might have affec-

Table 4. Toxicity of DDT and its derivatives to fish

Organism	Size (g)/ age[f]	Flow/ stat[a]	Tem- perat- ure(°C)	Salinity °/oo	Compound	Parameter	Water concentration (µg/litre)	Reference
Estuarine and marine fish								
Dwarf perch	1.2-11.0	Stat	13	28	DDT[c]	96-h LC_{50}	4.6	Earnest &
(Micrometrus minimus)	1.2-11.0	flow[b]	14-18	26-28	DDT[c]	96-h LC_{50}	0.26 (0.13-0.52)	Benville (1972)
Shiner perch	1.2-11.0	stat	13	26	DDT[c]	96-h LC_{50}	7.6	Earnest &
(Cymatogaster aggregata)	1.2-11.0	flow[b]	14-18	13-23	DDT[c]	96-h LC_{50}	0.45 (0.21-0.94)	Benville (1972)
Striped bass	2.7	flow[b]	17	28	DDT(77%)	96-h LC_{50}	0.53 (0.38-0.84)	Korn & Earnest
(Morone saxatilis)	0.6	flow[b]	17	30	TDE[c]	96-h LC_{50}	2.5 (1.6-4.0)	(1974)
Sheepshead minnow *(Cyprinodon variegatus)*	juv.	flow	15	30	DDT[c]	48-h LC_{50}	2.0	Mayer (1987)
Longnose killifish	juv.	flow	15	30	DDT[c]	48-h LC_{50}	2.8	Mayer (1987)
(Fundulus similis)	juv.	flow	16	28	TDE[c]	48-h LC_{50}	42.0	Mayer (1987)
Pinfish *(Lagodon rhomboides)*	juv.	flow	22	29	DDT[c]	48-h LC_{50}	0.3	Mayer (1987)
Striped mullet *(Mugil cephalus)*	juv.	flow	15	30	DDT[c]	48-h LC_{50}	0.4	Mayer (1987)
Spot	juv.	flow	12	26	DDE[c]	48-h LC_{50}	> 100	Mayer (1987)
(Leiostomus xanthurus)	juv.	flow	26	30	TDE[c]	48-h LC_{50}	20.0	Mayer (1987)
Three-spined	0.4-0.8	stat	20	5	DDT	24-h LC_{50}	22.0	Katz (1961)
stickleback	0.4-0.8	stat	20	5	DDT	48-h LC_{50}	21.0	Katz (1961)
(Gasterosteus	0.4-0.8	stat	20	5	DDT	72-h LC_{50}	18.5	Katz (1961)
aculeatus)	0.4-0.8	stat	20	5	DDT	96-h LC_{50}	18.0	Katz (1961)
	0.4-0.8	stat	20	25	DDT	24-h LC_{50}	18.0	Katz (1961)
	0.4-0.8	stat	20	25	DDT	48-h LC_{50}	15.0	Katz (1961)
	0.4-0.8	stat	20	25	DDT	72-h LC_{50}	14.5	Katz (1961)
	0.4-0.8	stat	20	25	DDT	96-h LC_{50}	11.5	Katz (1961)

Table 4. (Contd).

Organism	Size (g)	Flow/ stat[a]	Tem- perat- ure(°C)	Alkali- nity[d]	Hard- ness[d]	pH	Compound	Parameter	Water concentration (µg/litre)	Reference
Freshwater fish										
Black bullhead	1.2	stat	18		44	7.1	DDT[c]	24-h LC_{50}	36.8 (20.3-67.0)	Mayer &
(Ictalurus melas)	1.2	stat	18		44	7.1	DDT[c]	96-h LC_{50}	4.8 (3.4-6.8)	Ellersieck[g]
	1.2	stat	18		272	7.4	DDT[c]	24-h LC_{50}	26.2 (22.0-31.3)	Mayer &
	1.2	stat	18		272	7.4	DDT[c]	96-h LC_{50}	5.1 (3.9-6.7)	Ellersieck[g]
Channel catfish	1.5	stat	18		44	7.1	DDT[c]	24-h LC_{50}	22.0 (18.2-26.5)	Mayer &
(Ictalurus punctatus)	1.5	stat	18		44	7.1	DDT[c]	96-h LC_{50}	21.5 (17.7-26.1)	Ellersieck[g]
	1.5	stat	18		272	7.4	DDT[c]	24-h LC_{50}	18.4 (13.7-24.7)	Mayer &
	1.5	stat	18		272	7.4	DDT[c]	96-h LC_{50}	17.3 (13.0-23.1)	Ellersieck[g]
	0.7	stat	18		44	7.1	DDT[c]	24-h LC_{50}	17.9 (12.7-25.3)	Mayer &
	0.7	stat	18		44	7.1	DDT[c]	96-h LC_{50}	6.9 (5.7-8.5)	Ellersieck[g]
	1.6	stat	18		44	7.1	DDT[c]	24-h LC_{50}	44.0 (37.0-52.0)	Mayer &
	1.6	stat	18		44	7.1	DDT[c]	96-h LC_{50}	22.0 (19.0-26.0)	Ellersieck[g]
	1.4	stat	18		44	7.1	DDT[c]	24-h LC_{50}	30.0 (22.0-41.0)	Mayer &
	1.4	stat	18		44	7.1	DDT[c]	96-h LC_{50}	16.0 (9.4-29.0)	Ellersieck[g]
	1.4	stat	18		272	7.7	DDT[c]	24-h LC_{50}	29.0 (20.0-41.0)	Mayer &
	1.4	stat	18		272	7.7	DDT[c]	96-h LC_{50}	7.0 (4.3-11.0)	Ellersieck[g]
Atlantic salmon	0.45	stat	12		40	7.5	DDT[c]	24-h LC_{50}	6.2 (4.6-8.4)	Mayer &
(Salmo salar)	0.45	stat	12		40	7.5	DDT[c]	96-h LC_{50}	1.8 (1.3-2.6)	Ellersieck[g]
	0.5	stat	12		44	7.5	DDE[c]	96-h LC_{50}	96.0 (52.1-177)	Mayer & Ellersieck[g]
Coho salmon	2.7-4.1	stat	20	45-57		6.8-7.4	DDT	24-h LC_{50}	66.0	Katz (1961)
(Oncorhynchus	2.7-4.1	stat	20	45-57		6.8-7.4	DDT	48-h LC_{50}	46.0	Katz (1961)
kisutch)	2.7-4.1	stat	20	45-57		6.8-7.4	DDT	72-h LC_{50}	44.0	Katz (1961)
	2.7-4.1	stat	20	45-57		6.8-7.4	DDT	96-h LC_{50}	44.0	Katz (1961)
	1.0	stat	13		44	7.1	DDT[c]	24-h LC_{50}	10.0 (7.0-12.0)	Mayer &
	1.0	stat	13		44	7.1	DDT[c]	96-h LC_{50}	4.0 (3.0-6.0)	Ellersieck[g]
	6.0	stat	13		40	7.1	DDT[c]	24-h LC_{50}	26.9 (18.1-40.0)	Mayer &
	6.0	stat	13		40	7.1	DDT[c]	96-h LC_{50}	19.3 (9.6-38.8)	Ellersieck[g]

Table 4. (Contd).

Chinook salmon	1.5-5.0	stat	20	45-57		6.8-7.4	DDT	24-h LC_{50}	38.0	Katz (1961)
(Oncorhynchus	1.5-5.0	stat	20	45-57		6.8-7.4	DDT	48-h LC_{50}	17.0	Katz (1961)
tshawytscha)	1.5-5.0	stat	20	45-57		6.8-7.4	DDT	72-h LC_{50}	14.0	Katz (1961)
	1.5-5.0	stat	20	45-57		6.8-7.4	DDT	96-h LC_{50}	11.5	Katz (1961)
Rainbow trout	0.9	stat	7		44	7.1	DDT[c]	24-h LC_{50}	7.5 (6.7-8.3)	Mayer &
(Salmo gairdneri)	0.9	stat	7		44	7.1	DDT[c]	96-h LC_{50}	4.1 (3.6-4.6)	Ellersieck[g]
	0.9	stat	13		44	7.1	DDT[c]	24-h LC_{50}	8.2 (7.2-9.2)	Mayer &
	0.9	stat	13		44	7.1	DDT[c]	96-h LC_{50}	4.7 (4.2-5.3)	Ellersieck[g]
	0.9	stat	18		44	7.1	DDT[c]	24-h LC_{50}	12.0 (1.0-13.0)	Mayer &
	0.9	stat	18		44	7.1	DDT[c]	96-h LC_{50}	5.8 (5.2-6.5)	Ellersieck[g]
	3.2	stat	20	45-57		6.8-7.4	DDT	24-h LC_{50}	42.0	Katz (1961)
	3.2	stat	20	45-57		6.8-7.4	DDT	48-h LC_{50}	42.0	Katz (1961)
	3.2	stat	20	45-57		6.8-7.4	DDT	72-h LC_{50}	42.0	Katz (1961)
	3.2	stat	20	45-57		6.8-7.4	DDT	96-h LC_{50}	42.0	Katz (1961)
	1.8	flow	17		272	7.4	DDT[c]	96-h LC_{50}	> 3.0	Mayer &
	0.8	stat	12		44	7.1	DDE[c]	96-h LC_{50}	32.0 (26.0-40.0)	Ellersieck[g]
	1.0	stat	12		44	7.1	TDE[c]	96-h LC_{50}	70.0 (57.0-87.0)	Mayer &
	1.0	stat	12		272	7.4	TDE[c]	96-h LC_{50}	70.0 (58.0-85.0)	Ellersieck[g]
Cutthroat trout	1.0	stat	13		44	7.1	DDT[c]	24-h LC_{50}	8.4 (7.6-9.2)	Mayer &
(Salmo clarki)	1.0	stat	13		44	7.1	DDT[c]	96-h $_{50}$	5.5 (4.7-6.4)	Ellersieck[g]
	1.8	stat	9		162	7.4	DDT[c]	24-h LC_{50}	11.3 (9.4-13.6)	Mayer &
	1.8	stat	9		162	7.4	DDT[c]	96-h LC_{50}	7.9 (6.5-9.7)	Ellersieck[g]
Brown trout	1.7	stat	13		44	7.1	DDT[c]	96-h LC_{50}	1.8 (1.3-2.5)	Mayer &
(Salmo trutta)										Ellersieck[g]
Northern pike	0.7	stat	18		272	7.4	DDT[c]	24-h LC_{50}	5.5	Mayer &
(Esox lucius)	0.7	stat	18		272	7.4	DDT[c]	96-h LC_{50}	2.7	Ellersieck[g]

Table 4. (Contd).

Organism	Size (g)	Flow/ stat[a]	Temperature (°C)	Alkalinity[d]	Hardness[d]	pH	Compound	Parameter	Water concentration (µg/litre)	Reference
Guppy	0.1-0.2	stat	25	18	20	7.4	DDT[c]	24-h LC_{50}	135	Henderson
(Lebistes	0.1-0.2	stat	25	18	20	7.4	DDT[c]	48-h LC_{50}	72.0	et al.
reticulatus)	0.1-0.2	stat	25	18	20	7.4	DDT[c]	96-h LC_{50}	56.0	(1959)
River shiner	0.3	stat	18		44	7.1	DDT[c]	24-h LC_{50}	6.7 (4.9-9.1)	Mayer &
(Notropis blennius)	0.3	stat	18		44	7.1	DDT[c]	96-h LC_{50}	5.8 (3.6-9.1)	Ellersieck[g]
Fathead minnow	1.2	stat	18		44	7.1	DDT[c]	24-h LC_{50}	14.2 (11.0-18.0)	Mayer &
(Pimephales	1.2	stat	18		44	7.1	DDT[c]	96-h LC_{50}	12.4 (10.0-15.4)	Ellersieck[g]
promelas)	1.2	stat	18		272	7.4	DDT[c]	24-h LC_{50}	13.8 (10.3-18.3)	Mayer &
	1.2	stat	18		272	7.4	DDT[c]	96-h LC_{50}	13.2 (10.1-17.3)	Ellersieck[g]
	0.9	flow	12		314	7.6	DDT[c]	96-h LC_{50}	9.9 (6.5-15.0)	Mayer & Ellersieck[g]
	1.0-2.0	stat	25	18	20	7.4	DDT[c]	24-h LC_{50}	56.0	Henderson
	1.0-2.0	stat	25	18	20	7.4	DDT[c]	48-h LC_{50}	45.0	et al. (1959)
	1.0-2.0	stat	25	18	20	7.4	DDT[c]	96-h LC_{50}	42.0	Henderson
	1.0-2.0	stat	25	360	400	8.2	DDT[c]	24-h LC_{50}	78.0	et al. (1959)
	1.0-2.0	stat	25	360	400	8.2	DDT[c]	48-h LC_{50}	68.0	Henderson
	1.0-2.0	stat	25	360	400	8.2	DDT[c]	96-h LC_{50}	45.0	et al. (1959)
	1.0-2.0	stat	25	18	20	7.4	DDT	24-h LC_{50}	32.0	Henderson
	1.0-2.0	stat	25	18	20	7.4	DDT	48-h LC_{50}	26.0	et al. (1959)
	1.0-2.0	stat	25	18	20	7.4	DDT	96-h LC_{50}	26.0	Henderson
	1.0-2.0	stat	25	360	400	8.2	DDT	24-h LC_{50}	29.0	et al. (1959)
	1.0-2.0	stat	25	360	400	8.2	DDT	48-h LC_{50}	27.0	Henderson
	1.0-2.0	stat	25	360	400	8.2	DDT	96-h LC_{50}	26.0	et al. (1959)
Mosquitofish	0.2	stat	25				DDT[c]	24-h LC_{50}	22.7 (16.6-31.1)	El-Sebae (1987)
(Gambusia affinis)	0.2	stat	25				DDT[c]	96-h LC_{50}	9.9 (7.3-13.4)	El-Sebae (1987)
	0.2	stat	25				DDT[e]	24-h LC_{50}	58.6 (43.2-79.5)	El-Sebae (1987)
	0.2	stat	25				DDT[e]	96-h LC_{50}	27.7 (21.3-36.0)	El-Sebae (1987)

Table 4. (Contd).

