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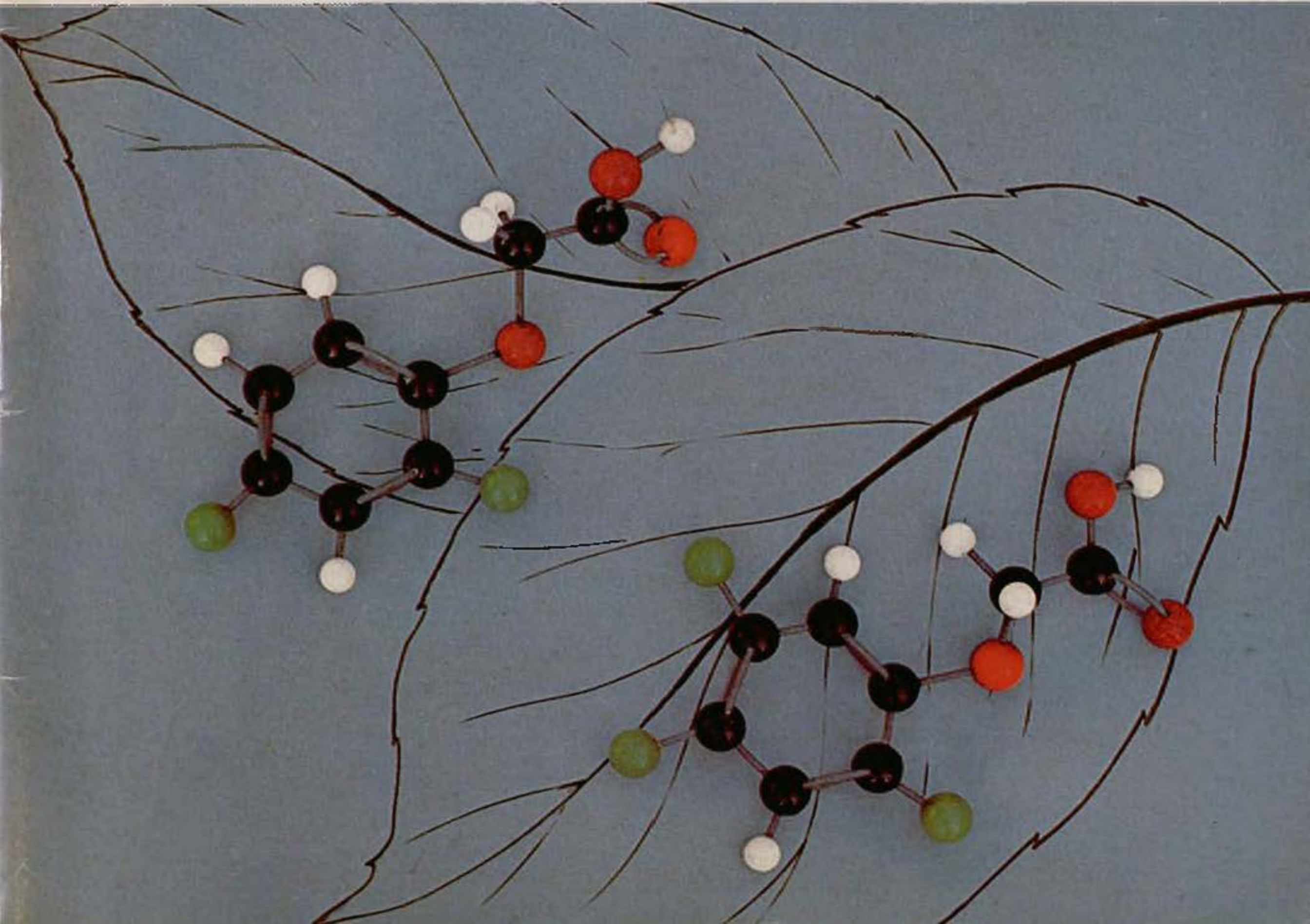
The Safety of the Herbicides 2,4-D and 2,4,5-T

D J Turner



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THE SAFETY OF THE HERBICIDES 2,4-D AND 2,4,5-T

D. J. TURNER, B.Sc., Ph. D.

Agricultural Research Council Weed Research Organization

LONDON: HER MAJESTY'S STATIONERY OFFICE

CONTENTS

		Page
SUMMARY		4
Chapter 1	**INTRODUCTION**	5
1	The historical background	5
2	2,4-D and 2,4,5-T as herbicides	5
3	2,4-D and 2,4,5-T as defoliants	7
4	The side effects of the defoliation programme	9
5	The present situation	9
6	Civil uses of defoliants	10
Chapter 2	**THE PROPERTIES, MANUFACTURE, MODE OF ACTION AND USES OF 2,4-D and 2,4,5-T**	10
1	Chemical and physical properties	10
2	The manufacture of 2,4-D and 2,4,5-T	14
3	Formulation ingredients	14
4	The mode of action of the herbicides	14
5	The choice of 2,4-D and 2,4,5-T formulations	16
6	The practical use of 2,4-D and 2,4,5-T for woody plant control	18
6.1	Overall foliage sprays	
6.2	Dormant shoot sprays	
6.3	Basal bark sprays	
6.4	Cut-bark and cut-stump treatments	19
Chapter 3	**THE EFFECTS OF 2,4-D AND 2,4,5-T ON DOMESTIC LIVESTOCK AND ON MAN**	20
1	Acute effects of 2,4-D and 2,4,5-T on animals	21
2	Chronic (subacute) effects of 2,4-D and 2,4,5-T on animals	22
3	The effects of 2,4-D and 2,4,5-T on humans	22
4	2,4-D and 2,4,5-T in animal products	23
5	2,4-D and 2,4,5-T residues in crops and fruit	23
6	Indirect effects of 2,4-D and 2,4,5-T on farm livestock	24
7	2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)	24
8	Teratogenic effects of 2,4-D and 2,4,5-T	26
Chapter 4	**THE EFFECTS OF 2,4-D AND 2,4,5-T ON OTHER LAND ORGANISMS AND THE PERSISTENCE OF THESE COMPOUNDS IN TERRESTRIAL ENVIRONMENTS**	27
1	The effects of 2,4-D and 2,4,5-T on higher plants	28
2	The persistence of 2,4-D and 2,4,5-T in plant material	28
3	The effects of 2,4-D and 2,4,5-T on wild animals and birds	29
4	The effects of 2,4-D and 2,4,5-T on bees	29
5	The effects of 2,4-D and 2,4,5-T on other insects	30
6	The effects of 2,4-D and 2,4,5-T on fungi and soil micro-organisms	30
7	The persistence of 2,4-D and 2,4,5-T in soil	31
8	The persistence and mobility of TCDD	32

		Page
Chapter 5	**THE ACTIVITY AND PERSISTENCE OF 2,4-D AND 2,4,5-T IN AQUATIC ENVIRONMENTS**	34
1	The effects of contaminated water on man and animals	34
2	The movement and persistence of 2,4-D and 2,4,5-T in water	34
3	Contamination of water by oils	36
4	The effects of 2,4-D and 2,4,5-T on aquatic plants	36
5	The effects of 2,4-D and 2,4,5-T on fish	37
6	The effects of 2,4-D and 2,4,5-T on other water animals	38
7	The incidence of TCDD in water	38
Chapter 6	**THE OVERALL PICTURE**	39
	BIBLIOGRAPHY	42
	GLOSSARY	54

Front Cover

Plate 1 – Molecular models of 2,4-D (top) and 2,4,5-T.

Carbon	– **black**,	Hydrogen	– **white**
Chlorine	– **green**,	Oxygen	– **red**

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THE SAFETY OF THE HERBICIDES 2,4-D AND 2,4,5-T

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SUMMARY

The properties, manufacture and uses of 2, 4-D and 2, 4, 5-T are described and the possible side effects of these herbicides are reviewed in detail. While concentrated preparations are moderately poisonous, the diluted solutions which are used for most weed control treatments present little direct hazard to humans and animals. In practice, foodstuffs and water supplies are unlikely to become contaminated. However if contamination should occur, serious consequences are unlikely. It is pointed out that (1) very large amounts of spray solution or contaminated material are needed to produce toxic effects, (2) the compounds are usually rejected by animals, because of their unpleasant taste and smell, (3) neither herbicide is a cumulative poison and (4) 2, 4-D and 2, 4, 5-T break down comparatively rapidly in plant or animal tissues, soil and natural water. At the concentrations which will be encountered in forests or on farmland, the herbicides have virtually no effect on most birds, fish, insects and soil living organisms. Populations of particular insects or soil organisms may sometimes be temporarily reduced but recovery is usually rapid.

Military uses of 2, 4-D and 2, 4, 5-T for defoliating forests and destroying crops present a greater potential hazard. However there is no evidence that the defoliation spraying programmes in Vietnam had any direct effects on humans or animals, even though large areas were treated repeatedly, at dose rates much above those used in agriculture and forestry.

While 2, 4-D and 2, 4, 5-T have little direct activity towards animals, they often have marked indirect effects. Changes in plant communities, whether or not they are caused by herbicides, are usually linked with changes in the populations of organisms which depend on particular species for food or shelter. It is often difficult to predict the full consequences of applying herbicides but unexpected indirect effects are always possible. Extensive programmes which involve spraying large areas present special risks.

In the past, some batches of 2, 4, 5-T contained a poisonous impurity, 2, 3, 7, 8-tetrachloridibenzo-p-dioxin (TCDD). This impurity not only has high acute toxicity but, in the laboratory, causes birth defects in unborn foetuses. Unlike 2, 4-D and 2, 4, 5-T, TCDD can have cumulative effects. It is eventually degraded in plant material or soil but by comparison with the herbicides is very persistent. Toxic effects due to TCDD have not been recorded in the field but the use of impure 2, 4, 5-T containing this dioxin is undoubtedly dangerous. All current production of 2, 4, 5-T is however now essentially free from the impurity.

Pure 2, 4, 5-T may also cause birth defects in mice and rats but only when administered in large doses, at particular stages of pregnancy. Many other commonly used materials, including many drugs, Vitamin A and caffeine have similar effects on laboratory rodents. Birth defects have not been observed in any other animals. However, if a similar order of activity in humans and livestock is assumed, the scaled-up doses are seen to be so large as to practically rule out accidental poisoning. As with acute effects, it would be hardly possible for a toxic dose of 2, 4, 5-T to be accidentally ingested from contaminated food or water.

CHAPTER 1

Introduction

1. The historical background

In one way or another, human activities have probably always been important factors determining the balance of species in forests and other ecological communities. Even where he has not directly attacked woodlands, man has often unintentionally affected their make up, for example by pasturing cattle and pigs or by introducing new organisms. Thus the introduction of *Rhododendron ponticum* from the Mediterranean in 1763 as an ornamental plant has led to this species becoming dominant in woodland on many acid sandy soils. *Rhododendron* hinders the regeneration of native trees[1] and is replacing at least one indigenous species, holly, in parts of Ireland.[2] Similarly, the introduction of the European Gipsy Moth has greatly altered the balance of species in forests of North America.[3] There are many other ways in which human activities can modify forest communities. In particular, during the past 80 years or so, man has discovered new methods of influencing plant growth by the use of chemicals. Chemical control of vegetation probably began with the use of salt, ashes, and similar materials on roads and paths. Towards the end of the 19th century, copper, arsenic, and iron salts came into use as "weedkillers." Around 1900, it was found that solutions of copper salts, ferrous sulphate, ammonium sulphate or sodium nitrate would often kill broadleaved weeds in cereals without seriously damaging the crop. Later, other chemicals were employed, including sodium chlorate, ammonium sulphamate and dinitrophenols.[4] By the mid 1930's studies with weedkillers or "herbicides" became a recognised branch of agricultural science. During this period, however, herbicides were little used in forests. Occasionally, materials such as sodium arsenate or ammonium sulphamate were employed for controlling unwanted growth but most herbicides then available were not particularly useful to the forester. Most of them showed little selectivity and were effective only at high doses, sometimes of the order of 100–200 kg per hectare.[5]

2. 2,4-D and 2,4,5-T as herbicides

During the 1940's it was found that certain chlorinated phenoxyacetic acid derivatives, including the herbicides now known as 2,4–D and MCPA, have remarkable herbicidal properties, including the ability to kill dicotyledonous plants without injuring grasses. The compounds were developed during wartime under conditions of great secrecy by workers in the U.S.A. and at Rothamsted and Jealotts Hill in Britain.[5] Although many related compounds have been tested, 2,4-D and MCPA are still the most important of the phenoxyacetic acid herbicides. A related compound, 2,4,5-T appeared a year or two later. While its effects generally resembled those of MCPA and 2,4-D, it was found to be more active against woody species.[7] 2,4,5-T is also effective for controlling bindweed wild roses and briars[6,8]. It was first sold commercially in 1944.

By contrast with previously available weedkillers the phenoxyacetic acids had at least three important advantages. First, they were active at relatively low doses; early reports mention the use of sprays containing only 500 to 2000 ppm of the compounds[9-10]. Secondly, unlike most earlier materials, the new compounds had little effect on grasses or conifers. Finally, the herbicides acted mostly through the foliage and persisted in the soil for a limited period so that land cleared by their use could quickly be replanted. These properties made the new compounds very useful to farmers and foresters alike. During the period immediately after World War II herbicide technology developed very rapidly. Thus, research summaries prepared for the 1946 and 1947 North Central Weed Control Conferences in the USA contained only fragmentary reports and observations of effects on trees and shrubs.[9,10] By 1948 there was considerable interest in the use of 2,4-D and 2,4,5-T for woody plant control. The claim was made that "we can now control all the woody plants normally found on rights of way in western Pennsylvania except... basswood, ash and beech".[11] The 1949 summary mentioned new types of formulation and application methods, including aerial spraying.[12] By 1950 the recommendations made for woody plant control were virtually those current today.[13] A reasonably complete picture of the effects of the herbicides on individual species was available and there was already a good understanding of the types of formulation best suited to each application technique.

The use of 2,4-D and 2,4,5-T in forests in North America increased steadily during the 1950's. Mixtures of both compounds were frequently used. Various alternative woody plant herbicides came into use during the 1950's and 1960's, including fenoprop (Silvex), related to 2,4,5-T. This compound is more active than its predecessors against American oaks.[14] However, 2,4-D, 2,4,5-T or mixtures of both herbicides usually gave adequate control of broadleaved species. In America the compounds were used extensively for "pine release", the reduction of competition from deciduous species in mixed stands containing conifers.[15 16 17 18] The herbicides were applied from aircraft, at rates which checked but did not kill the hardwoods. By the mid 1950's there was already a concept of using herbicides not just as weed killers but as tools to change the balance of species in forests. About this time, aerial applications of 2,4-D and 2,4,5-T began to be used for controlling unwanted woody growth on rangelands and along power line and other rights of way.[18 19]

Table 1 gives details of USA production of 2,4-D and 2,4,5-T during the period from 1946–1959.[20] Much more 2,4-D than 2,4,5-T was produced; unlike 2,4,5-T, 2,4-D has many agricultural uses. The production of both herbicides remained approximately constant from 1952 to 1959. The figures illustrate the rapid acceptance of herbicide usage in the USA

In Britain and in most European countries, the use of 2,4-D and MCPA in cereal crops was well established by the mid 1950's. However, European foresters adopted the use of the phenoxyacetic acid herbicides relatively slowly. Where herbicides were used, the practice was often to treat individual bushes or trees rather than whole forests, as in America. A report of heather control with 2,4-D and MCPA appeared in 1953.[21] Further papers dealing with the control of woody plants were published in 1956.[22 23] In this year for the first time the British Weed Control Council published tables showing the susceptibility of many trees and shrubs.[24] The introduced species *Rhododendron ponticum,* already mentioned, proved to be very resistant. To control this plant it was found necessary to use overall or bark sprays containing up to 20 kg (45lb) of 2, 4, 5-T in 450 litres (100 gals) of oil.[24 25]

Although the herbicides were adopted more slowly than in America, their use for woody weed control in Britain was fairly well established by the mid 1960's. The application methods used at this time have been described by Brown.[26] By comparison with the USA, herbicide usage in Britain was small. Table 2, from Brown's paper, shows the average weights of herbicide used by the Forestry Commission between 1964 and 1966. Much more 2,4,5-T than 2,4-D was employed. The materials were usually applied by knapsack sprayer By contrast, much of the material used in the USA (Table 1) was sprayed from the air. In Britain the use of aircraft is comparatively rare because of the small scale of forest plantation and the risks of

Table 1

USA production of 2,4-D and 2,4,5-T 1946 – 1959, in millions of pounds*

	1946	*1947*	*1948*	*1949*	*1950*	*1951*	*1952*
2,4-D	5	6	20	15	14.2	20.8	30.7
2,4,5-T	–	–	–	–	1.2	2.5	3.5

	1953	*1954*	*1955*	*1956*	*1957*	*1958*	*1959*
2,4-D	25.9	30.2	34.5	28.8	34.2	30.9	29.3
2,4,5-T	5.2	2.7	2.9	5.2	5.3	3.7	5.6

*Crafts and Robbins, 1962[20]

Table 2

Mean annual usage of herbicides by the Forestry Commission, 1964 - 1966*

	Acres treated	Active ingredient lb
2,4,5-T	5,800	19,000
2,4-D	300	1,000
2,4,5-T/2,4-D mixtures	700	2,000
Paraquat	2,800	2,000
Ammonium sulphamate	900	25,000
Dalapon	200	2,000

*Brown, 1969

contaminating surface water which is used for public water supplies.

While almost all the 2,4,5-T sold in Britain was used for woody plant control, small amounts were also employed against herbaceous weeds, particularly coltsfoot and nettle.[27] In tropical areas 2,4,5-T became important for weed control in rice[28] Additionally the herbicide came into use in Malaya as an agent for stimulating the flow of latex from rubber trees.[29]

3. 2,4-D and 2,4,5-T as defoliants

Both 2,4-D and 2,4,5-T can induce leaf shedding in a wide range of deciduous and evergreen woody species. While initially, during their wartime development, it was envisaged that the herbicides might be used for destroying crops, it was later realised that they might also provide a method of improving visibility in forests, so assisting military operations. There were also civil requirements for defoliants. In East Africa a large scale aerial spraying experiment was carried out in which 2,4,5-T was used to induce leaf fall. It was hoped that this would provide control of Tsetse fly *(Glossinia spp.)* An application of 2.2kg/ha (2 lb/ac) of the herbicide defoliated most of the woody species present in an isolated patch of forest near Lake Victoria. In many cases, 90% of the leaves were shed within 3-4 weeks of spraying. Many trees and shrubs were killed outright. Tsetse fly were not eliminated, but the trial demonstrated conclusively that 2,4,5-T could be an effective agent for reducing the shade canopy of tropical forests.

During the political emergency in Malaya in the 1950's, 2,4,5-T was used by the British Army to thin jungle cover near roads and so reduce the dangers of terrorist attack. The rates of chemicals which were employed are believed to have been around 2 - 4 lb. per acre (2.2 - 4.4 kg/ha). Osbourne[30] states that while complete defoliation was not achieved the operation was effective from the military point of view. By comparison with later operations by US forces in Vietnam, this first military use of herbicides was on a very limited scale.

Interest by US forces in military uses of defoliants and herbicides, which began with the wartime work on 2,4-D and 2,4,5-T, continued through the 1950's. Some 12,000 chemicals were screened as defoliants or agents for crop destruction at Fort Detrick, the US Defence Department's research station near Washington.[32] Some compounds were subsequently tested against tropical vegetation in Puerto Rico.[33] Despite the great effort put into the search for new defoliants, none of the materials had any real advantage over 2,4-D and 2,4,5-T. Large scale experiments with the undiluted butyl esters of these herbicides were carried out, initially near New York,[32] and later in South Vietnam. The tests showed conclusively that 2,4-D/2,4,5-T mixtures could be used to kill or defoliate most of the woody species present in Vietnam. Plans to use the new technique of defoliation as a military weapon were announced in January 1962. The first large scale application of mixed 2,4-D and 2,4,5-T n-butyl esters were made from the air, along a 70 mile highway leading to the US air force base at Bien Hoa.[32] This was the first occasion on which large scale aerial applications of the phenoxy-acetic acid herbicides were employed for military purposes.

Table 3 shows the quantities of defoliants which were used in Vietnam between 1965 and 1971. After a slow build up from 1962 to 1965, the extent of the military defoliation operation increased enormously, reaching its maximum in 1967. From the military aspect, there is no doubt that the programme was very successful. Defoliants containing 2,4-D and 2,4,5-T butyl esters were widely used throughout the war but increasing amounts of another herbicide, picloram, were employed during its later stages. A fourth compound, cacodylic acid, was important for crop destruction rather than defoliation. The military use of herbicides is now well documented, having been the subject of a lengthy report commissioned by the American National Academy of Sciences.[34]

It is worth emphasising the very large differences between normal agricultural or forestry uses of the herbicides and their role as defoliants in Vietnam. The recommended defoliation treatment was 3 gallons per acre (33 litres per hectare) of an undiluted mixture of equal volumes of n-butyl 2,4-D and 2,4,5-T esters. This mixture was given the military code name *'Orange'*. Expressed in terms of acid equivalents, the herbicide doses were 12 lb/acre (13 kg/ha) of 2,4-D and 11 lb/acre (12 kg/ha) of 2,4,5-T. These are between 5 and 10 times the amounts used for most summer foliage treatments in forestry.[35 36] Many areas of Vietnam are known to have been sprayed with *'Orange'* on several occasions.[34]

The butyl esters of 2,4-D and 2,4,5-T which were employed in Vietnam are not normally used in agriculture or forestry. Alternative formulations are discussed in Chapter 2 but at this stage it may be said that these esters used in *'Orange'* are much more volatile than those normally employed for weed control. They vaporize readily, particularly in tropical climates. Defoliation in Vietnam was probably caused as much by vapour effects as by direct contact of spray droplets with leaves. The high volatility of agent *'Orange'* partly accounts for the success of the defoliation programme.

Another major difference between civil uses of the herbicides and their use in Vietnam lies in the scale of application and the care taken to avoid drift during spraying. In agriculture and forestry

Table 3.