Bluegill sunfish	0.26	stat	19	138	192	8.2-8.5	DDT[c]	96-h LC_{50}	3.4 (2.6-4.1)	Randall
(Lepomis macrochirus)	0.26	stat	19	138	192	8.2-8.5	DDT (25%)	96-h LC_{50}	9.0 (7.4-10.6)	et al. (1979)
	1.0-2.0	stat	25	18	20	7.4	DDT[c]	24-h LC_{50}	26.0	Henderson
	1.0-2.0	stat	25	18	20	7.4	DDT[c]	48-h LC_{50}	21.0	et al.
	1.0-2.0	stat	25	18	20	7.4	DDT[c]	96-h LC_{50}	21.0	(1959)
	1.5	stat	18		44	7.1	DDT[c]	24-h LC_{50}	11.5 (8.4-16.0)	Mayer &
	1.5	stat	18		44	7.1	DDT[c]	96-h LC_{50}	8.6 (6.2-12.0)	Ellersieck[g]
	1.5	stat	18		272	7.4	DDT[c]	24-h LC_{50}	10.0 (8.5-12.9)	Mayer &
	1.5	stat	18		272	7.4	DDT[c]	96-h LC_{50}	6.3 (4.3-9.3)	Ellersieck[g]
	0.9	stat	17		44	7.1	DDE[c]	96-h LC_{50}	240 (201-286)	Mayer &
	0.9	stat	24		44	7.4	TDE[c]	24-h LC_{50}	56.0 (46.0-68.0)	Ellersieck[g]
	0.9	stat	24		44	7.4	TDE[c]	96-h LC_{50}	42.0 (36.0-49.0)	Mayer & Ellersieck[g]
Redear sunfish	3.2	stat	24		44	7.1	DDT[c]	24-h LC_{50}	19.0	Mayer &
(Lepomis microlophus)	3.2	stat	24		44	7.1	DDT[c]	96-h LC_{50}	15.0	Ellersieck[g]
Green sunfish	1.1	stat	18		44	7.1	DDT[c]	24-h LC_{50}	16.9 (12.7-22.3)	Mayer &
(Lepomis cyanellus)	1.1	stat	18		44	7.1	DDT[c]	96-h LC_{50}	10.9 (7.3-15.6)	Ellersieck[g]
	0.8	stat	18		44	7.1	DDT[c]	24-h LC_{50}	18.0 (13.0-24.0)	Mayer &
	0.8	stat	18		44	7.1	DDT[c]	96-h LC_{50}	6.5 (4.1-10.4)	Ellersieck[g]
	1.1	stat	18		272	7.7	DDT[c]	24-h LC_{50}	19.8 (15.0-25.6)	Mayer &
	1.1	stat	18		272	7.7	DDT[c]	96-h LC_{50}	9.9 (6.4-15.0)	Ellersieck[g]
Largemouth bass	0.8	stat	18		44	7.1	DDT[c]	24-h LC_{50}	3.7 (3.1-4.5)	Mayer &
(Micropterus	0.8	stat	18		44	7.1	DDT[c]	96-h LC_{50}	1.5 (0.9-2.4)	Ellersieck[g]
Salmoides)	0.8	stat	18		272	7.4	DDT[c]	24-h LC_{50}	2.1 (1.6-2.9)	Mayer &
	0.8	stat	18		272	7.4	DDT[c]	96-h LC_{50}	1.5 (0.9-2.4)	Ellersieck[g]
	0.7	stat	18		44	7.1	TDE[c]	24-h LC_{50}	50.0 (35.0-71.0)	Mayer &
	0.7	stat	18		44	7.1	TDE[c]	96-h LC_{50}	42.0 (34.0-51.0)	Ellersieck[g]

Table 4. (Contd).

Organism	Size (g)	Flow/ stat[a]	Tem- perat- ure(°C)	Alkali- nity[d]	Hard- ness[d]	pH	Compound	Parameter	Water concentration (µg/litre)	Reference
Black crappie	1.0	stat	18		44	7.1	DDT[c]	24-h LC_{50}	6.5 (5.4-7.8)	Mayer &
(Pomoxis nigromaculatus)	1.0	stat	18		44	7.1	DDT[c]	96-h LC_{50}	5.6 (4.6-6.7)	Ellersieck[g]
Yellow perch	1.4	stat	18		44	7.1	DDT[c]	24-h LC_{50}	10.0 (8.0-12.0)	Mayer &
(Perca flavescens)	1.4	stat	18		44	7.1	DDT[c]	96-h LC_{50}	9.0 (7.0-11.0)	Ellersieck[g]
Walleye	1.4	stat	18		44	7.1	DDT[c]	24-h LC_{50}	4.2 (3.2-5.6)	Mayer &
(Stizostedion v. vitreum)	1.4	stat	18		44	7.1	DDT[c]	96-h LC_{50}	2.9 (2.4-3.5)	Ellersieck[g]
	1.3	stat	18		272	7.4	DDT[c]	24-h LC_{50}	4.6 (3.9-5.4)	Mayer &
	1.3	stat	18		272	7.4	DDT[c]	96-h LC_{50}	4.6 (3.9-5.4)	Ellersieck[g]
	1.0	stat	18		44	7.1	TDE[c]	24-h LC_{50}	20.0 (16.0-24.0)	Mayer &
	1.0	stat	18		44	7.1	TDE[c]	96-h LC_{50}	14.0 (11.0-19.0)	Ellersieck[g]
Tilapia	0.8	stat	24		44	7.1	DDT[c]	24-h LC_{50}	19.0 (16.0-23.0)	Mayer &
(Tilapia mossambica)	0.8	stat	24		44	7.1	DDT[c]	96-h LC_{50}	17.0 (14.0-21.0)	Ellersieck[g]
	0.8	stat	24		272	7.4	DDT[c]	24-h LC_{50}	15.0 (13.0-17.0)	Mayer &
	0.8	stat	24		272	7.4	DDT[c]	96-h LC_{50}	14.0 (12.0-16.0)	Ellersieck[g]
	0.8	flow	18		272	7.4	DDT[c]	24-h LC_{50}	24.0 (17.0-32.0)	Mayer &
	0.8	flow	18		272	7.4	DDT[c]	96-h LC_{50}	5.1 (3.2-8.1)	Ellersieck[g]
Tilapia	0.8	stat	25				DDT[c]	24-h LC_{50}	21.8 (17.0-28.0)	El-Sebae (1987)
(Tilapia zilli)	0.8	stat	25				DDT[c]	96-h LC_{50}	15.5 (11.7-20.6)	El-Sebae (1987)
	0.8	stat	25				DDT[e]	24-h LC_{50}	12.8 (9.6-17.1)	El-Sebae (1987)
	0.8	stat	25				DDT[e]	96-h LC_{50}	9.5 (7.4-12.3)	El-Sebae (1987)

Table 4. (Contd).

Goldfish	1.0-2.0	stat	25	18	20	7.4	DDT[c]	24-h LC_{50}	180	Henderson
(*Carassius auratus*)	1.0-2.0	stat	25	18	20	7.4	DDT[c]	48-h LC_{50}	47.0	et al.
	1.0-2.0	stat	25	18	20	7.4	DDT[c]	96-h LC_{50}	36.0	(1959)
	0.9	stat	18		44	7.1	DDT[c]	24-h LC_{50}	24.0 (17.0-33.0)	Mayer &
	0.9	stat	18		44	7.1	DDT[c]	96-h LC_{50}	15.5 (9.1-26.0)	Ellersieck[g]
	0.9	stat	18		272	7.4	DDT[c]	24-h LC_{50}	22.2 (16.0-31.1)	Mayer &
	0.9	stat	18		272	7.4	DDT[c]	96-h LC_{50}	14.7 (10.0-20.0)	Ellersieck[g]
Common carp	0.6	stat	18		44	7.1	DDT[c]	24-h LC_{50}	14.0 (10.0-19.0)	Mayer &
(*Cyprinus carpio*)	0.6	stat	18		44	7.1	DDT[c]	96-h LC_{50}	9.7 (7.4-12.9)	Ellersieck[g]

[a] Stat = static conditions (water unchanged for duration of test); Flow = flow-through conditions (DDT concentration in water continuously maintained).
[b] Intermittent flow-through conditions.
[c] Technical grade (99%).
[d] Alkalinity and hardness expressed as mg $CaCO_3$/litre.
[e] 25% emulsifiable concentrate.
[f] Juv. = juvenile.
[g] 1986.

ted the osmoregulatory ability of the mosquitofish. Other possible explanations include a change in sensitivity of nerves to stimuli or a preference for the pre-exposure salinity, which was 15 g/litre.

Peterson (1973) monitored the selection of temperature by juvenile Atlantic salmon *(Salmo salar)* previously exposed to DDT or its metabolites. Low concentrations produced no effect on temperature selection, but at higher levels of exposure the temperature selected by the fish increased. Fish were most sensitive, in this respect, to *p,p'*-DDE and showed decreasing sensitivity to *o,p'*-DDT, *p,p'*-TDE, and *p,p'*-DDT. Increasing the exposure to *p,p'*-DDE from 0 to 1.0 mg/litre increased the preferred temperature from about 16 °C to 21 °C. There was no effect of *p,p'*-DDA on temperature selection at concentrations as high as 8 mg/litre. In a similar experiment, where brook trout *(Salvelinus fontinalis)* were exposed to a vertical rather than horizontal temperature gradient, fish previously exposed to *p,p'*-DDT and *p,p'*-TDE selected higher temperatures than controls. Conversely, Gardner (1973) found that DDT and its analogues induced selection of lower temperatures by the same species of fish over a dose range between 0 and 50 µg/litre; DDE did not produce any temperature preference. Ogilvie & Miller (1976) reported that Atlantic salmon exposed to DDT at a concentration of 50 µg/litre selected higher temperatures, the effect persisting for at least 4 weeks after exposure. The authors suggested that the temperature selection response to DDT exposure is "biphasic". At low exposure levels, similar to those used by Gardner (1973), lower temperatures are selected, whilst higher temperatures are preferred at higher exposure levels.

Dill & Saunders (1974) exposed the eggs of Atlantic salmon at gastrulation to DDT at water concentrations of 5, 10, 50, or 100 µg/litre, and observed behavioural development in hatched fry over 30 days following hatch. The two highest doses of DDT impaired balance and retarded behavioural development of the fry (i.e., the appearance of normal behaviour patterns was delayed). The authors considered that the effects observed would affect predation rates and feeding, in young fish, at "realistic" DDT exposure levels in the wild.

Davy et al. (1973) reported that exposure to DDT, at 10 µg/litre for 4 days, affected the exploratory behaviour of goldfish experiencing a novel environment. They attributed the effect to a central nervous system lesion caused by DDT. Weis & Weis (1974) showed an increase in individual activity and an increase in school-size in groups of goldfish exposed to DDT at 1 µg/litre for 7 days. After a frightening stimulus, schools scattered further and did not regroup as readily as control fish. The transfer of fish to clean water led to a return to normal behaviour within one week. An effect on the locomotor behaviour of goldfish after exposure to 10 µg DDT/litre persisted for the remainder of the observation period of 130-139 days (Davy et al., 1972).