Use of 2,4-D/2,4,5-T during Vietnam War*

Year	*lb. acid equivalent used (x 1000)*	*Acreage sprayed (x 1000)*
1965	100	76
1966	1310	413
1967	2670	1028
1968	1860	660
1969	2740	1009
1970	470	184
1971	0	0

*National Academy of Sciences, USA, 1974[34] limited areas are sprayed and it is usually possible to avoid serious contamination of food crops or drinking water. In Vietnam, forests, villages, cultivated land and streams were often sprayed almost indiscriminately.[34] On occasions, when it was necessary to evade anti-aircraft fire, entire loads of around 1000 gallons of herbicide were sometimes "dumped" within a few seconds, without regard to effects on water supplies or food crops.[34]

4. The side effects of the defoliation programme

The possible consequences of the massive use of herbicides in Vietnam soon caused serious public concern. From 1968 onwards many articles on the military defoliation programme appeared in newspapers and scientific journals. Many scientists, including Galston,[38] Perry,[39] and Pfeiffer,[40] stressed that there was little previous experience of the effects of repeated and massive doses of 2,4-D and 2,4,5-T. It was pointed out that apart from their known effects on plants, the herbicides might have other serious and unforseen types of activity. During this period, a few scientists emphasised that both herbicides had been in continued use for almost 20 years, without ill effects on humans or animals.[41 42] However, public opinion in both America and Europe was strongly against the military use of herbicides. The controversy came to a head with the publication, in July 1968, of a statement by the influential American Association for the Advancement of Sciences "On the use of herbicides in Vietnam." The statement expressed concern about the possible side effects of the de-foliation programme and urged that a full scientific study should be carried out, possibly under United Nations supervision.

At about this time other pesticides began to receive a great deal of unfavourable publicity. In particular, attention was drawn to fatal accidents involving parathion and other organo-phosphorous insecticides, and to the persistence of organochlorine products such as DDT and dieldrin. A tendency arose for writers in newspapers and non-scientific journals to treat all pesticides as a more or less uniform class of compounds, all with equally dangerous and unpleasant properties. To some extent, this tendency still persists.

5. The present situation

The subsequent history of 2,4,5-T has been related by Davis[44] and Whiteside.[45] The outcry against the defoliation programme increased. Then, on October 29th 1969, without warning, a press release from a US Government Department announced that 2,4,5-T had been found to have teratogenic properties, that is, to cause developmental defects in unborn rat and mouse embryos. The studies which led to this press release had been carried out at a privately operated research establishment, the Bionetics Laboratory.[46] In a country already torn with controversy about the military use of herbicides and the Vietnam war, this announcement had devastating effects. All use of the defoliant mixture *'Orange'* was abruptly terminated. In April 1970, the US Government restricted the use of liquid formulations of 2,4,5-T in gardens or near water. Subsequently, all uses on food crops were prohibited.

The principal manufacturers of 2,4,5-T appealed against the Government action, and an Advisory Committee was set up to consider evidence about the herbicide. During hearings, from February 1971 to May 1972, two new facts emerged. Firstly, it transpired that the sample of 2,4,5-T which had been used in tests for teratogenicity contained about 30 parts per million of an extremely poisonous impurity, 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). Secondly, when purified 2,4,5-T was examined for teratogenicity, moderate doses of up to 40 mg per kilogramme of the material body weight had no effect in mice and rats. The Advisory Committee recommended that registration for normal uses of 2,4,5-T should be restored on condition that TCDD levels did not exceed 0.5 ppm.[44] This recommendation has not yet been acted upon by the United States Government.

In Britain no action was ever taken under the Pesticides Safety Precautions Scheme to restrict the use of 2,4,5-T. As has been shown, the use of the herbicide in Britain is small and almost confined to forestry. However, because of uncertainty about the facts, and following the US Government decisions, the Forestry Commission temporarily discontinued the use of 2,4,5-T during 1970 and 1971. Work with the herbicide was resumed after the Scientific sub-committee of the Pesticides Safety Precautions Scheme had reviewed the evidence and reported the facts of the 2,4,5-T case to the Minister for Agriculture. In a written reply to a parliamentary question on April 22nd, 1970, the Minister informed Members that "2,4,5-T ... has been in use here for ten years and no harmful effects have been shown to be associated with its use. I do not think I would be justified in imposing limitations on our current uses."

6. Civil uses of defoliants

This account of the history of 2,4-D and 2,4,5-T would be incomplete without a further brief mention of civil requirements for defoliants. There is still an urgent need for materials such as 2,4-D and 2,4,5-T which can reduce the shade cover of tropical forests and so restrict the movement and breeding of tsetse flies. A second requirement is for an agent which can defoliate rubber trees, in the event of South American leaf blight *Dothidella ulei* being introduced into important rubber producing areas. At present Malaya and the neighbouring rubber-growing countries are free from this very serious disease. If an outbreak should occur, it could only be contained and isolated by quickly defoliating all rubber trees in and around the affected area. 2,4,5-T ester is the most effective defoliant agent which is available at present. Stocks are maintained at strategic points throughout the main rubber growing areas. As yet, it has not been necessary to bring these emergency control measures into operation.

CHAPTER 2

The properties, manufacture, mode of action and use of 2,4-D and 2,4,5-T

1. Chemical and physical properties.

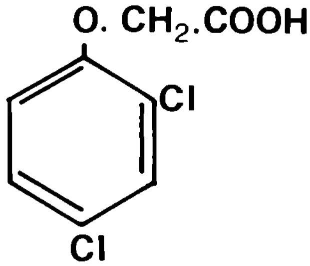
The chemical formulae of 2,4-dichlorophenoxyacetic and 2,4,5-trichlorophenoxyacetic acids are shown in Figure 1. Many related compounds have been made and tested but only those groups of phenoxy acids which are derived from acetic, propionic or butyric acids have strong herbicidal properties.[48] 2,4-D and 2,4,5-T are white crystalline solids, melting at 140°C and 153°C respectively[49]. Both are only slightly soluble in water, 2,4-D to a rather greater extent than 2,4,5-T[49]. This greater solubility may have an important bearing on differences in the behaviour of the herbicides. Normally, neither acid dissolves in oil, but oil-based solutions can, if necessary, be prepared by using a third material such as tributyl phosphate as a co-solvent[50]. Like other organic acids, 2,4-D and 2,4,5-T can form salts or esters. The physical and chemical properties of these derivatives are often markedly different from those of the free acids. However the herbicidal properties of esters and salts usually resemble those of the parent substances. In many ways, these derivatives are much more convenient to use than the acids. Although preparations containing 2,4-D and 2,4,5-T acids were formerly used commercially,[50 51] they are now uncommon.

The chemical structures of some typical salts are shown in Figure 2. The sodium and ammonium salts are easily made but are unfortunately only moderately soluble in water[49]. To obtain a salt with high water solubility the acids must be combined with another more complex type of base, an amine. The example shown in Figure 2 is the triethanolamine salt of 2,4,5-T but many other amine salts can be made and are used in practical herbicide formulations.

The structure and chemical preparation of typical butyl and octyl esters of 2,4,5-T, made by reacting 2,4,5-T acid with butyl or octyl alcohols, are shown in Figure 3. All simple esters of phenoxyacetic acids are non-polar compounds whose "oily" properties increase with the molecular weight of the alcohol.[49] Esters are insoluble in water but dissolve readily in petroleum oils. They are sometimes used without dilution or as "straight" oil solutions but are often mixed with an emulsifying agent so that they may be diluted with water and applied as an emulsion.

The butyl esters and other esters made by reacting 2,4-D and 2,4,5-T with alcohols of low molecular weight evaporate easily and so are liable to "drift" from sprayed areas as fumes or vapour.[52] Esters made with higher alcohols, such as octyl alcohols, are much less volatile and therefore more suitable for use in forests or on farms. Other examples of esters which are used in practical formulations are the butoxyethanol and

2,4-dichlorophenoxy acetic acid



2,4,5-trichlorophenoxy acetic acid

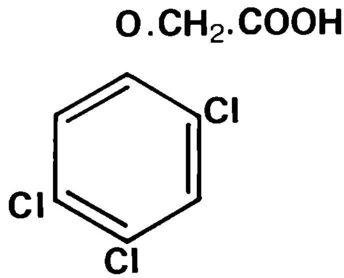


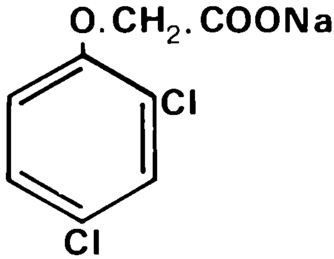
Figure 1 – Chemical structure of 2,4-D and 2,4,5-T.

propylene-glycol-butyl-ether (PGBE) derivatives of 2,4-D and 2,4,5-T.[48] [49] These particular esters are more commonly used in the USA than in Britain.

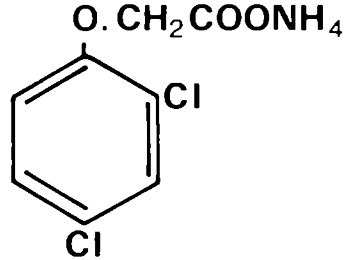
Apart from the variation in volatility, there are no major differences in the herbicidal activities of different esters. However, it is worth emphasising that only the phenoxyacetic acid part of an ester molecule (to the left of the vertical broken lines in Figure 3) confers herbicidal properties. The alcohol part of the molecule has no herbicidal activity. The concentration of phenoxyacetic herbicides is usually expressed in terms of "acid equivalents", i.e. the content of the parent acid, in grammes per litre of formulation.

Most of the 2,4-D and 2,4,5-T formulations which are available at present contain either water-soluble amine salts or oil-soluble esters. Another type of formulation, the oil-soluble amine salts, was quite widely used in the 1960's but is now largely neglected. These salts are made from long chain amines whose chemical structures contain lipophilic or "oil loving" groups. The lipophilic groups make the amines and also their salts, soluble in oil, to about the same extent as esters. The structure of a typical oil soluble amine and its 2,4,5-T salt are shown in Figure 4. As well as dissolving readily in oil, the products are slightly soluble in water. The point will be referred to again later.

2,4-D

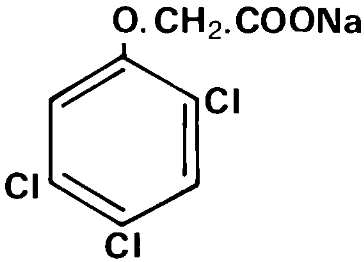


sodium salt

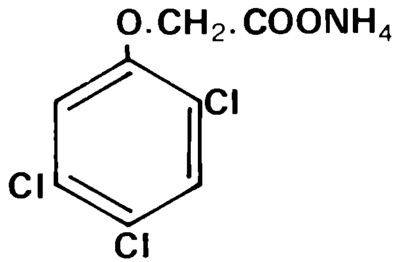


ammonium salt

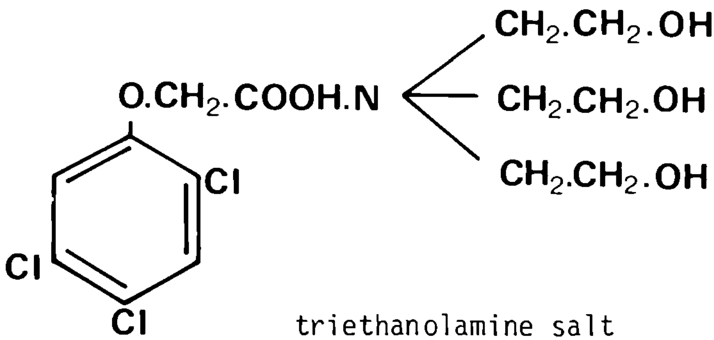
2,4,5-T



sodium salt

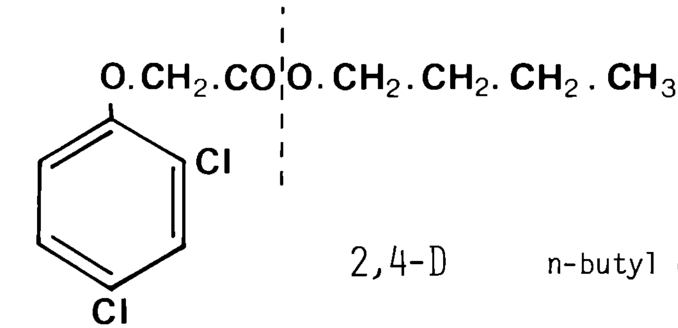


ammonium salt

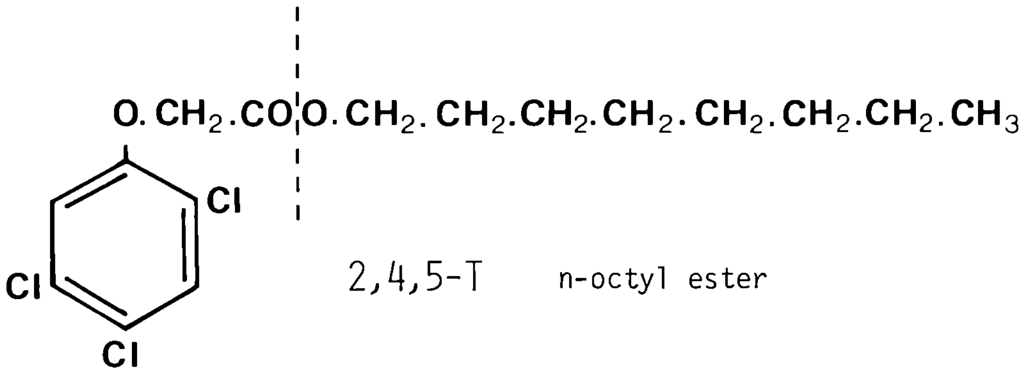


triethanolamine salt

Figure 2 Salts of 2,4-D and 2,4,5-T.



2,4-D n-butyl ester

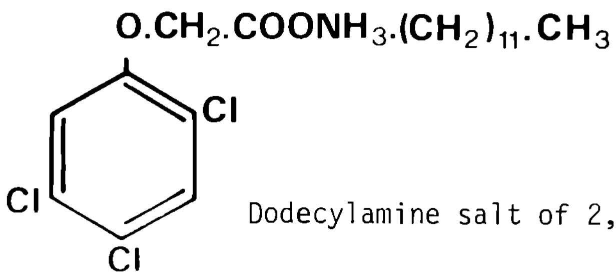


2,4,5-T n-octyl ester

Figure 3 — Esters of 2,4-D and 2,4,5-T.

$NH_2.(CH_2)_{11}.CH_3$

Dodecylamine



Dodecylamine salt of 2,4,5-T

Figure 4 — Dodecylamine and the salt it forms with 2,4,5-T acid.

2. The manufacture of 2,4-D and 2,4,5-T

Figure 5 shows the more important steps in the usual route of manufacture.[34] 2,4-D is made by reacting 2,4-dichlorophenol with sodium monochloracetate, while 2,4,5-T is usually derived from tetrachlorobenzene, via 2,4,5-trichlorophenol. Technical grades of 2,4-D and 2,4,5-T are normally about 98% and 90% pure, respectively.[34] The commonest impurities are chlorinated phenols and other chlorinated phenoxyacetic acids. In the past, particularly when American production was greatly increased during the Vietnam war, some batches of 2,4,5-T contained small amounts (up to 40 ppm) of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). This substance, whose structure is shown in Figure 6, is a member of a large group of compounds consisting of two benzene rings linked by adjacent oxygen bridges. Dioxins other than TCDD can occur in samples of 2,4,5-T but TCDD is the commonest. Under certain conditions, it is formed during the hydrolysis of tetrachlorobenzene to 2,4,5-trichlorophenol (Figure 5.) The conditions which favour dioxin production are alkalinity, high temperature and high pressure.[34] If reaction conditions are properly regulated dioxins are not formed.

3. Formulation ingredients

Apart from the herbicides themselves, commercial preparations of 2,4-D or 2,4,5-T often contain materials such as paraffinic oils, other organic solvents and surfactants or detergents. These are added to improve stability during storage, to assist dilution, or to improve herbicidal activity.[59, 60]

4. The mode of action of the herbicides

2,4-D and 2,4,5-T are most active when applied to the aerial parts of plants or introduced directly into vascular systems. The most usual route of entry is through foliage, but uptake through branches and twigs can also occur, particularly when oil-based spray solutions are used. This type of spray is sometimes used for treating the bark of woody plants during winter. Even when foliage is present, bark uptake may also be important, for example when leaves are shed shortly after spraying, as occurs with *Acacias* and some other leguminous tropical species. Significant amounts of herbicide probably enter through bark when overall sprays are applied to species with multi-branched twigs, such as birch, poplar, and willow. 2,4-D and 2,4,5-T can be assimilated through the roots of plants but with woody species, soil action is only of minor importance.

Although many workers have studied the mode of action of the herbicides, their precise effects on plant cells are not yet fully understood. Like many other herbicides, the phenoxyacetic acids can produce a range of responses which vary with the species, the size and condition of the test plants, and the dose and formulation of the herbicide. The most important effect is on cell co-ordination: 2,4-D and 2,4,5-T act upon some nuclear function which alters the balance of enzyme systems and has widespread effects on cell division and growth.[5] One obvious and immediate effect is to cause unequal cell elongation on the opposite sides of stems or leaf petioles. This causes epinasty, a characteristic twisting of young growth. An example is shown in Plate 2, page 17 . At low doses the herbicides sometimes stimulate growth, perhaps by increasing production of ribonucleic acid (RNA). However large doses cause reduced and abnormal development, probably by causing imbalance of RNA and other growth regulating substances. Treated plants often develop grotesquely malformed leaves and stems, as in Plate 3 p. 17 . Adventitious roots are often formed. In lima bean, sprays containing from 1.5 ppm to 3 ppm of 2,4,5-T cause flower shedding and an initial inhibition of growth.[62] Later, when presumably the concentration of herbicide in the plants has declined, lateral shoot growth is stimulated and 2,4,5-T treated plants may weigh more than untreated material. Both 2,4-D and 2,4,5-T can upset normal cell division in *Allium cepa* by delaying spindle formation and by increasing the number of chromatid breaks.[63] In this species, the effects and the herbicides resemble those of X-ray radiation.[63] 2,4,5-T treatments have recently been shown to cause the formation of *Tradescantia* cells with more than one nucleus.

The application of strong solutions of phenoxyacetic acid herbicides to leaves often leads to desiccation and abscission. Effects of this kind occur with many types of herbicide. However,

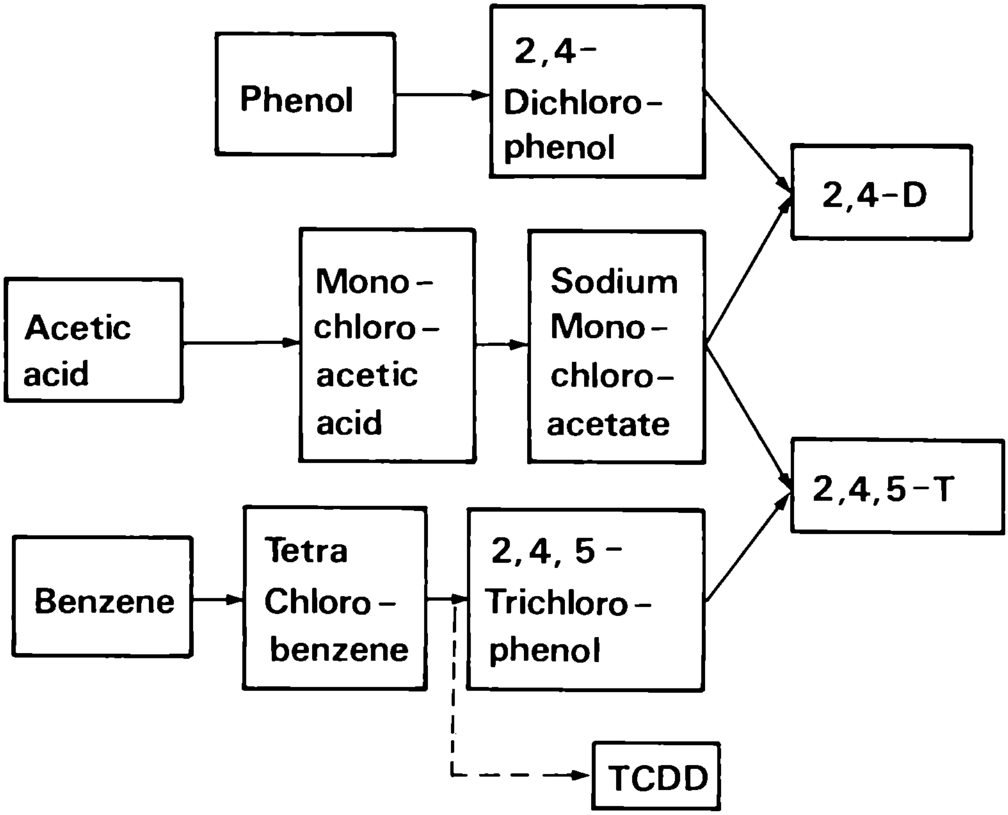


Figure 5 The routes of manufacture.

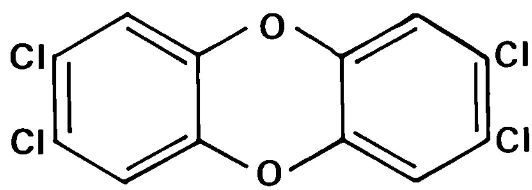


Figure 6 The chemical structure of TCDD.

phenoxyacetic acids have a more specific defoliant effect on many woody species. The rates which are needed to bring about defoliation are normally greater than those currently used for controlling woody weeds. However, relatively low doses can bring about leaf shedding if they are applied late in the season, when natural leaf fall is imminent [29] Osborne[65] has examined the defoliant effects of the herbicides in detail. Localized applications of 2,4-D and 2,4,5-T esters retard ageing at the point of treatment but accelerate this process on adjacent parts of the leaf. This response is thought to result from an imbalance between auxins and the gas ethylene. Normally a high concentration of auxins in leaves prevents ageing and leaf abscission. Ethylene has an opposite effect; exposure to the gas or treatment with a substance which releases ethylene causes leaves to become yellow and fall. Osborne[65] suggests that 2,4-D or 2,4,5-T applied to leaves increase ethylene production but, as the herbicides are themselves powerful auxins, the net effect at the point of treatment is to retard ageing. However, the rest of the leaf is exposed to ethylene diffusing outward from the treated areas. Here the effects of the gas are counteracted only by the small amounts of natural auxin which are present. Most of the leaf, that is the part which is not directly in contact with the herbicide solution, becomes yellow and before long, leaf-fall occurs.

Other effects of the herbicides include disturbance of the balance between synthesis and use of nutrient materials.[51] 2,4-D and 2,4,5-T treatments often cause a rapid decline in the carbohydrate reserves of roots, rhizomes and other storage organs. Plants which are slowly killed by moderate doses of the herbicides often have almost no food reserve at the time of death.[51] Many workers have shown that the herbicides are most effective when applied under conditions of active photosynthesis, when translocation from leaves to storage organs is at a maximum.[73 74 75]

Attention has been drawn to the close chemical relationship between 2,4-D and 2,4,5-T. Many experiments comparing the uptake, translocation, degradation and toxic effects of the herbicides have been undertaken.[66 67 68 69 70] While there is much variation, it appears that 2,4-D is often absorbed and translocated more readily, but may break down more rapidly, in plant tissues. Thus, in oaks, 2,4-D is more readily degraded than 2,4,5-T.[69] However studies of this kind have been undertaken with only a limited number of species; further work may alter existing views about the factors which govern 2,4-D and 2,4,5-T activity.