5.2.3 Physiological effects on fish

Hanke et al. (1983) investigated the effects of DDT on a range of physiological functions in carp *(Cyprinus carpio)*. At water concentrations of 100 or 500 µg/litre, the insecticide induced changes in plasma cortisol and glucose levels, liver glycogen level, and plasma and brain acetylcholinesterase activity. The response was biphasic in all cases. Initially, after 6 hours, there was a stimulation of these parameters which, within 24 hours, changed to an inhibition. Ramalingam & Ramalingam (1982) reported that the chronic effect of DDT on glycogen utilization in fish led to the use of protein as an energy source. The protein content of tissues declined after chronic exposure to DDT.

Janicki & Kinter (1971) found that DDT impaired fluid absorption in the intestinal sacs of eels adapted to sea water and exposed to the insecticide at 50 µg/litre. DDT also inhibited Na^+-, K^+-, and Mg^{2+}-dependent ATPases in homogenates of the intestinal mucosa. In a later study, Kinter et al. (1972) showed that plasma osmolarity was also affected in sea-water-adapted eels exposed to DDT (1 mg/litre) for 9 to 10 hours. Haux & Larsson (1979) reported effects of DDT on plasma electrolytes in the flounder *Platichthys flesus* kept in hypotonic, brackish water. The fish were force-fed with DDT in gelatin capsules to give a total DDT dose of 1.5 or 15.0 mg/kg body weight. Plasma sodium was reduced but not significantly; plasma chloride was significantly reduced in a dose-related manner after 3 weeks but not after 6 weeks. Waggoner & Zeeman (1975) reported similar effects on plasma electrolytes in the black surfperch *(Embiotoca jacksoni)*, but only at high DDT exposure levels. They injected DDT doses of 1, 10, 100, or 200 mg/kg; the only effect occurred with the dose of 200 mg/kg, but the fish did not survive to 72 h. The authors suggested that osmoregulatory effects are not the major cause of DDT-induced mortality in marine fish.

Desaiah et al. (1975) exposed fathead minnows *(Pimephales promelas)* for long periods to DDT at water concentrations of 0.5 or 2.0 µg/litre and also via the food, and monitored the activity of ATPases in brain and gill. This study followed up several previous studies on *in vitro* effects on these enzymes. After 266 days of exposure, there was an approximately 50% reduction in brain oligomycin-sensitive (mitochondrial) Mg^{2+}-ATPase activity. In contrast, oligomycin-insensitive Mg^{2+}-ATPase activity was increased by almost 40%. Total Mg^{2+}-ATPase activity was, therefore, almost unaffected by DDT. There was a less obvious (about 18%) activation of Na^+-K^+-ATPase activity in the brain. Gill tissue showed different results; all the ATPases studied were inhibited by DDT. The authors suggested that a major factor in the toxicity of DDT to fish (and other organisms) could be the inhibition of oxidative phosphorylation. Moffett & Yarbrough (1972) investigated the enzyme succinic dehydrogenase in insecticide-resistant and insecticide-susceptible mosquitofish *(Gambusia affinis)* in an attempt to discover if

resistance could be related to membrane effects of DDT. They found that the effect on membrane-bound enzymes was, indeed, reduced in resistant fish. This may not explain all the factors involved in resistance, since DDT uptake from water may also be reduced.

5.2.4 Development of tolerance

The development of tolerance to DDT in fish has been reported. Vinson et al. (1963) reported DDT tolerance in mosquitofish *(Gambusia affinis)* exposed long-term to DDT in the wild, and Boyd & Ferguson (1964) showed TDE tolerance in the same species. However, fish exposed long-term to DDT do not always show tolerance. Ferguson et al. (1964) recorded tolerance to a variety of organochlorine insecticides in three species of freshwater fish from the Mississippi delta area of cotton cultivation, but there was no tolerance to DDT. El-Sebae (1987) determined the LC_{50} values for two populations of *Tilapia zilli* from different areas of Egypt. Fish from the Behera Governate which had been taken from agricultural drains showed exactly the same susceptibility to DDT (25% EC) as fish taken from a less contaminated area in the Alexandria Governate. Tolerance had developed to other insecticides in these different strains.

5.3 Toxicity to Amphibians

Appraisal

The toxicity of DDT and its metabolites to amphibians varies from species to species; although only a few data are available, amphibian larvae seem to be more sensitive than adults to DDT. TDE seems to be more toxic than DDT to amphibians, but there are no data available for DDE. All the studies reported have been static tests and, therefore, results should be treated with caution.

The toxicity of DDT and TDE to amphibians is summarized in Table 5. Both compounds are toxic to amphibian larvae at low water concentrations.

Two studies (Harri et al., 1979; Hudson et al., 1984) showed that DDT is moderately toxic to adult frogs when given orally. Repeated oral dosing of adult common frogs *(Rana temporaria)* with DDT at 0.6 mg/kg body weight twice weekly for 8 weeks, led to no mortality when the animals were fed (Harri et al., 1979). Frogs dosed in the same way, but not fed, showed 50% mortality by the end of dosing. The first animal died after the fifth dose and all others showed symptoms of poisoning.

A study by Sanders (1970) indicated that the toxicity of DDT to tadpoles of Fowler's toad increased with age of the tadpole. The 24- and 96-h LC_{50} values of 5.3 and 0.75 mg/litre for one-week-old tadpoles fell to 1.4 and 0.03 mg/litre, respectively, by the time the tadpoles were 7 weeks old. TDE was only tested on one age range of

Table 5. Toxicity of DDT and its derivatives to amphibians

Organism	Flow/ Stat[a]	Temperature (°C)	Alkalinity[b]	pH	Compound	Parameter	Water concentration (µg/litre)	Reference
Fowler's toad (tadpole) *(Bufo woodhousii)*								
1 week old - 15 mg	stat	15.5	30	7.1	DDT	24-h LC_{50}	5300 (2900-9900)	Sanders (1970)
	stat	15.5	30	7.1	DDT	48-h LC_{50}	1800 (950-3300)	Sanders (1970)
	stat	15.5	30	7.1	DDT	96-h LC_{50}	750 (280-2000)	Sanders (1970)
2-3 weeks old - 56 mg	stat	15.5	30	7.1	DDT	24-h LC_{50}	5400 (2900-10 000)	Sanders (1970)
	stat	15.5	30	7.1	DDT	48-h LC_{50}	1300 (320-5300)	Sanders (1970)
4-5 weeks old - 74 mg	stat	15.5	30	7.1	DDT	24-h LC_{50}	2400 (730-8000)	Sanders (1970)
	stat	15.5	30	7.1	DDT	48-h LC_{50}	1000 (40-6500)	Sanders (1970)
	stat	15.5	30	7.1	DDT	96-h LC_{50}	1000 (20-3600)	Sanders (1970)
	stat	15.5	30	7.1	TDE	24-h LC_{50}	700 (250-2000)	Sanders (1970)
	stat	15.5	30	7.1	TDE	48-h LC_{50}	320 (210-450)	Sanders (1970)
	stat	15.5	30	7.1	TDE	96-h LC_{50}	140 (100-210)	Sanders (1970)
6 weeks old - 350 mg	stat	15.5	30	7.1	DDT	24-h LC_{50}	2200 (550-15 000)	Sanders (1970)
	stat	15.5	30	7.1	DDT	48-h LC_{50}	410 (280-600)	Sanders (1970)
	stat	15.5	30	7.1	DDT	96-h LC_{50}	100 (20-600)	Sanders (1970)
7 weeks old - 600 mg	stat	15.5	30	7.1	DDT	24-h LC_{50}	1400 (900-2000)	Sanders (1970)
	stat	15.5	30	7.1	DDT	48-h LC_{50}	750 (610-1100)	Sanders (1970)
	stat	15.5	30	7.1	DDT	96-h LC_{50}	30 (6-400)	Sanders (1970)

Table 5. (Contd).

Organism	Flow/ Stat[a]	Temperature (°C)	Alkalinity[b]	pH	Compound	Parameter	Water concentration (µg/litre)	Reference
Western chorus frog	stat	15.5	30	7.1	DDT	24-h LC_{50}	1400 (910-2800)	Sanders (1970)
(*Pseudacris*	stat	15.5	30	7.1	DDT	48-h LC_{50}	900 (400-1500)	Sanders (1970)
triseriata)	stat	15.5	30	7.1	DDT	96-h LC_{50}	800 (500-2300)	Sanders (1970)
(1-week-old tadpole)	stat	15.5	30	7.1	TDE	24-h LC_{50}	610 (410-820)	Sanders (1970)
	stat	15.5	30	7.1	TDE	48-h LC_{50}	500 (210-750)	Sanders (1970)
	stat	15.5	30	7.1	TDE	96-h LC_{50}	400 (210-750)	Sanders (1970)
Bullfrog (*Rana catesbeiana*)					DDT(77.2%)	acute LD_{50}[c]	> 2000 µg/kg	Hudson et al. (1984)
Common frog (*Rana temporaria*)		15			DDT	acute LD_{50}[c]	7600 µg/kg	Harri et al. (1979)

[a] Stat = static conditions (water unchanged for duration of test); flow = flow-through conditions (DDT concentration in water continuously maintained).

[b] alkalinity expressed as mg $CaCO_3$/litre.

[c] acute LD_{50} was calculated by administering a single oral dose.

tadpoles for a maximum of 96 h, and was found to be 3-8 times more toxic than DDT. The pattern of pesticide poisoning progressed through irritability and loss of equilibrium to death. Tadpoles were affected irreversibly by concentrations well below their calculated short-term LC_{50} values and, therefore, would succumb to DDT over time. DDT was re-tested several times during over the period of the study in an attempt to identify any development of resistance in the population. None was found; the 24-h LC_{50} values were stable throughout a 4-month period.

Cooke (1970) exposed tadpoles of the common frog *(Rana temporaria)* to 0.1, 1.0, or 10 mg DDT/litre for only one hour and reported a period of uncoordinated hyperactivity beginning less than one hour after the end of the exposure period. Body weight decreased during this hyperactive period and development was restricted in some of the tadpoles. Smaller tadpoles were more vulnerable to the effects of DDT than larger ones. In a later study (Cooke, 1979b), the same author reared tadpoles of the common frog at two different densities. The densities differed 5-fold and resulted in a 2-fold average difference in body weight between the two groups. The larger tadpoles, reared at the lower density, were completely tolerant of concentrations of DDT that caused severe sublethal effects in smaller tadpoles. Field populations of tadpoles included individuals with weights corresponding to the two experimental groups, but these were at the two extremes of the natural weight range.

Cooke (1972) exposed both spawn and tadpoles of the common frog *(Rana temporaria)*, the common toad *(Bufo bufo)*, and the smooth newt *(Triturus vulgaris)* for 24 and 48 hours to concentrations of DDT between 0.8 µg/litre and 0.5 mg/litre. Results indicated that DDT did not penetrate well-developed spawn and was only detectable in tadpoles hatched from spawn that had been treated with DDT immediately after it had been laid. Tadpoles hatching from spawn treated when newly laid showed hyperactivity, symptomatic of DDT poisoning, only later in their development at the point where external gills were lost. In the experiments where tadpoles were exposed to DDT, they were most susceptible either just before or just after the development of hindlimb buds. At these two stages, the characteristic hyperactivity was shown when DDT tissue concentrations reached between 2-3 mg/kg before the tadpoles developed limb buds, and when they reached 3-4 mg/kg, immediately after the tadpoles developed limb buds. During resorption of the tail, small frogs, but not small toads, were susceptible to DDT residues that had been acquired during larval development. At all stages of development, toads were more resistant to DDT than were frogs, and some toad tadpoles survived despite tissue residues in excess of 300 mg/kg. The metabolite DDE was often detectable in newt tadpoles and in frog and toad tadpoles with hindlimbs.

DDT has an anatomical effect on developing frog tadpoles (Cooke, 1970; Osborn et al., 1981). Exposure of tadpoles to 0.1 mg DDT/litre for 2 days or to 0.1, 1.0, or 10 mg/litre for one hour produced some individuals with abnormalities in the snout. A detailed histological

and behavioural study suggested that the effect was caused by two separate factors. DDT had a direct effect on the development of skin glands in the region above the upper mandible. The uncoordinated hyperactivity that followed DDT treatment caused the lower mandible to strike the upper, distorted mandible and resulted in further damage. Some individuals recovered from this abnormality at various stages of development. However, froglets that were affected at the tadpole stage frequently have blunt snouts and deformed brains. The authors suggested that DDT caused the disruption by preventing the organisation of the epithelial cells into gland units, possibly by affecting cell membranes and disrupting cell-to-cell communication. The mechanism of recovery remained unclear and a full explanation of the very specific nature of the abnormality was not possible.