5. The choice of 2,4-D and 2,4,5-T formulations

The chemical and physical properties of the commoner derivatives, particularly salts and esters, have been discussed. There are often large differences in the herbicidal activity of these derivatives, which are sometimes greater than differences between the effects of the parent herbicides. Although some formulations do not fit well into simple grouping, one way of classifying them is by their water or oil solubility. Oil-soluble formulations include the esters, oil soluble amine salts and "acid in oil" preparations.[50] Only the salt formulations such as the triethylamine salt shown in Figure 2 have high water solubility. In woody plant control, oil based herbicide solutions often have an important advantage because of their ability to pass through bark or the cuticle of thick fleshy leaves. Where barriers of this kind must be passed, the use of an oil solution of the herbicide is almost essential. This might be a straight oil solution or an oil solution which is emulsified with water. "Invert" or "mayonnaise" water-in-oil emulsions, as well as the more usual oil-in-water type, can be used.[57] Oil solutions are usually prepared from esters. However although esters in oil can enter plants readily, they are then immobile.[53] Before any systemic effects can occur the esters must hydrolyse, that is, break down, to release the parent acid, which is slightly water soluble. This process takes place rapidly in actively growing plants but may be slower in dormant tissues. Sometimes an ester may break down so slowly that lethal concentrations of free herbicides do not occur. Where slow hydrolysis is likely, oil soluble amine or acid-in-oil formulations may have advantages.

When herbicide solutions do not have to pass through bark or thick cuticle, a water-soluble type of formulation is most effective.[36] This type of formulation is appropriate where the herbicides are introduced directly into vascular tissues, as in the "cut-bark" treatments described in the following section. Water-soluble amine salts are

Plate 2 Epinastic effects due to 2,4,5-T.

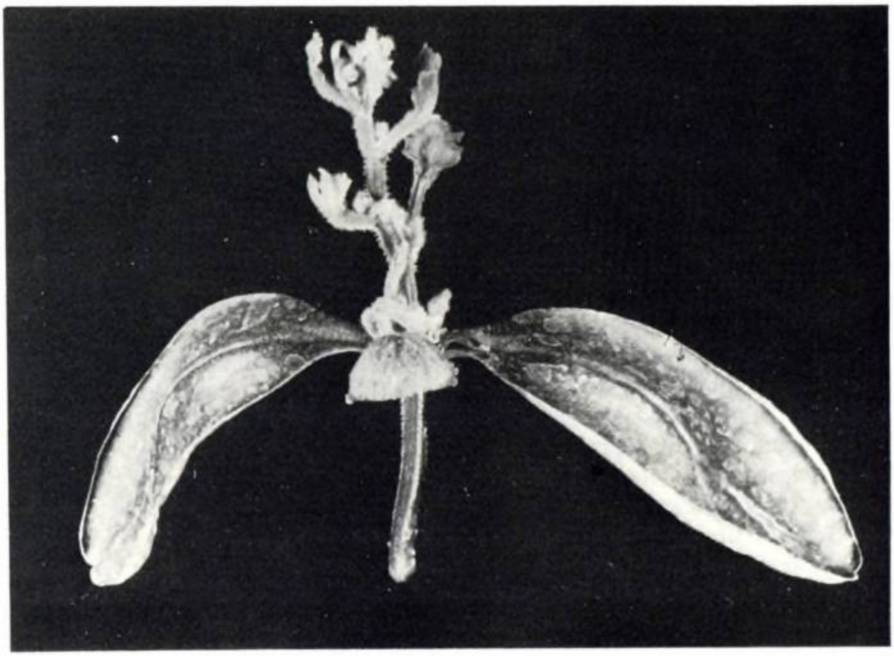


Plate 3 Abnormal growth following 2,4-D treatment.

also sometimes more effective than oil solutions for foliage spraying.[66, 79] Although salt formulations of 2,4,5-T are often used in North America, they are not as yet generally available in Britain.

6. The practical use of 2,4-D and 2,4,5-T for woody plant control

Current recommendations for forestry uses of 2,4-D and 2,4,5-T in Britain are given in Volume II of the British Crop Protection Council's Weed Control Handbook[35], and in Forestry Commission publications[78]. While 2,4-D has many agricultural uses, for practical purposes 2,4,5-T is now employed only against woody weeds. Woody weeds may of course occur in a variety of situations, not only in forests.

In forestry, the herbicides may be used either in the presence or absence of a crop. This may be either a conifer or, less commonly, a broadleaved tree. In either situation, a variety of application methods may be used, some of which have no parallel in herbaceous weed control. Woody weeds are unique in that they are often so large as to make treatment of individual plants a practical possibility. They are often so tall as to make spraying from the ground difficult, and they frequently occur in places which cannot easily be reached with large equipment.

While it is not intended to give a detailed account of the practical uses of 2,4-D and 2,4,5-T, the principal methods used for treating woody plants with these herbicides are discussed briefly. At least 5 methods can be employed:-

6.1. Overall foliage sprays.

Widely used in Britain, these are best applied when leaf growth has reached its maximum but when the leaves are still healthy and fully functional[73, 74, 75]. Ground equipment can be used for spraying regrowth or low-growing species such as heather and brambles, but aerial spraying is often the only practical method of applying overall foliage sprays to tall trees or thickets. Volume rates of from 20 litres/hectare (2 gallons per acre) to 1100 litres/hectare (100 gallons per acre) have been used. Low volume sprays are only just beginning to be used in Britain but will probably become important in future[214]. Aerial spraying usually involves applying from 30 - 60 litres/hectare of solution. However, 2,4,5-T has seldom been applied from the air in Britain since 1969. While low volume applications do not usually give quite as good results as the more conventional medium or high volume treatments they are adequate for most purposes[36]. The herbicide doses which are employed vary widely, according to the weed species present. From 1 - 5 kg/ha is usual but higher rates are necessary for some resistant species[78]. Directed sprays, applied with a hand-held lance, can be safely used among planted conifers at any time except during very hot weather or periods of very active growth[78]. Undirected overall sprays are only safe if the conifers are dormant, that is, shoot elongation has ceased and buds have formed. In Britain this is usually from mid August onwards. However, even in autumn, overall sprays may cause slight damage to susceptible species such as Lodgepole pine, Western hemlock and larches[78].

Oil-soluble formulations, usually esters, are most frequently used but salt formulations also sometimes give good results particularly on species which do not have thick fleshy leaves. The addition of emulsified oil to aqueous formulations sometimes increases herbicide activity[36].

6.2. Dormant shoot sprays.

This method of treatment, used only occasionally in Britain[35] but more commonly in America, involves applying oil solution of the herbicides in winter to the bare twigs and branches of dormant deciduous species. The most suitable time for treatment is when the buds have started to swell but have not yet opened[36]. In Britain it is recommended that 2,4,5-T esters be applied at concentrations up to 22.4 kg (20 lb) acid equivalent, in 1125 litres (100 gallons) of paraffin, to wet all branches to the point of runoff[35]. Such mixtures are normally applied at 536 - 530 l/ha (30 - 50 gpa). In North America both 2,4-D and 2,4,5-T are used and much lower volumes of oil solution are applied, either by helicopter or with ground operated mist blowers.[36]

6.3. Basal bark sprays.

With this type of treatment, oil solutions are sprayed or painted onto the lower part of stems. Water solutions are almost inactive when applied to bark, while oil-in-water emulsions are only moderately effective. Basal bark treatments may be applied at any season but are most effective in Britain between January and March[78]. In North America they are applied from March to October[36]. Normally only the lower 20 - 30 cm of each stem is treated with a concentrated solution in oil containing from 1½ to 2½ per cent w/v of 2,4-D or 2,4,5-T. This is sprayed to the point of run-off[35 36]. The requirement for herbicide and oil naturally varies with the size and number of the stems to be treated but 25 - 30 ml of solution is usually needed for each 25 cm of stem diameter[35 78]. Moss covered stems soak up much larger volumes of solution. Sometimes the effects of basal bark treatments do not become apparent for a considerable time after spraying; treated trees may produce apparently healthy leaves which subsequently die back during one or two seasons after treatment. In America, spring treatments provide the most rapid kill of aerial growth but summer or autumn basal bark sprays have a greater effect on regeneration from below ground level[36]. Provided that herbicide formulations of low volatility are used and reasonable care is taken during spraying there is little risk to crop species. In Britain basal bark sprays are mostly applied to small or multistemmed growth but in America these treatments are successfully used on fully grown trees[35 36].

A modified form of treatment is sometimes employed in North America which involves spraying both the trunk and as much of the foliage and branches as can be reached from ground level[36]. This method of treatment is very effective but naturally requires more herbicide and oil than conventional basal bark spraying.

6.4. Cut-bark and cut-stump treatments.

These application methods entail introducing herbicide solutions directly into the vascular tissues of woody growth. Variations of "cut-bark" treatments include "frilling", "notching", and tree injection[35 36 78]. Stump treatments involve applying solutions to freshly cut surfaces with the object of preventing regrowth. Details of methods used in Britain are given in Volume I I

Table 4.

The use of phenoxyacetic acid herbicide formulations for woody plant control

Type of treatment	*Formulation and solvent*				
	Acid in oil	*Low volatile ester in oil*	*Low volatile ester emulsified in water*	*Amine salt in water*	*Oil soluble amine in oil*
Conventional medium or high volume foliage spray	g	X	G	g	g
Very low volume foliage spray	g	G	X	X	g
Dormant shoot spray	g	G	X	X	g
Basal bark treatment	g	G	X	X	g
Cut-bark and cut-stump	g	G*	X	g*	g

G commonly used g used only occasionally or not at all
X unsuitable * see text

of the Weed Control Handbook[35] and in Forestry Commission publications[78 242]. When herbicide solutions are introduced directly into vascular tissues, as in all these treatments, water-soluble formulations are more effective than oil based solutions[36]. Oil soluble formulations are currently recommended in Britain[35 78 80 81] but this probably originates from the practice of using frill girdling and basal bark spraying as complementary techniques, the former for the larger trees and the latter for smaller multistemmed growth. The two types of treatment are generally applied as a combined operation by one person, using the same ester-in-oil solution. When herbicides are applied by sprayer, large areas of bark are wetted as well as the frill girdles or cut surfaces so that ester-in-oil solutions are often reasonably effective. However where only cuts or the surfaces of stumps are treated, water soluble formulations are likely to give superior results[36]. In the United States, the recommended method of treating stumps is to apply concentrated 2,4,5-T amine salt solution to the area of active vascular tissue just beneath the bark. With this type of application a single teaspoonful of concentrate is sufficient for even a large stump[36]. In Britain it is recommended that cut-stump treatments should not be carried out when the stumps are wet[35 78]. However there is also evidence that herbicidal effects may be reduced if the treated stumps are shielded from rain or dew[81].

Table 4 shows the formulations of 2,4-D and 2,4,5-T which are recommended for these five methods of application.

CHAPTER 3

The effects of 2,4-D and 2,4,5-T on domestic livestock and on man

From the time of their discovery it was realised that 2,4-D and 2,4,5-T might affect organisms other than plants. The effects of the compounds on farm animals and humans are obviously of special importance. While the introduction of any new chemical compound into general use can involve hazards, there are particular dangers with agricultural chemicals, which are handled by relatively unskilled workers and applied to thousands of acres of farmland and forest, under conditions where some contamination of food crops or drinking water is almost inevitable. The special hazards associated with agricultural chemicals have long been recognised. Legislation regulating their use in Britain exists under the Agriculture (Poisonous Substances) Act, 1932. The Pesticides Safety Precautions Scheme administered by the Ministry of Agriculture, Fisheries and Food, keeps risks associated with pesticides under constant review and issues recommendations for their safe use.

In practice, pesticides present two main kinds of hazards to humans and animals. Firstly there are risks associated with compounds of high acute toxicity. This type of poisoning usually follows the assimilation of a single relatively large dose. Secondly, there may be more insidious risks due to chronic toxicity, that is the effects of repeated or prolonged exposure to the material. While symptoms of acute poisoning usually develop quite quickly, those of chronic or sub-acute toxicity may not appear until many small doses have been assimilated, for example from contaminated food or water or from inhalation of spray droplets. Materials with properties of chronic toxicity are responsible for many industrial diseases. Chronic toxicity is usually most serious when a substance is not broken down or excreted by the body. Sometimes repeated exposure to sublethal doses can induce cancers or nervous disorders. Teratogenic effects, the production of developmental defects in unborn foetuses, are yet another possibility.

When a pesticide is being tested for toxicity towards animals, commercial or formulated products should be evaluated as well as the pure material. Derivatives such as esters and salts often differ in toxicity from their parent compounds. Formulation ingredients such as surfactants and oils may themselves be poisonous or in one way or another modify the toxicity of other compounds. For example, the presence of oils can enhance the rate at which a chemical passes through skin. Commercial products sometimes contain impurities which may be more harmful than pure materials. Thus, in the past, some batches of 2,4,5-T have contained up to about 30 ppm of the very poisonous compound 2,3,7,8-tetrachlorodibenzo-p-dioxin[34]. This contaminant is discussed in a later section (see p 24).

1. **Acute effects of 2,4-D and 2,4,5-T on animals.**

When a new chemical compound is being examined, it is standard practice to carry out toxicity studies with laboratory animals. During the early stages of evaluation these are usually rats or mice but later other species including monkeys may be used. In the case of agricultural chemicals, experimental programmes often include farm livestock. The results of tests for acute toxicity are usually expressed as the average dose, in milligrams per kilogramme of body weight, which kills 50 per cent of the experimental animals. This is usually abbreviated to "LD_{50}". When the compound is administered by mouth the value is termed the "acute oral LD_{50}". The test species should always be specified because there are often large differences in the susceptibility of different animals. Compounds with high acute oral toxicity to rats or mice tend to have the same kind of effect on other animals or humans but there is often little correlation between the responses of different species. In any case, oral effects represent only one aspect of acute toxicity. Some chemicals are inactive when taken orally but very poisonous when inhaled or introduced into the bloodstream. Also, it must be emphasised that acute oral LD_{50}'s give no guide at all to chronic or sub-acute effects.

Bearing in mind these limitations of acute toxicity data, all the evidence which is available suggests that when pure or formulated 2,4-D or 2,4,5-T are taken by mouth they are only moderately poisonous, having about the same toxicity as aspirin. Many earlier studies with the herbicides have been summarised by Rowe and Hymas [83]: some representative acute oral LD_{50} values from their paper are shown in Table 5. This Table also shows the range of doses within which mortality occurred. The data were obtained from tests with mixed butyl esters. In general, other derivatives, including amine salts, had similar toxicities. Formulated products usually had higher LD_{50}'s, suggesting that the adjuvants present did not, in this instance, enhance toxicity. To provide a standard for comparison, aspirin has an acute oral LD_{50} for rats of about 1200 mg/kg [5].

Other studies have generally confirmed Rowe and Hymas' figures. However, esters with a large esterifying group such as propylene-glycol-butyl-ether appear to be more toxic than those with a smaller group [34].

A summary of toxicological data for 2,4-D published up to 1967 has been prepared by House *et al*[32]. The quoted acute oral LD_{50} values, mostly between 350 mg/kg and 2000 mg/kg, are similar to those given by Rowe and Hymas[83]. However dogs have been found to be more susceptible than most other species, with an acute oral LD_{50} of about 100 mg/kg[32].

Less attention has been given to 2,4,5-T. The accepted oral toxicity values for this herbicide are still those published 20 years ago by Rowe and Hymas[83]. Such other data as is available agrees reasonably well with Rowe and Hymas' figures[84 85 88].

Table 5.

Acute oral LD_{50} values (mg/kg) for 2,4-D and 2,4,5-T as mixed butyl esters.

Test animal	*2,4-D*	*2,4,5-T*
Rat	620 (320- 954)	481 (313- 739)
Guinea Pig	848 (604-1190)	750 (500-1000)
Rabbit	424 (252- 712)	712 (500-1000)
Mouse	713 (500-1000)	940 (674-1312)
Chick	2000 (1350-2760)	not available

* From Rowe and Hymas [83]

2. Chronic (Sub-acute) effects of 2,4-D and 2,4,5-T on animals.

Many long term feeding studies have been undertaken with domestic and farm livestock. Several animal species have been found to be unaffected by repeated small or moderate doses of 2,4-D and 2,4,5-T formulations. For example Palmer fed 50 mg/kg per day of 2,4-D (as an alkanolamine salt) to steers for 112 days, without ill effects[86]. The treatment involved feeding more than an ounce of herbicide daily to each animal. Higher daily doses of 100 mg/kg or 250 mg/kg produced toxic symptoms. From consideration of these results, Palmer suggested that 2,4-D was not a cumulative poison. Palmer and Radeleff[87] found that sheep could tolerate up to 481 daily doses of 100 mg/kg 2,4-D, as the alkanolamine salt or the propylene-glycol-butyl-ether (PGBE) ester. Cattle were slightly more susceptible. Larger doses of 2,4-D, equivalent to 250 mg or 500 mg per kilogramme of body weight, were usually lethal, causing liver and kidney degeneration and heart haemorrhages in both species. Drill and Hiratzka[88] found that dogs survived daily doses of 10 mg/kg 2,4,5-T for 90 days but died when given twice this quantity of herbicide. Grigsby and Farwell[89] did not observe any toxic effects when cattle, sheep, horses, pigs and chickens were allowed to graze pasture sprayed with 3.7 or 4.5 kg/ha of 2,4-D or 2,4,5-T, as salt or acid formulations. The unpleasant taste of 2,4-D reduced the amount of forage which was taken. Sheep have been fed 481 daily doses of 100 mg/kg of 2,4,5-T as the triethylamine salt, without ill effect[87]. However, as with 2,4-D, repeated dosing with larger quantities of 2,4,5-T causes liver and kidney degeneration which can lead to death[87]. Erne and Bjorklund[90] found that the addition of 1000 ppm of either 2,4-D or 2,4,5-T to drinking water, from the 5th day after hatching until chickens were 7 months old, caused kidney enlargement and a high proportion of deaths. Other toxicological studies with repeated sub-acute doses of the herbicides are reviewed by Way[92] and Warren[93].

Although there is now much evidence to show that 2,4-D and 2,4,5-T are only mildly toxic, the use of these and other herbicides is often followed by reports of the death or illness of animals. Fertig[94] carefully investigated many suspected causes of herbicide poisoning in farm livestock, over a 2-year period. None of the reported deaths were found to be actually due to herbicides. Causes of death included old age, disease, drowning, agricultural chemicals other than herbicides, and even gunshot wounds.

One particular type of sub-acute effect, teratogenicity, is discussed in a separate section (see p 26)

3. The effects of 2,4-D and 2,4,5-T on humans

Only a few well documented reports of the effects of 2,4-D on humans have been published and there is almost no information about illness or death caused by 2,4,5-T. In view of the very widespread use of these herbicides for more than 25 years, this absence of reports of poisoning is reassuring. 2,4-D occasionally causes an illness termed peripheral neuropathy, but this reaction occurs in only a small proportion of individuals who are predisposed to the condition[91]. House *et al* discuss reports of the effects of large doses of 2,4-D[32]. These are few but include the case of a man who ate 500 mg of pure 2,4-D per day for 21 days without ill effect and a death by suicide following the consumption of about 6.5 g of the herbicide. From the slender evidence now available House suggests that between 50 and 100 mg/kg of 2,4-D might be a lethal dose for a human. This makes 2,4-D about as poisonous to humans as aspirin or DDT[34].

The Academy of Sciences Report[34] discusses several health studies carried out on groups of persons who have been exposed to the herbicides for long periods. Some workers in factories which make 2,4,5-T have suffered from TCDD poisoning. This contaminant is discussed separately (p.26). Excluding TCDD poisoning, however, there is little evidence of adverse effects resulting from normal occupational contact with the herbicides. Exposure for long periods to high concentrations of 2,4-D or 2,4,5-T, for example in a factory where the atmospheric concentration of 2,4-D dust reached 35 mg per cubic metre, sometimes causes fatigue, headache, loss of appetite and other symptoms of illness. However these symptoms are usually transitory, disappearing within a day or so. The results of these health studies agree well with those of experiments involving the repeated administration of sub-lethal doses to animals.

4. 2,4-D and 2,4,5-T in animal products

It has been suggested that 2,4-D and 2,4,5-T may be accumulated from forage by domestic animals and that this may lead to unacceptably high levels of the herbicides in meat or milk. Much of the work discussed in previous sections provides evidence against accumulation[86 87 88 89] Erne[96] has studied in detail the distribution and elimination of the herbicides, in rats, pigs, calves, and chickens. In all species, salt formulations were more rapidly assimilated and distributed than esters. When esters were included in diets, only small amounts of the herbicides could be detected in body tissues, mostly in the liver and plasma.

With salt formulations more herbicide was assimilated, the highest concentrations occurring in the liver, kidneys, lungs, and spleen. The concentrations in these organs sometimes slightly exceeded the levels in the blood plasma. Only small amounts of the herbicides were present in blood cells, usually about 10–20 per cent of the level in the plasma. 2,4-D penetrated very slowly into fat tissue of the cells of the central nervous system. In pigs, some transfer of this herbicide through the placenta took place. No retention or accumulation occurred in any species. Usually, 50 per cent. of a moderate dose of 2,4-D and 2,4,5-T was excreted through the kidneys and urine within 5 - 30 hours of ingestion. In the hen, small amounts of 2,4-D were excreted in the eggs. St. John *et al* [96] showed that when cows were fed a diet containing 5 ppm of 2,4,5-T or fenoprop, the herbicides did not appear in the milk but were eliminated in the urine. Klingman[97] found that the milk of cows grazed on pasture sprayed with 2,4-D contained only minute amounts of the herbicide, detectible for only 2 days. When cows were given food containing 1000 ppm of 2,4-D, 2,4,5-T, fenoprop or MCPA, concentration of the herbicides in the milk did not reach 1 ppm[98] . After the herbicides are withdrawn from the diet, residues in the milk disappear very quickly[98] .

Clark *et al*[99] assessed residues of 2,4-D, 2,4,5-T and fenoprop in the meat of sheep and cattle which had been fed 2000 ppm of these herbicides. At 4 weeks when the cattle were slaughtered, up to 0.1 ppm of 2,4-D, 2.7 ppm of 2,4,5-T and 2 ppm of fenoprop were detected. In experiments with a 2,4,5-T ester, Clark and Palmer[100] observed that much of the ester was excreted in the urine of sheep and cattle within 24 hours mostly unchanged.