This toxicity of DDT to amphibians is of significance in its use as an insecticide. The use of DDT to control mosquito larvae has been a major source of exposure of tadpoles and has led to toxic effects (Mulla, 1963; Cooke, 1973a).

The widespread use of DDT has led to the development of some resistance in two species of cricket frog *(Acris crepitans* and *Acris gryllus)*. Boyd et al. (1963) found that cricket frogs collected from areas of high DDT usage for the control of cotton pests were more tolerant to DDT than were frogs from other areas.

6. TOXICITY TO TERRESTRIAL ORGANISMS

There is evidence that DDT and its metabolites have affected wildlife in terrestrial ecosystems. Laboratory studies covered in this section give clear indication of a variety of lethal and sublethal effects. The range of organisms studied is not comprehensive. No review has been made here of the effects of DDT on insects, the target organisms. The lethal effect of DDT on insects is thought to result from changes in nerve transmission.

6.1 Terrestrial Invertebrates

Appraisal

There have been few reports on the effects of DDT and its metabolites on non-target terrestrial invertebrates.

Earthworms are insensitive to the acutely toxic effects of these compounds at levels higher than those likely to be found in the environment. The uptake of DDT by earthworms is related to the concentrations in soil and to the activity of the worms; seasonally greater activity increases uptake. Thus, although earthworms are unlikely to be seriously affected by DDT, they pose a major hazard to predators because of the residues they can tolerate.

Both DDT and DDE are classified as being relatively non-toxic to honey bees, with a topical LD_{50} at 27 µg/bee.

There are no reports on laboratory studies using DDE or TDE, in spite of the fact that these are major contaminants of soil.

The toxicity of DDT to insects, the target organisms, is extensively documented. Uptake of DDT and its metabolism by other terrestrial invertebrates is also well covered in the literature. However, there are few reports of effects of either DDT or its metabolites on non-target invertebrates.

Johansen (1962) classified DDT as "moderately" toxic to honey bees in both laboratory and field tests. Atkins et al. (1973) quoted a topical LD_{50} for honey bees of 12.09 µg/bee and classified DDT as "relatively non-toxic".

DDT has little or no effect on earthworms at dose levels likely to be encountered in the field; worms were unaffected by 2000 mg/kg soil (Goffart, 1949). The early literature has been examined by Davey (1963), whose review includes reports on a variety of earthworm species that live in surface soil or deeper layers. Thompson (1971) treated an area of grassland with an emulsifiable concentrate of DDT at the rate of 5.6 kg/ha. Although there was a reduction in earthworm numbers and biomass of about 30%, the author considered this to be of little significance. Results in tropical areas are similar to those of temperate regions. Cook et al. (1980) examined the effects of cultivation and DDT treatment on earthworm activity and populations in Nigeria

following the application of DDT (1 kg/ha) as a foliar spray on cowpea plots. The number of casts on the surface was reduced by DDT application, but there was no effect on the number of worms in the soil.

Cooke & Pollard (1973) treated Roman snails *(Helix pomatia)* with *p,p'*-DDT applied to lettuce leaves. The snails were fed a 365 x 2.5 cm square of leaf that had been treated with 0.1 ml of an acetone solution of DDT (either 0.025, 1.0, or 40 mg/ml). The dosing started when the snails were 2 weeks old and continued for 17 weeks, at which point the dose was doubled and continued for a further 12 weeks. The snails were then transferred outside to stimulate hibernation. Low doses of DDT reduced the weight of the shell and operculum whereas higher doses did not. After re-emergence from hibernation, the incidence of operculum eating was significantly higher among snails hibernating late in the season, and as exposure to DDT increased so operculum eating became more prevalent. The authors suggest that shell-thinning is likely to have occurred in snails in heavily-treated agricultural areas if the response of all snail species to DDT is similar to that of *Helix pomatia*.

Critchley et al. (1980) investigated the effects of the use of DDT for 4 years on a cultivated forest soil in Nigeria on the numbers of epigeal (surface-living) and subterranean species of invertebrates. DDT was applied as a foliar spray to crops of cowpeas at a rate of 1 kg/ha annually. After the first application of DDT there was no effect on ant or millipede numbers but the numbers of lycosid spiders and crickets were reduced. At the end of the study, after four applications of DDT, ants and millipedes were also reduced in number.

When Shires (1985) treated cereals on clay loam soil in experimental plots with DDT (1 kg/ha), the numbers of predatory beetles (Carabidae) were reduced by 50% one week after application. However, the numbers increased again after 4 to 6 weeks and remained at control levels. The use of other insecticides led to a second decrease in Carabidae numbers; this was attributed by the authors to a reduction in the food supply of aphids. DDT failed to control the aphids, which were tolerant to the compound.

6.2 Birds

Appraisal

DDT and its metabolites can lower the reproductive rate of birds by causing eggshell thinning (which leads to egg breakage) and by causing embryo deaths. However, different groups of birds vary greatly in their sensitivity to these chemicals; predatory birds are extremely sensitive and, in the wild, often show marked shell thinning, whilst gallinaceous birds are relatively insensitive. Because of the difficulties of breeding birds of prey in captivity, most of the experimental work has been done with insensitive species, which have often shown little or no shell thinning. The few studies on more

sensitive species have shown shell thinning at levels similar to those found in the wild. The lowest dietary concentration of DDT reported to cause shell thinning experimentally was 0.6 mg/kg for the black duck. The mechanism of shell thinning is not fully understood.

6.2.1 Short-term and long-term toxicity to birds

DDT and its derivatives DDE and TDE have moderate to low toxicity to birds when given as an acute oral dose or in the diet. The acute oral and dietary toxicities of DDT, DDE, and TDE to birds are summarized in Table 6.

These compounds have been studied in a wide variety of species in tests ranging from a single acute dose to 100 days of dietary dosing. All three compounds, DDT, DDE, and TDE, have low to moderate toxicity to young and adult birds. There is no obvious pattern of relative toxicity between the three compounds. In some species it is DDT that is the most toxic, while in other species it is TDE. Most of these laboratory tests have been conducted on species that are easy to maintain and breed in captivity. These species are unusual in many respects; they tend to be gallinaceous birds with young that are not fed by the adults after hatching. They also tend to have long breeding seasons untypical of most birds in the wild. In the wild, the most severely affected species of birds are raptors at the top of food chains. There is little direct laboratory data on toxicity to these birds. Toxicity to small songbirds, which make up the majority of bird species, has not been examined either in the laboratory or the field.

Porter & Wiemeyer (1972) fed American kestrels on a diet containing *p,p* -DDE at a concentration of 2.8 mg/kg. Two birds died after 14 and 16 months of treatment; they showed residues of DDE in brain tissues of 212 and 301 mg/kg, respectively. This compared with mean residues of 14.9 (range: 4.47-26.6) mg/kg in 11 adult males sacrificed after 12-16 months on the diet. Van Velzen et al. (1972) investigated the lethal effect of stored DDT mobilization by brown-headed cowbirds. Cowbirds were fed for 13 days on a diet containing 100, 200, or 300 mg *p,p* -DDT/kg, and were then given reduced rations of approximately 43% of normal daily intake for a 6-day period. Of 30 birds dosed, 21 died (6, 7, and 8 from the three dose levels, respectively). After 4 months, the remaining birds were subjected to a second period of 6 days on a reduced diet. Four more birds, out of six, died. In a second experiment, cowbirds were fed 100 mg *p,p* -DDT/kg diet for 13 days and then subjected to 4 days of reduced food intake. Seven out of 20 birds died. There were no deaths in any of the control groups (i.e., birds dosed but not starved, undosed and starved, or undosed and unstarved).

6.2.2 Toxicity to birds'eggs

Dunachie & Fletcher (1969) injected chicken eggs with DDT or TDE to give concentrations, in the egg, varying between 10 and 500 mg/kg.

Table 6. Toxicity of DDT and its derivatives to birds

Species	Sex[a]	Age	Route[b]	Compound	Purity[c] (%)	Parameter	Concentration (mg/kg)	Reference
Bobwhite quail		23 days	diet	DDE	99.9	5-day LC_{50}	825 (697-976)	Hill et al. (1975)
(Colinus virginianus)		23 days	diet	DDT	100	5-day LC_{50}	611 (514-724)	Hill et al. (1975)
		23 days	diet	TDE	TG	5-day LC_{50}	2178 (1835-2584)	Hill et al. (1975)
		young	diet	DDT		5-day LC_{50}	881 (796-975)	Stickel & Heath (1964)
(wild)			diet	DDT	TG	5-day LC_{50}	1170 (830-1650)	Hill et al. (1971)
(farm-reared)			diet	DDT	TG	5-day LC_{50}	1610 (1331-1948)	Hill et al. (1971)
		young	diet	DDT		10-day LC_{50}	1000	DeWitt et al. (1963)
		young	diet	DDT		100-day LC_{50}	400	DeWitt et al. (1963)
		adult	diet	DDT		10-day LC_{50}	2500	DeWitt et al. (1963)
		adult	diet	DDT		100-day LC_{50}	1000	DeWitt et al. (1963)
Japanese quail		7 days	diet	DDE	99.9	5-day LC_{50}	1355 (1111-1648)	Hill et al. (1975)
(Coturnix coturnix japonica)		7 days	diet	DDT	100	5-day LC_{50}	568 (470-687)	Hill et al. (1975)
		7 days	diet	TDE	TG	5-day LC_{50}	3165 (2534-3978)	Hill et al. (1975)
	M	2 months	oral	DDT	77.2	acute LD_{50}	841 (607-1170)	Hudson et al. (1984)
California quail	M	6 months	oral	DDT	TG	acute LD_{50}	595 (430-825)	Hudson et al. (1984)
(Callipepla californica)	F	6 months	oral	TDE	> 95	acute LD_{50}	> 760	Hudson et al. (1984)
Mallard duck		17 days	diet	DDE	99.9	5-day LC_{50}	3572 (2811-4669)	Hill et al. (1975)
(Anas platyrhynchos)		17 days	diet	DDT	100	5-day LC_{50}	1869 (1500-2372)	Hill et al. (1975)
		17 days	diet	TDE	TG	5-day LC_{50}	4814 (3451-7054)	Hill et al. (1975)
	F	3 months	oral	DDT	77.2	acute LD_{50}	> 2240	Hudson et al. (1984)
	F	3 months	oral	TDE	> 95	acute LD_{50}	> 2000	Hudson et al. (1984)
		young	diet	DDT		5-day LC_{50}	875 (650-1140)	Stickel & Heath (1964)
		young	diet	DDT		10-day LC_{50}	500	DeWitt et al. (1963)
		young	diet	DDT		100-day LC_{50}	> 200	DeWitt et al. (1963)
		adult	diet	DDT		100-day LC_{50}	1000	DeWitt et al. (1963)

Table 6. (Contd).

Pheasant		10 days	diet	DDE	99.9	5-day LC_{50}	829 (746-922)	Hill et al. (1975)
(*Phasianus colchicus*)		21 days	diet	DDT	100	5-day LC_{50}	311 (256-374)	Hill et al. (1975)
		10 days	diet	TDE	TG	5-day LC_{50}	445 (402-494)	Hill et al. (1975)
	F	3-4 months	oral	DDT	> 99	acute LD_{50}	1334 (894-1990)	Hudson et al. (1984)
	F	3-4 months	oral	TDE	> 95	acute LD_{50}	386 (270-551)	Hudson et al. (1984)
		young	diet	DDT		5-day LC_{50}	804 (686-942)	Stickel & Heath (1964)
		young	diet	DDT		10-day LC_{50}	1000	DeWitt et al. (1963)
		young	diet	DDT		100-day LC_{50}	100	DeWitt et al. (1963)
		adult	diet	DDT		10-day LC_{50}	1000	DeWitt et al. (1963)
		adult	diet	DDT		100-day LC_{50}	> 100	DeWitt et al. (1963)
Red-winged blackbird			diet	DDT		10-day LC_{50}	1000	DeWitt et al. (1963)
(*Agelaius phoeniceus*)			diet	DDT		30-day LC_{50}	500	DeWitt et al. (1963)
Cardinal			diet	DDT	TG	5-day LC_{50}	535 (420-700)	Hill et al. (1971)
(*Richmondena cardinalis*)								
House sparrow			diet	DDT	TG	5-day LC_{50}	415 (370-465)	Hill et al. (1971)
(*Passer domesticus*)								
Blue jay			diet	DDT	TG	5-day LC_{50}	415 (320-540)	Hill et al. (1971)
(*Cyanocitta cristata*)								
Rock dove	M,F		oral	DDT	77.2	acute LD_{50}	> 4000	Hudson et al. (1984)
(*Columba livia*)								
Sandhill crane	M,F	adult	oral	DDT	> 99	acute LD_{50}	> 1200	Hudson et al. (1984)
(*Grus canadensis*)								
Clapper rail	M		diet	DDT		5-day LC_{50}	1612	Van Velzen & Kreitzer (1975)
(*Rallus longirostris*)	F		diet	DDT		5-day LC_{50}	1896	Van Velzen & Kreitzer (1975)

[a] M = male; F = female.
[b] oral = acute oral test (result expressed as mg/kg body weight); diet = dietary test (result expressed as mg/kg diet).
[c] TG = Technical grade.

Two different vehicles were used to dissolve the insecticides (corn oil and acetone), controls being injected with vehicle alone. The authors monitored egg hatchability and survival of chicks to 4 days of age. Some chicks were fed and some were not. No dose of DDT, applied in either vehicle, had any significant effect on egg hatchability when compared to controls. However, there was a profound effect on the chick survival rate. All chicks hatched from eggs treated with DDT at 100 mg/kg, and which were not fed, were dead within 4 days after hatching. Feeding the chicks eliminated this effect; the survival rate of fed young was similar to that of controls. Chicks hatched from eggs treated with 50 mg DDT/kg survived as well as controls, whether they were fed or not. TDE was found to affect hatchability, but only when applied in corn oil; the acetone-dissolved material did not have any significant effect. TDE dissolved in corn oil reduced hatchability to 60% of control levels at 100 and 200 mg/kg, to 7% at 300 and 400 mg/kg, and to 0% at 500 mg/kg. The effects on chick survivability were similar to those of DDT. All chicks hatched from eggs treated with 100 mg TDE/kg were dead after 4 days if they were not fed, whereas chicks from eggs treated with 50 mg/kg survived as well as controls. Chicks from either 100 or 200 mg/kg treatments survived as well as controls as long as they were fed. The significance of the different vehicles was discussed by Cooke (1971) and Gilman et al. (1978). Acetone causes coagulation of yolk protein whereas corn oil allows the injected organochlorine to float through the yolk to a position directly under the blastodisc.

6.2.3 Reproductive effects on birds

DDT, or more specifically its metabolite DDE, causes the shells of birds' eggs to be thinner than normal. Results on eggshell thinning are summarized in Table 7. There is considerable variation between species for this effect. Galliform species are very resistant to shell thinning whereas birds of prey are particularly susceptible.

Lincer (1975) dosed captive American kestrels and established a clear relationship between dietary DDE and thinning of eggshells. There was a similar close correlation between the residues of DDE in individual eggs and the degree of shell thinning. The kestrels were fed with day-old cockerels (which were injected with 0.2 ml of corn oil, containing the DDE, into the breast muscle) and received either 0.3, 3, 6, or 10 mg DDE/kg diet. Residues of DDE in eggs laid by the birds correlated closely with dietary DDE concentration; residues of 1.9 mg/kg wet weight were associated with the lowest dose and 245 mg/kg with the highest dose given. There was no shell thinning associated with the dose of 0.3 mg/kg. The other doses showed 15.1%, 22.8%, and 29.2% thinning (at 3, 6, and 10 mg/kg, respectively). There was a straight-line relationship between the degree of shell thinning and the logarithm of the DDE residue in the egg. Data obtained from the field showed exactly the same trend (Fig. 1). This represents the best evidence for the effect of DDE on shell thickness in a species actually adversely affected in the field.

Table 7. Thinning effects of DDT and its derivatives on bird egg shells

Species	Route	Compound[c]	Dose (mg/kg)	Percentage change	Significance[e] (p)	Reference
Ring dove	diet	DDE	10	- 9.2	0.01	Peakall et al. (1973)
(Streptopelia risoria)	diet	DDE	40	- 6.8	0.01	Haegele & Hudson (1973)
Mallard	diet	TDE	10	- 2.6	NS	Heath et al. (1969)
(Anas platyrhynchos)	diet	TDE	10	- 5.4	NS	Heath et al. (1969)
	diet	TDE	40	- 2.6	NS	Heath et al. (1969)
	diet	TDE	40	- 5.4	NS	Heath et al. (1969)
	diet	DDT	2.5	- 5.3	NS	Heath et al. (1969)
	diet	DDT	10	- 7.9	NS	Heath et al. (1969)
	diet	DDT	40/25	-13.2	0.01	Heath et al. (1969)
White pekin duck	diet	DDE	40	-20.3	0.001	Peakall et al. (1973)
4 day	diet	DDE	40	- 3.3	0.01	Miller et al. (1976)
1-3 months	diet	DDE	40	-18.2	0.01	Miller et al. (1976)
Black duck	diet	DDE	10	-17.6	0.01	Longcore et al. (1971)
(Anas rubripes)	diet	DDE	30	-23.5	0.01	Longcore et al. (1971)
Screech owl *(Otus asio)*	diet	DDE	2.8	-13.3	0.01	McLane & Hall (1972)
American kestrel	diet	DDE	3	-15.2	0.05	Peakall et al. (1973)
(Falco sparverius)	diet	DDE	6	-21.0	0.01	Peakall et al. (1973)
	diet	DDE	10	-26.3	0.001	Peakall et al. (1973)
	diet	DDE	2.8	- 8.7	0.001	Wiemeyer & Porter (1970)
	diet	DDE	0.3	+ 2.1	NS	Lincer (1975)
	diet	DDE	3	-15.1	0.05	Lincer (1975)
	diet	DDE	6	-22.8	0.01	Lincer (1975)
	diet	DDE	10	-29.2	0.001	Lincer (1975)

Table 7. (Contd).

Species	Route	Compound[c]	Dose (mg/kg)	Percentage change	Significance[e] (p)	Reference
Japanese quail	diet	o,p'-DDT	100	- 4.0	0.001[d]	Bitman et al. (1969)
(*Coturnix coturnix*	diet	DDT	100	- 5.6	0.001[d]	Bitman et al. (1969)
japonica)	diet	DDT	100	0	NS	Cecil et al. (1971)
	diet	DDE	100	- 2.5	NS	Cecil et al. (1971)
	diet	DDE	2	+ 1.9	NS	Davison et al. (1976)
	diet	DDE	10	+ 6.3	NS	Davison et al. (1976)
	diet	DDE	40	+ 5.0	NS	Davison et al. (1976)
	diet	DDE	200	- 0.6	NS	Davison et al. (1976)
strain 1[a]	diet	DDT	2.5	+ 1.0	NS	Davison et al. (1976)
	diet	DDT	10	- 1.5	NS	Davison et al. (1976)
	diet	DDT	40	- 0.5	NS	Davison et al. (1976)
strain 1[b]	diet	DDT	2.5	- 2.7	NS	Davison et al. (1976)
	diet	DDT	10	- 1.6	NS	Davison et al. (1976)
	diet	DDT	40	- 7.1	NS	Davison et al. (1976)
strain 2[a]	diet	DDT	2.5	+ 0.5	NS	Davison et al. (1976)
	diet	DDT	10	+ 1.6	NS	Davison et al. (1976)
	diet	DDT	40	+ 1.0	NS	Davison et al. (1976)
strain 2[b]	diet	DDT	2.5	- 3.7	NS	Davison et al. (1976)
	diet	DDT	10	- 2.6	NS	Davison et al. (1976)
	diet	DDT	40	- 5.7	NS	Davison et al. (1976)

[a] Individually caged.
[b] Caged in pairs.
[c] DDT in the *p,p'*- form unless stated otherwise.
[d] Low calcium diet (0.56%).
[e] NS = not significant.

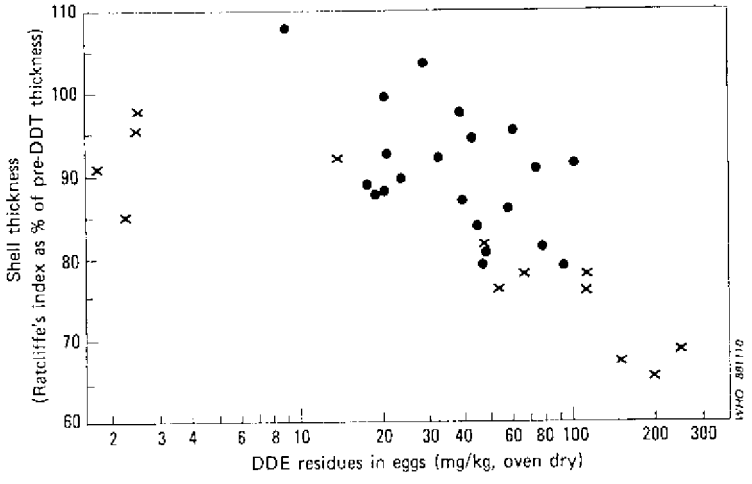


Fig. 1: Relationship between mean clutch shell thickness and DDE residue of American kestrel eggs collected in Ithaca, New York during 1970 (•) and the same relationship experimentally induced with dietary DDE (x). From: Lincer (1975).

Haegele & Tucker (1974) dosed egg-laying Japanese quail with a single oral dose of *p,p'*-DDE, *o,p'*-DDT, *p,p'*-DDT, or technical DDT, all at 125 mg/kg body weight. None of the treatments caused appreciable eggshell thinning. When Smith et al. (1969) fed Japanese quail with DDT at 100, 200, or 400 mg/kg diet, the two lower doses had no effect on hatchability or fertility of eggs laid. At 400 mg/kg, there was 50% mortality amongst dosed birds; survivors showed a decline in hatchability and fertility after 30 days. Bitman et al. (1969) dosed Japanese quail with *o,p'*-DDT or *p,p'*-DDT at a dietary level of 100 mg/kg. The quail were given a low calcium diet (0.56%) and were, therefore, under calcium stress during egg laying. Both isomers of DDT caused significant thinning of eggshells ($P<0.001$) and a significant ($P<0.01$) reduction in shell calcium content. Eggs produced by birds dosed with the *p,p'* isomer were significantly lighter than those laid by birds dosed with the *o,p'* isomer.

Cecil et al. (1971) investigated the effects of *p,p'*-DDT and *p,p'*-DDE on the egg production and eggshell characteristics of Japanese quail receiving an adequate calcium diet, and compared their results with previous studies of the effects of these compounds on quail receiving low calcium diets. They found a delay in the onset of egg production in quail fed a concentration of 100 mg/kg of either DDT

or DDE for about 3 weeks. This result was similar to that of studies with low calcium diets. In contrast to the earlier studies, there was no effect of either DDT or DDE on shell thickness or egg weight when dietary calcium was higher. There was an increased incidence of egg breakage in birds fed DDT and DDE, but this was less pronounced than with the low calcium diets.

Robson et al. (1976) studied the effects of DDE and DDT fed to Japanese quail in two different diets containing adequate or low calcium. DDT was fed at 100 mg/kg diet, whereas DDE was given at 0, 199, or 300 mg/kg diet, and the two calcium levels were 0.5% and 3%. DDE at 300 mg/kg was detrimental to adult body weight, fertility, and survivability. There was no effect of either DDT or of DDE at up to 100 mg/kg diet on adult body weight, food consumption, egg production, egg weight, fertility, hatchability, cracking of eggs, or eggshell thickness. Low dietary calcium had the effect of reducing the thickness of eggshells, increasing the incidence of cracked shells and decreasing egg production and hatchability.

Davison et al. (1976) fed DDE (0, 2, 10, 40, or 200 mg/kg diet) to female Japanese quail that were individually caged and had 14 g of food available each day. There was no effect on body weight, egg laying, egg weight, eggshell thickness, or on shell calcium content. Quail were then fed a diet containing DDT at 0, 2.5, 10, or 40 mg/kg. There was no effect on eggshell thickness, number of eggs laid, fertility, or hatchability. Quail fed 40 mg DDT/kg diet and caged in pairs, broke more eggs than birds fed lower concentrations of DDT or any concentration when the birds were caged individually. Paired quail laid fewer eggs than single quail and in one experiment they laid eggs with thinner shells.

When Davison & Sell (1972) dosed white leghorn hens with 100 or 200 mg DDT/kg diet for 12 weeks, the average egg production per bird, egg weight, dry shell weight, shell thickness, and shell calcium were all found to be unaffected by DDT at either dose level.