After feeding sheep a single dose of 25 mg per kg or daily doses of 0.15 - 0.75 mg/kg of 2,4,5-T, levels in the flesh were below 0.1 ppm. However when animals were acutely poisoned with repeated massive doses of the herbicide, up to 368 ppm occurred in some tissues, presumably as a result of the breakdown of the normal processes of excretion through kidneys and liver.

Zielinski and Fishbein[101] compared the rates of disappearance of various 2,4-D and 2,4,5-T derivatives from the bodies of mice. 2,4-D butyl ester was eliminated most rapidly, followed in order of rates of disappearance by 2,4-D octyl ester, 2,4-D acid and 2,4,5-T acid. Pretreatment of the mice by feeding repeated small doses of 2,4-D butyl ester enhanced the rate at which subsequent larger doses disappeared. A similar effect has been reported in pigs and chickens[94] .

To sum up, it appears that appreciable amounts of 2,4-D or 2,4,5-T are most unlikely to occur in meat or milk. Accumulation does not take place, small doses such as might be ingested from treated pasture or crops being rapidly eliminated. Concentrations of more than 1 or 2 ppm could only be present in animal tissues following the consumption of a very large and almost certainly lethal dose. As the herbicides have a very unpleasant taste, animals are likely to reject badly contaminated food.

5. 2,4-D and 2,4,5-T residues in crops and fruit.

In general, phenoxyacetic herbicides are unlikely to occur in vegetables or fruit which are consumed by humans. In Britain and in most other countries, there are regulations which stipulate a time interval - usually at least 4 weeks - between the application of herbicides to food crops and their harvest and sale[35] . However wild fruit, particularly blackberries and raspberries, are common in forests and waste places and could obviously be eaten very shortly after spraying. In Britain, Brown and Mackenzie[105] . sprayed fruiting blackberry bushes with 3 kg/ha of 2,4,5-T. Following this treatment, immature fruit failed to ripen. Ripe fruit remained in good condition for about 3 days but thereafter moulded rapidly. Unwashed fruit collected within 3 days of spraying contained up to 100 ppm of 2,4,5-T. Even this high level would probably not be dangerous in most circumstances. However as a precaution Brown and Mackenzie suggest that bushes bearing ripe fruit which are accessible to the public should not be sprayed. Similar studies in Germany with wild raspberries have been carried out by Olberg[106]. After application of 6 kg/ha of 2,4,5-T, residues of from 0.05 ppm to 3 ppm were present in the fruit. However, as Olberg points out, even these comparatively low levels of herbicide are well above those allowed in foodstuffs in most countries.

6. Indirect effects of 2,4-D and 2,4,5-T on farm livestock

2,4-D and 2,4,5-T can have many kinds of indirect effects on farm animals. These are often beneficial, for example the use of the herbicides can markedly improve the botanical composition and quality of pastures. There are however at least two less obvious ways in which the compounds could affect livestock.

Firstly, herbicide applications can sometimes modify the palatability or toxicity of poisonous plants. Studies in this field, reviewed by Way[92] and Warren[93] show that the palatability of toxic but normally unpalatable species can often increase as plants die and dry out. This may, for example, result from changes in the sugar content of foliage. Occasionally toxicity may be induced in plants which are normally harmless, as for example where herbicide treatments increase the content of nitrates. Sometimes 2,4-D or 2,4,5-T can change alkaloid contents making poisonous plants either more or less dangerous. Where toxic plants are present or nitrate poisoning occurs, the use of phenoxy-acetic acid herbicides may slightly increase the danger of poisoning. In most circumstances however the extra risks which are involved are probably slight.

Secondly, the herbicides could conceivably affect nutrition through effects on the micro-organisms of the rumen. This possibility was examined by Kutches *et al*[104], who found that most species which colonise the rumen can tolerate at least 500 ppm of 2,4-D or 2,4,5-T. Very high levels of 2,4,5-T may reduce the rate of dry matter breakdown but it is unlikely that digestion is ever measurably affected by herbicides ingested with sprayed herbage.

7. 2,3,7,8 - tetrachlorodibenzo-p-dioxin (TCDD)

As early as 1899 an industrial disease known as "chloracne" was recorded among workers in factories producing chlorinated phenols. During the 1950's and 1960's several outbreaks of this serious condition occurred in chemical plants producing 2,4,5-T. The illness was found to be associated with side reactions which occasionally took place during the preparation of trichlorophenol[34] (Figure 5.) When reaction conditions were not properly controlled, poisonous contaminants were produced. One such impurity, 2,3,7,8 - tetrachlorodibenzo-p-dioxin (TCDD), was shown to be the causal agent of chloracne[115]. In experiments with laboratory animals TCDD caused liver damage and other pathological changes as well as skin disorders. Unpublished data supplied by the Dow Chemical Co. and quoted by Sparschu *et al*[116] place the acute oral LD_{50}'s for male and female rats at 0.023 mg/kg and 0.045 mg/kg respectively. These values are very low indeed, TCDD being among the most toxic substances ever investigated. The LD_{50}'s for 2,4-D and 2,4,5-T, 620 mg/kg and 481 mg/kg, (Table 5) are more than 10,000 times greater. Extrapolation from the TCDD acute toxicity data places the order of dose which is likely to be lethal to humans at from 1.5 to 3 milligrammes, about the weight of 5 grains of fine table salt.

The chemical structure of TCDD has been discussed in Chapter 2. TCDD is the dioxin which most often occurs in trichlorophenol but others have also been detected. Some of these, such as hexachlorodibenzo-p-dioxin, are almost as poisonous as TCDD[34]. From about 1955 onwards, the very poisonous properties of TCDD and certain of its chemical relatives were well known to chemical manufacturers. Most firms took active steps to prevent the impurities being formed. It was found that careful control of reaction conditions, particularly avoidance of high temperatures and pressures, reduced the amount of dioxin in trichlorophenol and 2,4,5-T to negligible levels. In a survey of 2,4,5-T samples

Table 6.

LD_{50} values for single doses of TCDD

Species	*Application*	*LD_{50} mg/kg*	*Authority*
Rat	Oral	0.022 - 0.045	Schwetz *et al*[120]
	Oral intubation	0.100	Harris *et al*[121]
Mouse	Oral intubation	0.100 - 0.200	Harris *et al*[121]
Guinea Pig	Oral	0.0006 - 0.0021	Schwetz *et al*[120]
	Oral intubation	0.001 - 0.003	Harris *et al*[121]
Rabbit	Oral intubation	0.115	Schwetz *et al*[120]
	Skin	0.275	Schwetz *et al*[120]
	Intraperitoneal	0.063 - 0.500	Schwetz *et al*[120]
Dog	Oral intubation	0.100 - 3.00	Schwetz *et al*[120]

manufactured in the United States between 1966 and 1970 Woolson *et al*[118] found that 22 out of 42 batches had less than 0.5 ppm of TCDD. However, some material contained 20 - 30 ppm of the dioxin. It is known that on certain occasions when reactive processes became uncontrollable relatively large amounts of TCDD were formed. Appreciable amounts were, for example, present in debris after an explosion at a British chemical plant in 1968[119].

Existing information about the acute toxicity of TCDD is summarised in Table 6, taken from the US National Academy of Sciences Report.

It will be seen that acute toxicity varies considerably between species. Guinea pigs are extremely susceptible while rabbits and dogs are relatively resistant. The figure for rats, established by Schwetz *et al*[120], agrees well with the earlier data given by Sparschu[116].

In longer term feeding studies with TCDD, Harris *et al*[121] found that 30 daily doses of 0.001 mg/kg or six weekly doses of 0.005 mg/kg reduced the growth rate of rats. Many similar studies have been undertaken[34]. As with acute effects, guinea pigs are particularly sensitive, while mice, rabbits, and dogs can tolerate higher levels in diet.

Earlier it was suggested that a single dose of from 1.5 to 3.0 mg of TCDD might be fatal to an adult human. It is instructive to calculate the amount of impure 2,4,5-T, assumed to contain 30 ppm of TCDD, which provides this dose of the dioxin. The quantity is between 50 and 100 grammes acid equivalent or perhaps 100 - 200 ml of a typical formulated product. While there is no question about the very dangerous properties of TCDD it is worth noting that consumption of this large amount of 2,4,5-T would in any case probably cause death. Extrapolation from the values in Table 5 suggests that a lethal dose of 2,4,5-T for a human may be about 5 - 10 grammes. Insofar as acute effects towards humans are concerned, therefore, the presence of up to 30 ppm or so of TCDD does not appreciably increase the risks associated with 2,4,5-T. However TCDD has other dangerous sub-acute effects and is an extremely stable persistent compound. Its presence in 2,4,5-T should always be a matter of grave concern.

In most experiments with animals, the toxic effects of TCDD have developed slowly. At dose levels near the LD_{50} the average time to death in rats, guinea pigs and mice is around 20 days[120]. The clinical effects of TCDD are diverse and include necrotic changes in the liver, kidneys, thymus and other lymphoid tissues, together with intestinal haemorrhages[34]. Elimination of the compound from the body is slow. In studies with C^{14} - labelled TCDD, only half of a moderate dose disappeared from the bodies of rats within 17 days[129]. Elimination was mostly in the faeces and urine but a little radioactivity was exhaled as carbon dioxide through the lungs. Most of the radioactivity retained by the rats at the time of death was in their liver and fat.

While the high acute toxicity of TCDD was well established by the early 1950's, teratogenic activity was not suspected until much later. The sequence of events which led to the banning of most uses of 2,4,5-T in the United States has been related in Chapter 1, In brief, it was reported that large daily doses of impure 2,4,5-T significantly increased the proportion of foetus abnormalities in rats[46]. When the report was first prepared, the authors were apparently unaware that they had used impure 2,4,5-T. However, a footnote to the paper stated that the sample of herbicide had subsequently been found to contain 30 ppm of TCDD. Normally, this study would probably have attracted little notice. However, public disquiet about military uses of defoliants gave the report tremendous news value. The US Government had no alternative but to suspend the use of 2,4,5-T in Vietnam and at home[34]. The announcement of teratogenic effects stimulated much new work with both 2,4,5-T and TCDD. It now seems that the reported teratogenic effects were largely or entirely due to the impurity rather than 2,4,5-T. Studies with 2,4,5-T are discussed separately. However, teratogenic effects of TCDD in rats and mice have now been established beyond any reasonable doubt. In studies with mice, single doses of .005 mg/kg or repeated daily doses of 0.001 or 0.002 mg/kg significantly increased the proportion of abnormal foetuses[34, 123]. These effects were reproducible and highly specific as almost all the defects were cleft palates or kidney anomalies. Limb malformations are not increased by TCDD even when the compound is given at a stage of pregnancy where limbs are being formed. The effects of TCDD vary considerably with the stage of pregnancy. There are large differences in the susceptibility of particular mouse strains, effects in individual races sometimes varying by a factor of five times[34]. As well as inducing deformities TCDD often kills a proportion of mouse embryos[34].

Courtney and Moore[108] have observed kidney anomalies and stomach haemorrhages in the young of rats given daily doses of 0.0005 mg/kg of TCDD. Similar effects within species have been observed by Sparschu *et al*[116] and Khera and Ruddick[124]. However there is remarkably little information about effects on other animals. In experiments with hamsters, Collins and Williams[112] showed that the proportion of birth defects was correlated with the TCDD content of 2,4,5-T samples. On the other hand, Wilson[125] found that pregnant rhesus monkeys given daily doses of 40 mg/kg of 2,4,5-T containing 0.5 ppm TCDD produced entirely normal offspring.