Egg-laying mallard ducks treated by Haegele & Tucker (1974) with a single oral *p,p'*-DDE dose of 500, 1000, or 2000 mg/kg body weight showed a clear effect on eggshell thickness at all dose levels. Unfortunately, whilst the results are clear, no statistical analysis of the results was presented. The effect on eggshells was dose related, quick acting, and persistent. Heath et al. (1969) dosed mallard for two seasons with DDE or TDE at 10 or 40 mg/kg diet and with DDT at 2.5, 10, or 40 mg/kg diet. The highest dose of DDT was reduced to 25 mg/kg in the second season. DDE at both concentrations severely impaired reproductive success, a more rapid initiation of the effect being seen with the higher dose. DDE significantly affected eggshell thickness; eggs from birds dosed with 40 mg/kg laid, in their second season, eggs with shells 13% thinner than controls. There was a significant increase in egg cracking and decrease in egg hatchability at both DDE dose levels. TDE did not have a significant effect on shell thickness. It impaired reproductive success, but not as severely as did DDE. DDT induced eggshell thinning at a dose of 25 mg/kg, shells being 18%

thinner than controls, and reduced duckling survival during 14 days post-hatch by 35%. DDT at 2.5 and 10 mg/kg had no effect.

Vangilder & Peterle (1980) fed mallard a diet containing 10 mg DDE/kg, and brought the birds into breeding condition using long daylength. Relative to controls, egg laying was delayed, eggshell thickness was decreased, and hatchability was reduced in treated birds. Ducklings, hatched from eggs laid by treated females, showed a significantly reduced survival time, and a greater proportion of ducklings were unable to initiate normal body temperature regulation.

When Longcore et al. (1971) dosed black ducks with 10 or 30 mg DDE/kg diet, there was significant eggshell thinning and an increase in shell cracking, compared to controls, at both dose levels. The survival of ducklings to 21 days was also significantly reduced at both dose levels. Longcore & Stendell (1977) fed DDE (10 mg/kg diet) to black ducks over two breeding seasons and then untreated food for a further 2 years. The eggshells of treated birds during dosing were 20% thinner than controls. When dosing stopped, eggshell thickness gradually increased but shells were still 10% thinner than controls 2 years after dosing had finished. Similarly, there was still a reduced survival of ducklings, to 3 weeks of age, 2 years after dosing with DDE had ceased.

Peakall et al. (1973) studied the effects of dietary DDE on eggshell thinning in three species of bird (white pekin duck, American kestrel, and ringdove). In addition to shell thinning, they reported a reduced rate of water loss from eggs laid by DDE-treated birds; the permeability constants of the eggs were significantly decreased. Scanning electron micrographs revealed a decrease in the number of pores per unit shell area and an increase in the number of globular inclusions in eggshells from treated birds. Greenburg et al. (1979) showed, also using scanning electron microscopy, that DDE affected both organic and inorganic constituents of the eggshells of mallard dosed in their diet. The literature concerning the effects of DDE on eggshell structure has been reviewed in detail by Cooke (1973b).

In studies by Miller et al. (1976), laying white pekin ducks and white leghorn hens were dosed with 40 mg DDE/kg diet. The ducks showed significant eggshell thinning within 4 days, and again between 1 and 3 months of the start of dosing, but the hens did not show significant eggshell effects within 2 weeks.

Peakall et al. (1975a) dosed white pekin ducks at a dietary level of 250 mg DDE/kg for 10 days, and, approximately 2 months later, started to collect eggs and measure shell thickness for a period of 27 weeks. At the beginning of the collection period, shells from treated birds were found to be 20% thinner than controls. Recovery was slow and shells were still 10% thinner at the end of the study. Haseltine et al. (1974) dosed mallard and pheasant (10 mg DDE/kg diet) and ring doves (40 mg DDE/kg diet) and found significant eggshell thinning and depression of serum calcium levels in both mallard and ring dove. However, neither parameter was significantly changed in pheasant. Peakall et al. (1975b) maintained paired ring doves on a diet

containing 100 mg DDE/kg for 3 weeks and white pekin ducks on 250 mg DDE/kg diet for 10 days. Although both species showed significant eggshell thinning, there was no significant difference between the levels of serum calcium of treated and control birds.

Miller et al. (1976) removed the shell glands from white pekin ducks and white leghorn chickens, dosed with 40 mg DDE/kg diet, when a calcifying egg was present within the gland, and assessed enzymatic activity. There was a significant decrease in Ca^{2+}-ATPase and carbonic anhydrase activities in the shell glands removed from dosed ducks, but no difference from controls in chicken shell glands. Kolaja (1977) maintained mallard ducks on a diet containing either DDT, DDE, DDT sulphonate, or DDE sulphonate at dose levels of 10 or 50 mg/kg. Eggs were collected for 30 days and were weighed and measured. There was no significant difference between egg weights at the different dose levels. The thickness of eggshells of birds fed DDE was significantly reduced. Ducks fed DDT laid eggs with significantly thinner shells only after day 14. The two sulphonate-treated groups were not significantly different from each other and were only significantly different from controls on day 18; eggshell weights followed a similar pattern.

Mendenhall et al. (1983) dosed breeding barn owls with 3 mg DDE/kg diet during two breeding seasons, and found that treated birds laid thin-shelled eggs and laid significantly more eggs per pair in both seasons. In both years the percentage of eggs broken was increased, relative to controls, and the mean number of eggs hatched and young fledged per pair was reduced. There was a significant increase in embryo deaths in one of the two years.

Eggshell thickness has been monitored in different ways by different authors. Some direct measurement has been made with membranes intact and some without. Other methods have been used to compare recent eggs with museum specimens, which could not be broken to measure thickness directly. The various methods were reviewed by Cooke (1973b), who suggested standards. Generally a log-linear relationship between DDE load and shell thinning is claimed. In a recent consideration of the theoretical treatment of such data, Moriarty et al. (1986) suggested that the main methods of assessing shell thickness do not adequately take into consideration the effects of shell size and shape. This does not detract from the conclusion that shell thinning occurs, but suggests that the relationship may be more properly described as curvilinear.

6.2.4 Reproductive hormones and behaviour

After feeding mallard a diet contaminated with 3 mg *p,p'*-DDE/kg and artificially incubating eggs laid by the females, Heinz (1976) found that the average egg residue of DDE was 5.8 mg/kg. Ducklings from treated eggs were hyperresponsive to a tape-recorded maternal call; treated ducklings were significantly more likely to approach the recorder. In contrast, treated ducklings moved shorter distances away

from a frightening stimulus, compared to controls. Japanese quail chicks fed a diet containing 50 mg DDE/kg for 8 days, starting at 7 days of age, and then a clean diet for a further 6 days showed no significant effect on avoidance response to a moving silhouette.

Haegele & Hudson (1977) paired ring doves for 12.5 min each day, for 5 days, prior to dosing their diet with 10 or 50 mg *p,p'*-DDE/kg. The birds were also paired between days 31 and 35 and between days 59 and 63 after the start of dosing. Two measures of the courtship behaviour of males were made: total courtship activity time and mean bow-coo frequency. Bow-cooing behaviour is the initial behaviour displayed by males to attract females. In control birds, the total courtship activity time was 25% (days 31-35) and 23% (days 59-63) longer than it was in the predose period. In birds dosed with 10 mg DDE/kg, the courtship activity between days 31 and 35 was not different from that in the predose period, whereas the final pairing produced a decrease of 55% in activity. In birds dosed with 50 mg DDE/kg, the courtship activity decreased by 30% and 67% for the two later pairing periods compared to the predose period. After dosing at 10 mg/kg, there was no change in bow-cooing between days 31 and 35, but a reduction of 53% between days 59 and 63. Birds dosed at 50 mg/kg showed decreases in bow-cooing behaviour of 43% and 84%, in the two subsequent pairings respectively, when compared to the predose period.

When Richie & Peterle (1979) paired ring doves and fed them with either 10 or 40 mg *p,p'*-DDE/kg diet, there was a significant delay in the period between pairing and egg laying at both dose levels. Leutinizing harmone levels in blood plasma, sampled throughout the experiment, were not significantly altered by the DDE. Similarly, Jefferies (1967) reported an increase in the time between pairing and egg laying in Bengalese finches fed a range of doses of *p,p'*-DDT between 75 and 1200 mg/kg diet. Treated birds were fed for 2 h/day, immediately following a period of 1 h of starvation. There was a significant correlation between DDT intake by the female and the delay in egg laying. Dobson (1981) measured circulating hormone levels and nest-building behaviour in pigeons dosed orally with DDE and found a delay in egg laying. Hormone measurements showed that ovulation was not delayed. Nest building was reduced in treated birds. The delay in egg laying resulted from a lengthening of the period between ovulation and oviposition. Since the laying of eggs is dependent on the stimulus of adequate nest material, this lengthening of the period between pairing and egg laying was considered to be primarily an indirect effect on reproduction, triggered by a direct effect on behaviour; the egg was retained longer in the oviduct.

Peakall (1970) maintained ring doves on a diet containing 10 mg *p,p'*-DDT/kg for 3 weeks. They were kept in isolation (with short daylengths) and then paired (with long daylengths) to induce breeding. The females were killed either 8 days after pairing or after completion of their clutch of two eggs. In those killed 8 days after pairing, circulating oestradiol levels were significantly reduced and hepatic enzyme activity was significantly increased.

There was a significant delay in the laying of the first egg and a decrease in egg weight. In the same study, the birds were given oral ^{45}Ca (7.4×10^4 Bq; 2 μCi) on the day before pairing. There was a significant decrease in the radioactivity of eggs and in the bones of females killed 8 days after pairing. In a separate experiment, *p,p'*-DDT was injected intraperitoneally at a dose of 150 mg/kg body weight, into female ring doves within 1 day of their first egg being laid. The birds were killed after completing their clutch of two eggs. The shell weight of the second egg was significantly reduced when compared to the first and there was a significant decrease in carbonic anhydrase activity in the oviduct. This enzyme is associated with deposition of calcium into the shell.

6.2.5 Reproductive effects on the male

Burlington & Lindeman (1950) administered a daily subcutaneous injection of DDT to male white leghorn chicks, gradually increasing the dose from 15 to 300 mg/kg body weight. The birds were treated for 60-89 days, and the cockerels were killed and their testes removed, weighed, and sectioned. Treated birds were found to have smaller testes, more intertubular tissue, and retarded tubular development. These effects were accompanied by an inhibition of testosterone-dependent secondary sexual characters; combs and wattles were reduced in both size and colour development in treated birds. Locke et al. (1966) dosed male bald eagles at dietary levels of 10 mg DDT/kg for 60 or 120 days, and found no effects on spermatogenic activity. There were some degenerative effects on the testis, but only at doses which had severe neurological effects and ultimately led to death.

6.2.6 Effects on the thyroid and adrenal glands in birds

When Jefferies & French (1972) fed pigeons on a diet containing either 18, 36, or 72 mg *p,p'*-DDE/kg for a period of 56 days, paired thyroid weights were found to be greater in treated birds than in controls. There was no apparent dose relationship to this effect, but bird numbers were small. Taking the dosed birds as a single group, the results were significantly different from those of control birds. Liver weights were similarly increased, and, at the two highest dose levels, there was an increase in paired adrenal weights.

Biessman & von Faber (1981) dosed Japanese quail for 9 weeks with technical DDT (either 50 or 250 mg/kg diet) or for 5 weeks with *p,p'*-DDT or *p,p'*-DDE (250 and 300 mg/kg, respectively). Adrenal weights increased with all treatments but the increase in size was only significant for the 300 mg DDE/kg dose. The percentage of cortical tissue, measured from areas of sections of the gland, showed a similar trend, but results were not statistically significant. No changes were detectable in nuclear size of either cortical or medullary cells. Lehman et al. (1974) studied the effect of technical grade DDT on the adrenal glands of bobwhite quail, which were maintained on a diet

containing 10, 50, or 150 mg DDT/kg for 242 days and then killed. No effect was found on adrenal weight expressed as a percentage of body weight, but there was a significant dose-related increase in the ratio between areas of cortex and medulla.

6.2.7 Special studies in birds

Dieter (1974) fed Japanese quail on a diet containing 5, 25, or 100 mg DDE/kg and, after 12 weeks of dosing, assessed the activity of five plasma enzymes (creatine kinase, aspartate aminotransferase, lactate dehydrogenase, cholinesterase, and fructose-diphosphate aldolase). There was an increase in the activity of all these enzymes, which, in each case, was proportional to the logarithm of the DDE dose.

Bend et al. (1977) dosed immature puffins, orally by intubation, with DDE at 6 mg/day (equivalent to 50 mg/kg diet) for 16 to 21 days, and, after killing the birds, determined the effect of DDE on hepatic mixed-function oxidases. Both aniline hydroxylase and benzphetamine demethylase activities were increased in treated birds; the yield of microsomal protein remained unchanged. In contrast, Sell et al. (1972) demonstrated a depression in aniline hydroxylase and N-demethylase activities after feeding Japanese quail with DDT at 200 mg/kg diet. Both DDT and DDE inhibited aniline hydroxylase in vitro activity, when present at concentrations of 10^{-7} mol/litre or more.

Bunyan et al. (1970) measured the activities of glucose-6-phosphate dehydrogenase (G-6-P) and 6-phosphogluconate dehydrogenase (6-P-G) in the liver of Japanese quail fed diets containing low levels of *p,p'*-DDT or a number of saturated and unsaturated analogues of *p,p'*-DDT. Generally, saturated compounds lowered G-6-P levels and increased 6-P-G levels. *p,p'*-DDMU was anomalous in elevating G-6-P. The authors suggested that these effects might be due to interference with protein metabolism primarily by the unsaturated analogues and metabolites of DDT. Bunyan et al. (1972) fed either DDT or DDE to Japanese quail and monitored hepatic microsomal protein, cytochrome P450, aniline hydroxylase, aromatic nitroreductase, phenylbenzoate esterase, and total vitamin C. Changes in these factors were more readily explained in terms of residues of DDE in the liver than in terms of dietary dose. DDE was found to be a more potent inducer of microsomal protein, cytochrome P450, and aniline hydroxylase than was DDT. The effects of DDT could be explained in terms of the effects of the DDE produced by DDT metabolism. Aromatic nitroreductase was unaffected by either compound. Vitamin C levels were raised by DDT more than by DDE. Phenylbenzoate esterase showed a biphasic response following the feeding of DDE. Bunyan & Page (1973) extended these studies by examining the effects of DDE and DDMU on hepatic microsomal enzyme systems. Most of the changes observed in quail were greater with DDMU than with any other DDT metabolite. The authors suggested that DDT metabolism in birds may be different to metabolism in mammals.

Metabolism probably gives rise, via the production of DDMU, to a highly active liver inducer.

Heinz et al. (1980) fed ring doves on a diet containing 2, 20, or 200 mg DDE/kg for 8 weeks, and found at the end of the dosing period, a significant decrease in dopamine concentration in brain tissue from birds fed on the two higher doses. Brain noradrenalin concentration was also affected but only at the highest dose. There was a significant, negative correlation between concentration of both dopamine and noradrenalin and the residue of DDE in the brain tissue.

Friend et al. (1973) fed a dietary dose of 10, 100, or 1000 mg DDE/kg. to male mallard that had been previously maintained on either fresh water or 1% salt water. Birds were given a concentrated salt solution either 1, 3, 6, or 9 days after the beginning of DDE treatment, the salt being administered both intraperitoneally (12 ml of a 10% solution) and intravenously (3ml of a 5% solution). The rate of sodium chloride excretion was not reduced, relative to controls, in DDE-treated birds maintained previously on salt water, but was reduced significantly in DDE-treated birds not previously given salt.

When Mahoney (1975) fed caged white-throated sparrows on technical DDT (either 5 or 25 mg/kg), the onset of spring nocturnal migratory restlessness *(Zugunruhe)* and weight increase was delayed by at least 1 week. Although *Zugunruhe* onset was delayed, when migratory nocturnal activity did commence it was more pronounced than in control birds. The increase in *Zugunruhe* was related to body residues of DDT.

Haynes (1972) dosed male bobwhite quail with DDT (100 mg/kg diet) for 10 weeks and, 1 week before the study was terminated, some birds were transferred to clean food while others were starved for 4 days and then given clean food for 3 days before being killed. There was no significant effect on liver glycogen, either from dosing with DDT or from starvation, but liver lipid levels were significantly increased by both DDT and starvation. Body lipid levels were not significantly affected by DDT but were reduced after starvation.

6.2.8 Synergism with other compounds in birds

Kreitzer & Spann (1973), in a study on combined effects of pesticides, found that mixtures of DDT and dieldrin in Japanese quail, and DDE and Ceresan M (organomercury fungicide) in pheasants, were additive rather than synergistic in their action. The study compared known LD_{50} values with expected ones. Mallard, maintained on a diet containing a mixture of DDE (40 mg/kg) and Aroclor 1254 (40 mg/kg) for at least 30 days, laid eggs with significantly thinner shells than did controls. This result was not significantly different from that produced by DDE alone (Risebrough & Anderson, 1975). In a similar study on American kestrels, Lincer (1972) dosed the birds with Aroclor 1254 (10 mg/kg) and DDE (3 mg/kg) in the diet, both separately and in combination. There was no eggshell thinning with Aroclor alone, but Aroclor and DDE together had a significantly greater effect on shell thickness than DDE alone, indicating synergism.

Japanese quail exposed to dietary doses of 5 or 50 mg DDE/kg for 12 weeks, and subsequently dosed orally with either parathion or paraoxon at 2 µl/g body weight, showed synergism between the compounds with respect to mortality and to inhibition of brain cholinesterase. The synergistic action of DDE on cholinesterase inhibition was apparent 3 days after exposure to 50 mg/kg and one week after exposure to 5 mg/kg. Mortality due to DDE was increased from 10% to 90% in the presence of the organophosphorus compounds. Anticholinesterase effects were increased by 50% in the presence of DDE (Ludke, 1977).

6.3 Non-laboratory Mammals

Appraisal

Experimental work suggests that some species, notably bats, may have been affected by DDT and its metabolites. Species which show marked seasonal cycles in fat content are most vulnerable, but few experimental studies on such species have been made. In contrast to the situation in birds, where the main effect of DDT is on reproduction, the main known effect in mammals is to increase the mortality of migrating adults. The lowest acute dose which kills American big brown bats is 20 mg/kg. Bats collected from the wild (and containing residues of DDE in fat) die after experimental starvation which simulates loss of fat during migration.

In studies into the effect of DDE on bats, Geluso et al. (1976) captured young Mexican free-tailed bats *(Tadarida brasiliensis)* before their first migratory flight and transferred them to the laboratory. This species migrates north from Mexico to the USA in spring and returns to winter in the south. Three groups of bats were used. A reference set was killed on capture and, when the bats were analysed for residues of organochlorines derived from environmental source, DDE was the only chemical found in significant amounts. Brain residues of DDE were low; the median being 3.7 (range: 1.5 to 17.0) mg/kg in eight younger animals and 1.3 (range 1.1 to 11.0) mg/kg in older animals. Two further groups were maintained in the laboratory where the bats were given water but not fed. One group was regularly exercised, while the other was given no exercise. All exercised bats died within 9 days; 4 bats in the unexercised group died and the other 4 were killed after 9 days. Analysis of brain DDE residues showed considerably elevated levels compared to the reference group. For the unexercised bats, the median residue values were 47 (range 18 to 76) mg/kg in younger animals and 70 (range 10 to 95) mg/kg in older animals. In exercised bats, the values were 160 (range 66 to 330) mg/kg for younger animals and 160 (range 37 to 260) mg/kg for older animals. Those animals that died before the end of the study showed symptoms characteristic of pesticide poisoning, including hyperactivity, intermittent audiogenic seizures, and violent contractions of chest muscles. The high brain residues of DDE were considered to be the cause of death of

the animals. It should be noted that these animals had not been artificially dosed with DDE. The effects resulted from residues of DDT in body fat, taken up in the maternity roost. The authors considered that their studies confirmed the suggestion that bats were being killed by accumulated residues of DDE during the period of migration, when their fat reserves were used up.

Clark & Kroll (1977) experimentally fed adult females of the same species of bat *(Tadarida brasiliensis)* for 40 days with mealworms containing 107 mg DDE/kg and then killed four of the bats. They had a whole body burden of 2.345-2.929 mg DDE (and 78-90 mg DDE/kg in the brain). Twelve of the dosed bats were then starved, and they died within 8 days. The total body burden of DDE ranged from 1.952 to 3.711 mg DDE and brain residues from 379 to 564 mg/kg. These brain residues were considered to be diagnostic of death from DDE poisoning. Tremors characteristic of poisoning were seen in the bats before death occurred.

The toxicity of single oral doses of DDT to bats has been estimated in two studies. Jefferies (1972) derived an approximate LD_{50} of 63 mg/kg body weight for the pipistrelle bat *(Pipistrellus pipistrellus)*, a small British species. There was no mortality at doses below 45 mg/kg and 100% mortality at doses above 95 mg/kg. Luckens & Davis (1964) found that the lowest dose which killed American big brown bats *(Eptesicus fuscus)* was 20 mg/kg and that 40 mg/kg was invariably lethal. The LD_{50} for this species lies somewhere between 25 and 40 mg/kg for a single oral dose.

Blus (1978) determined dietary LC_{50} values of DDT, given in food either as a powder or dissolved in oil, for short-tailed shrews *(Blarina brevicauda)* of different ages and sex. In 2-week tests, the range of LC_{50}s for DDT dissolved in oil was 651 to 1160 mg/kg diet, and for DDT added as powder it was 839 to >2552 mg/kg. The influence of age and sex was sometimes more important in determining DDT toxicity than was body weight, though heavier shrews tended to be more tolerant of the chemical. Among older animals, males were more tolerant of DDT than females. Braham & Neal (1974) found an effect of DDT on the metabolic rate of the same species of shrew after feeding it with earthworms contaminated with the insecticide. After one week of this diet, the metabolic rate was significantly higher than that of undosed shrews, but after 2-3 weeks of dosing there was a return to oxygen consumption rates not different from controls. Two shrews were fasted for 18 h, after being fed earthworms containing DDT for 3 weeks, and compared to untreated shrews similarly fasted. The DDT-treated animals showed 12.6% and 12.1% increases in metabolic rate after fasting, whereas controls showed decreases of 8.7% and 8.0%. The DDT exposure was environmentally realistic because earthworms used for feeding were not artificially dosed with DDT but were collected from an area where DDT had been used.

7. ECOLOGICAL EFFECTS FROM FIELD APPLICATION

There have been kills of fish (Hunt & Linn, 1970) and aquatic invertebrates (Ide, 1957) reported after normal usage of DDT as a terrestrial insecticide and after its application to water for mosquito control. Reproductive failure in commercial fisheries has also been attributed to DDT (Hunt & Linn, 1970). In addition, it has been shown to be toxic to amphibia after water application (section 5.3). The setting of safe water levels of DDT and its metabolites is difficult because its high bioaccumulation and high lipid solubility mean that it can have effects remote in time from its application. The toxicity of DDT to aquatic microorganisms and invertebrates is very variable between species. Exposure to DDT or its stable metabolites would, therefore, be expected to kill certain species selectively. Short-term, there is close correspondence between the 96-h LC_{50} for a moderately sensitive fish (16 µg/litre) and the expected water concentration after application of DDT at the normal rate.

DDT and its metabolites, principally DDE, have been implicated in reproductive effects on birds in the field. Large population declines in some bird species, mainly birds of prey, have been blamed on DDT or on combinations of DDT with other persistent organochlorines. The evidence for this rests on correlations. There is a correlation in time between the onset of effects on eggshells and the onset of major DDT use in agriculture. There is also a correlation between geographical areas of high DDT use and effects on local populations of birds (compared to populations living in areas of low use). There is a clear correlation between DDE residues in eggs and the degree of thinning of the shells of those eggs, collected from the wild. Storage of DDT in body fat means that the effects of the compound can be remote in time from the application of the chemical to an area. Only some species of birds are affected by DDT or its metabolites. There are considerable data on the variability between species in their susceptibility to these compounds. Widespread monitoring programmes have related the recovery of bird populations to reduced levels of DDE and the residual material of aldrin/dieldrin use in the tissues of birds sampled from the wild, following attempts to limit or ban the use of the parent pesticide in agriculture. Because DDT is seldom the only chemical residue found in bird tissues from the wild, there is some disagreement on whether DDT alone can cause population declines in birds.

Ratcliffe (1967, 1969, 1970), Hickey & Anderson (1968), and Anderson & Hickey (1972, 1974) were the first, in Britain and North America, respectively, to compare the thickness of eggshells sampled from the wild with that of specimens measured from museums and private collections which predated the use of DDT. These authors examined a wide range of bird species but mainly those high in food chains. Later studies, along the same lines, include those of Dilworth et al. (1972) on the woodcock, Wiemeyer et al. (1975) on the osprey, Fox (1976) on

the common tern, Cooke et al. (1976) on the grey heron, and Koeman et al. (1972) and Newton & Haas (1984) on the sparrowhawk. Ratcliffe (1970) collected eggshell data on 17 species of British birds, 9 of which showed significant decreases in shell thickness when comparing the period before 1947 with the period after 1947. The birds affected were predominantly raptors, exceptions being the carrion crow, rook, and shag. Anderson & Hickey made eggshell comparisons between pre- and post-DDT use on 25 different species of birds. The same species from different geographical areas of North America were investigated, making 166 comparisons in all. Of these, 62% showed significant decreases, 37% showed non-significant decreases or no change, and only 1% showed an increase in shell thickness.

King et al. (1978) found significant decreases in eggshell thickness in 15 out of 22 aquatic species of birds, in Texas, USA, when comparing shells from 1970 with museum specimens from before 1943. All of these studies, and many more, demonstrated that, in those species that showed effects on eggshell thinning, the effect began suddenly and markedly at the same time as the onset of DDT use. In Britain, the use of DDT in large quantities began in 1947. Fig. 2 reproduces the data (from Newton & Haas, 1984) on sparrowhawks from 1870 to 1980. The persistence of DDT in bird tissues means that recovery is still not complete, despite controls on the use of DDT. In Alaska, populations of peregrine falcons did not show the effects of DDT until much later than other regions of North America. These birds breed in Alaska, where use of DDT was low, but winter in Central and South America. Residues of DDT and its metabolites in Alaskan peregrines began to rise in 1967, along with the use of DDT in its wintering grounds. Concomitant reductions in breeding success and populations of peregrines occurred (White & Cade, 1977). These data are indicative of a bird breeding and survival effect of DDT use, correlated both with time and geographical area. The index of eggshell thickness has reflected the pattern of use of the insecticide (Ratcliffe, 1970). Before 1947, there was no significant geographical variation in the mean thickness of peregrine falcon eggshells in Britain. Since 1947, eggshells from non-agricultural areas, notably the central and eastern Scottish highlands, have shown a smaller decrease in shell thickness than shells from highly agricultural regions.

Anderson & Hickey (1974) showed that shells of the white-tailed eagle in Greenland were thicker than shells of the same species collected in the Baltic. Compared to early reference shells from museums, the Greenland shells showed a slight increase in thickness of 3%, whereas Swedish shells showed a decrease of 16%.

Lincer (1975) established a dose relationship between dietary DDE and eggshell thinning in captive American kestrels and, also, a relationship between DDE residues in the eggs and the thickness of their shells. He then compared shell thickness with egg DDE residue in kestrels sampled from the wild. The relationship was identical. Many other authors have shown a good correlation between egg DDE residues and the degree of eggshell thinning. These studies cover the

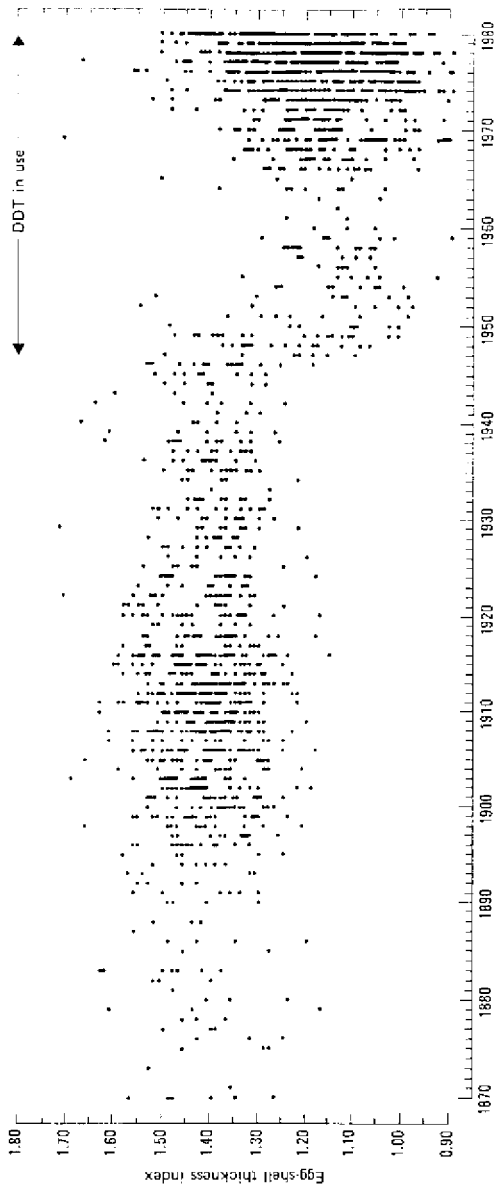


Fig. 2: Shell thickness index of British sparrowhawks, 1870-1980. Shells become thin abruptly from 1947, coincident with the widespread introduction of DDT in agriculture. Each spot represents the mean shell-index of a clutch, and more than 1000 clutches have been represented from all regions of Britain. Shells were made available for measurement from museums and private collections. From: Newton & Hass (1984).

following species: double-crested cormorant (Anderson et al., 1969); great blue heron (Vermeer & Reynolds, 1970); prairie falcon (Enderson & Berger, 1970); peregrine falcon (Peakall et al., 1975c); grey heron (Cooke et al., 1976); sparrowhawk (Newton & Bogan, 1978); and gannet, shag, and great black-backed gull (Cooke, 1979a). In many of these studies, there is not only a correlation between eggshell thickness and DDE but there are also correlations between DDE residues and residues of other organochlorines. Therefore, it is often difficult to determine solely from the field data, exactly which chemical is responsible for the effect. This problem has been addressed by Newton & Bogan (1978). They conducted a statistical analysis of their data that showed a correlation between DDE and shell thickness, egg breakage, egg addling, and hatching failure, in addition to a correlation between DDT, PCB, and dieldrin residues. After multivariate analysis, DDE appeared only to be responsible for eggshell thinning and egg breakage. Relating laboratory studies to field observations suggests that DDE is the only organochlorine that causes eggshell thinning.

Population declines in birds of prey differed between much of North America and eastern North America and western Europe. In North America, apart from in the East, declines were gradual, whereas in Europe and eastern North America declines were sudden and catastrophic. The sudden declines in Europe have usually been attributed to the use of the chlorinated cyclodienes, which kill adult birds, rather than to DDT. A study of the recoveries of European birds of prey populations provides evidence for this attribution. Populations began to rise at a time when residues of DDE in tissues were stable but when use of the cyclodienes and, therefore, residues of HEOD (dieldrin) were declining. Some populations in North America did not show high contamination with cyclodienes and may have declined due to DDT use alone. Henny (1972) showed that in American populations of osprey, American kestrel, and red-shouldered hawk there was a decrease in breeding performance, but no increase in adult mortality, in response to DDT. The reproductive effects of DDT may have prevented population recoveries after the cessation of dieldrin use and the return of mortality rates to normal. The question has been reviewed by Newton (1979) and Newton & Haas (1984).

The populations of many species of birds of prey were monitored throughout a period of high DDT use. This was done by large scale surveys and studies of population dynamics (Ratcliffe, 1972; Henny, 1977; Lindberg, 1977; White & Cade, 1977), migration counts at observation points (Rosen, 1966; Hackman & Henny, 1971; Edelstam, 1972; Ulfstrand et al., 1974; Nagy, 1977), and by sample counts (Ash, 1965; Bezzel, 1969). Some species showed marked declines (in some areas this led to local extinction), whilst others showed only temporary effects or no effects at all. Declines were most marked in bird-eating species, such as the sparrowhawk and peregrine falcon, and fish-eating species, such as the white-tailed and bald eagles, and were less marked in mammal-eating species, such as the kestrel, golden eagle, and buzzard. These variations in decline correspond to the DDE levels found in these particular species (Newton, 1979).

Perfect (1980) reported the results of a 4-year study on the overall effects of the use of DDT as an insecticide on cowpeas crops in a Nigerian forest soil. In addition to effects on soil invertebrates (section 6.1), there were effects on the decomposition of plant material. The remains of the plants after harvesting were ploughed into the soil and this resulted in an increase in the residues of DDT and its metabolites in lower levels of the soil. To confirm an effect on decomposition, these plant remains were buried in mesh bags and the loss of weight due to decomposition was recorded over time. There was a significant reduction in the rate of decomposition of plant material treated with DDT and also of untreated plant material buried in contaminated soil. Shires (1985) reported no significant effect on the decomposition of sweet chestnut leaf litter in a temperate area after the application of DDT at 1 kg/ha.

Perfect et al. (1979) investigated the effects of repeated DDT applications on cowpea crop yield in Nigeria. Yields varied considerably from season to season and from year to year in untreated plots because of differences in pest damage and climate. DDT was applied to the treated plots weekly between planting and harvest at a rate of 1 kg/ha, and the site was studied for 4 years. Over the 4-year period there was a considerable benefit in yield from DDT application; the yield was 1.45 tonnes/ha in the untreated and 3.42 tonnes/ha in the treated plots. However, the benefit was most noticeable in the first year of cultivation and declined over the four years to the point where DDT use did not significantly increase yield. The authors attributed the effect to the deleterious action of the insecticide on soil biota.

8. EVALUATION

In evaluating the environmental hazard of DDT and its metabolites the following general points have to be kept in mind.

(a) The environmental distribution and effects of DDT are spread wider than the area of use, because the parent compound or its metabolites are carried worldwide by air and ocean currents and in biota.
(b) Some of the breakdown products of DDT, principally DDE, are highly persistent in soil, sediment, and biota. Thus, problems with residues of these materials last long after the cessation of use.
(c) The bioaccumulation of DDT, or more usually of its metabolites, is well established and occurs from very low environmental concentrations of DDT. The use of "bioconcentration factors" (the ratio of concentration in the organism with concentration in the medium) to estimate the capacity of organisms to take up DDT can be misleading if the exposure is high, since these values are ratios.
(d) Residues and effects are often highly seasonal, corresponding to changes in body fat, since DDT metabolites are very lipid-soluble. Measurements of these metabolites in the tissues of organisms must be conducted over a period of time if they are to give any indication of the degree of contamination of the environment.
(e) There are insufficient data on the effects of DDT and its metabolites on communities of organisms and ecosystem functioning. Hazard assessment is, therefore, often made by extrapolation from single species studies.
(f) Research and monitoring have concentrated on a few effects of DDT observed in the wild. This could give the mistaken impression that the effects of these compounds are restricted to a few species. Other effects could be predicted but have received little or no attention from the scientific community.
(g) The major remaining use of DDT is for malaria control operations that are normally carried out in tropical countries. However, the majority of environmental studies on DDT have been carried out in conditions relevant to temperate regions. Care must be exercised in extrapolating these results to tropical conditions.

8.1 Aquatic Organisms

The widespread use of DDT as an insecticide has resulted in worldwide contamination of the environment. Due to the physicochemical characteristics of DDT and its metabolites, concentrations have been recorded in different environmental compartments, including soil, sediments, and terrestrial and aquatic organisms. The bioconcentration of DDT and its metabolites is a real hazard to non-target organisms. DDT and its metabolites cause adverse effects at all trophic levels of

aquatic ecosystems, particularly on primary producers, which are the most sensitive. Although no data are available for the effects of DDT on ecosystem function, it should be regarded as a major environmental hazard in this respect. DDT and its metabolites are highly toxic to fish and, besides their lethal effect, they affect development, behaviour, and biochemical processes. DDT and its metabolites, should be regarded as hazardous to fish productivity and distribution and, hence, to human food supplies. Accumulated DDT and its metabolites are further transferred from aquatic organisms to consumers, including birds, mammals, and, ultimately, human beings.

8.2 Terrestrial Organisms

DDT-type compounds are resistant to breakdown and are readily adsorbed onto soils and sediments, from whence they can act as long-term sources of exposure and contribute to terrestrial organisms. Accumulation in terrestrial organisms is via the food chain.

These chemicals are hazardous to microorganisms, but repeated application can lead to the development of tolerance in some species. DDT causes fluctuations in some populations of microorganisms, and this could eventually lead to changes in species composition, disruption of nutrient cycles, and changes in soil fertility.

Earthworms are insensitive to the acute toxic effects of DDT residues in soil. However, they are known to take up DDT from soil and this uptake presents a major hazard to predators.

DDT is a non-selective insecticide and leads to mortality in natural enemies of the insect pest. This results in impairment of the balance between predators and prey and leads to outbreaks of secondary pests and occurrence of the primary pest in larger numbers.

Laboratory studies confirm field findings that bat populations are adversely affected by DDE, especially during migration. These studies are indicative of the potential hazard to other mammals, exposed to DDT in the environment, when fat containing DDT residues is mobilized, e.g., during migration or temporary starvation.

One of the most widely studied effects of DDT is eggshell thinning in birds, particularly in predatory species. The metabolite DDE, not DDT, has been shown to be responsible for this effect. Other effects on reproduction and survival of birds have been demonstrated. Large population declines in birds of prey can be, at least partially, attributed to DDT. It has been shown that DDE residues in birds and their eggs reduced the rate of recovery of affected raptor populations. A factor that has received less attention is the secondary effect of the increasing numbers of pest rodents that were controlled principally by birds of prey in some countries.

* * *

Because of their lack of degradation, their resulting widespread persistence in the environment, their high acute toxicity to organisms

at the base of food chains, and their high potential for bioaccumulation, DDT and its metabolites should be regarded as a major hazard to the environment. DDT should not be used when an alternative insecticide is available.

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