Although news reports spoke of toxic and teratogenic effects in humans and animals exposed to defoliants in Vietnam, these were never confirmed. When the US Academy of Sciences team visited the country during 1971, 1972, and 1973, they were unable to find evidence of any significant increases in chromosomal aberrations, stillbirths, or birth defects[34]. While laboratory experiments provide convincing evidence of the teratogenic effects of TCDD in rats and mice these effects have never been observed in other species or outside the laboratory. There is, however, no question about the potential dangers of using 2,4,5-T containing the dioxin. It is fortunate that, by exercising reasonable care, it is possible to make grades of 2,4,5-T which are essentially free from impurities. All current production, both in Europe and in the United States, now contains less than 0.05 ppm of TCDD[34].

8. Teratogenic effects of 2,4-D and 2,4,5-T.

As has been related, the early reports of teratogenic effects of 2,4,5-T are suspect because they were based on results obtained with an impure sample of the herbicide. Courteney *et al*[46] fed pregnant mice large doses of technical 2,4,5-T at rates of from 46 to 113 mg/kg. As the material contained about 30 ppm of TCDD, the treatments would have included between .001 and .003 mg/kg of this dioxin, exactly the order of dose which is now known to cause embryo defects. Furthermore, the abnormalities which Courteney observed were identical with those caused by TCDD. It is possible that the teratogenic effects were entirely due to this dioxin. However, recent studies[107 108 109] show that very large doses of pure 2,4,5-T which does not contain detectable amounts of TCDD can sometimes affect unborn mouse embryos. The effects are less marked than those of TCDD and occur only when pregnant mice are given large doses of the herbicides. The most usual foetal abnormality is cleft palate but there may also be increases in the number of kidney anomalies[108]. For example, Roll[107] found that birth defects were increased by daily doses of 35 to 100 mg/kg of 2,4,5-T between the 6th and 15th days of pregnancy. An equivalent scaled-up daily dose for humans would be from 2.5 to 7.5 grammes, perhaps from 1 to 3 teaspoonfuls of a typical formulated product. The incidence of cleft palate has also been increased by single doses of 150 or 300 mg/kg. Teratogenic effects in other animals have not been established with certainty. In rats, malformations were increased by daily doses of 80 - 100 mg/kg but only slightly larger amounts of 2,4,5-T caused foetal and maternal deaths. Sparschu *et al*[110] and Emerson *et al*[111] did not observe teratogenic effects in rats from daily doses of 50 mg/kg. Foetal abnormalities have not been produced in several larger species including rabbits[111], sheep[113], and rhesus monkeys[114].

To bring these results into perspective it must be emphasised that many chemical compounds are teratogenic when administered in large amounts to experimental rodents. Many common drugs can cause birth defects, including various antihistamines and antibiotics[34]. Environmental conditions such as high levels of carbon monoxide or exposure to noise or heat can also have this effect[34]. Different species and even different strains within a species can vary considerably in susceptibility. The classic example is thalidomide which is highly teratogenic in humans but has only slight effects in rats or mice[34].

Recently Neubert *et al*[123] have demonstrated an interaction between TCDD and 2,4,5-T which may have important implications. At relatively low dose levels, mixtures of the compounds produced greater teratogenic effects in mice than would be expected from a knowledge of their separate activities. The presence of 2,4,5-T enhanced the activity of TCDD, and vice versa. In Neubert's experiments the interaction occurred only with mice and with TCDD concentrations of over 3 ppm. This high level of TCDD would not occur in the grades of 2,4,5-T now available. However, similar interactions in other species, involving lower levels of TCDD, cannot be ruled out. There may also of course be interactions with compounds other than 2,4,5-T.

Less work has been undertaken with 2,4-D. Studies which are summarised in the Academy of Sciences Report[34] suggest that moderate or large doses of some batches of 2,4-D may slightly increase the proportion of birth defects in rats and hamsters. However, dose response relationships were not very evident and most of the differences between treated and untreated batches were not statistically significant.

CHAPTER 4

The effects of 2,4-D and 2,4,5-T on other land organisms and the persistence of these compounds in terrestrial environments

The effects of 2,4-D and 2,4,5-T on humans and domestic animals have been discussed. While these effects are obviously important, so also is the activity of the herbicides towards other forms of life. Sometimes a compound may directly affect a particular organism. Often, however, pesticides act indirectly, for example, by bringing about changes in the food supply of birds and animals. A pesticide can directly or indirectly affect all parts of an ecological community from large plants and animals down to small micro-organisms. Several writers[32 127 128] point out that the full consequences of using pesticides are very difficult to predict but are often of great practical importance. Edwards and Van Dyke[128] stress the special need for research on the movement, accumulation and degradation of pesticides and, in particular, the transference of these compounds from plants to animals. The side effects of herbicides depend very much on the scale of usage. Obviously, extensive or repeated applications will usually have more drastic secondary effects than smaller scale uses.

For example, one would expect large differences between the effects of a military defoliation programme and those of ordinary weed control practices.

While it is not intended to discuss forest ecology in detail, it is worth emphasising that herbicide usage is only one of many factors which can affect woodland communities. For example fire, diseases, insect pests, and a wide range of human activities can all bring about sweeping changes which may be much greater than those due to herbicides.

1. **The effects of 2,4-D and 2,4,5-T on higher plants.**

Like their chemical relatives, 2,4-D and 2,4,5-T are always more or less active against broadleaved dicotyledonous plants but usually have little effect on grasses and other monocotyledons. The direct effects of the herbicide treatments used in forestry depend on the species which are present, the method of application, and the season. Summer sprays may be expected to severely injure most broadleaved plants. Winter bark treatments usually cause less injury, while cut bark applications, particularly injections, are unlikely to have direct effects on herbaceous species at any time. However while direct injury to wild flowers can usually be avoided, indirect effects almost always follow changes in the shade canopy. In Britain, the removal of shade, whether achieved by the use of herbicides or otherwise, is almost always followed by increased growth of grasses, brambles, *Rubus* spp, wild rose, *Rosa* spp, and coarse herbaceous species like thistles, *Cirsium* spp and rose bay willow herb, *Chamaenerion augustifolium*. A moderate amount of shade is essential for many of the less aggressive woodland plants.

Treatment of individual trees with 2,4-D or 2,4,5-T, for example by injections or bark sprays, are an effective means of selectively thinning woody plants. In some circumstances, these selective treatments are useful aids for managing recreation areas or nature reserves; they can, for example, be used to reduce heavy shading and so promote flowering of species like primrose or bluebell. The herbicides can also be used to increase the numbers of plants which provide food for birds and animals [129, 130]. In North America low doses of herbicides which kill the crowns of trees and induce basal sprouting are used to augment the amount of foliage which can be browsed by deer [130, 144]. 2,4,5 T sprays can be used to control gorse bushes growing in association with heather[35], a species which provides better shelter and food for game birds. Differences in the susceptibility of hardwoods to herbicides have been exploited to increase the growth of Dogwood *(Cornus)* and other species which are important to deer[130]. 2,4-D and 2,4,5-T have been used to eliminate competitive plants and so promote the growth of sweet fern, *Comptonia perigrina*, another North American species which is browsed by game[133]. The phenoxyacetic acid herbicides are often useful tools for the conservationist and game manager. However the elimination of what is considered to be an unwanted species sometimes leaves a vacant ecological niche which is quickly filled by even less desirable plants. For example in the southern United States the use of 2,4-D and 2,4,5-T to control sagebrush *(Artemisia)* often leads to the dominance of another woody species, *Chrysothamnus*[134].

2. **The persistence of 2,4-D and 2,4,5-T in plant material**

2,4-D and 2,4,5-T are not usually very persistent in plant tissues. As has been shown, esters usually break down rapidly to release the parent acids within a period of days or even hours[53, 54, 55]. 2,4,5-T is the more stable of the two acids. Slife *et al*[71] showed that while 75 per cent. of a sublethal dose of 2,4-D disappeared from cucumber plants within 24 hours, almost all of a comparable dose of 2,4,5-T was still present after 8 days. Klingman *et al*[97] found that 90 per cent. of a dose of 2,4-D applied as a spray to pasture had disappeared within 13 days. Breakdown takes place in dead foliage as well as in living plants. For example the apparent half life of 2,4,5-T in living and dead grass was from 1.6 - 2.6 weeks and 1.7 - 4.0 weeks, respectively[132]. From laboratory experiments with forest litter, Norris[135, 136, 137] concluded that 2,4,5-T is more persistent in dead vegetation than 2,4-D. While 88 per cent. of a moderate application of 2,4-D (2.2 kg/ha) disappeared from dead leaves within 12½ days, only 22 per cent. of a similar dose of 2,4,5-T was degraded within this period. However 90 per cent. of the 2,4,5-T was lost after 4 months[136]. Repeated treatment of forest floor litter with 2,4-D led to the phenomenon termed enrichment, that is, a rapid increase in the rate of degradation[136]. With 2,4,5-T this effect was not very evident.

3. The effects of 2,4-D and 2,4,5-T on wild animals and birds.

The effects of the herbicides on domestic animals have been reviewed in Chapter 3. Only a limited amount of information is available about the direct effects of 2,4-D and 2,4,5-T on wildlife; this in itself probably indicates that most wild animals and birds are not particularly susceptible. Newton and Norris[130] found that residues in the meat of deer from sprayed forests were always low, rarely reaching detectable levels. Norris[140] suggests that the herbicides are degraded by deer. Martin[141] states that, from one season's observations, 2,4,5-T treatments appeared to have no marked adverse effects on any nesting species of birds.

News of adverse effects of 2,4-D sprays on pheasant and partridge eggs received publicity as a result of a report published in 1970[240]. It was claimed that 0.5 kg/ha or 1 kg/ha of an unspecified 2,4-D formulation caused severe embryo mortality together with paralysis and morphological anomalies in the surviving chicks. However no numerical data was given relating to "control" (untreated) eggs. In a better designed series of trials, Somers[141] showed that there were no differences between untreated eggs and batches which had been sprayed with 11 kg/ha of formulated 2,4-D or 2,4,5-T - iso-octyl esters. Both the treated and the "control" eggs contained about the same somewhat high proportion of dead and abnormal chicks.

Herbicides can often benefit or reduce populations of particular species through effects on their habitat. Insofar as wildlife is concerned, the indirect effects of 2,4-D and 2,4,5-T are usually much more important than their direct effects. For example, the elimination of tall trees near power lines often leads to the formation of a stable plant association which includes bracken, sedge and blueberry, *Vaccinium* spp. This type of community provides much highly nutritious food and often supports more birds and animals than the hardwood dominated woodland which it replaces[142]. On the other hand, herbicide treatments can reduce populations of particular species. For example, the numbers of pocket gophers, *Thomomys bottae*, on a mountain area was dramatically reduced by spraying 2,4-D from the air[143]. This was probably due to a fall in the amount of food provided by herbaceous plants.

4. The effects of 2,4-D and 2,4,5-T on bees.

The use of 2,4-D and 2,4,5-T sprays has sometimes been followed by reports of mortality among bees. However, considering how widely these herbicides are used, reports of this kind are few and far between. There is good evidence that under certain circumstances both herbicides can be toxic. For example in experiments with caged bees Twinn[145] recorded up to 100 per cent. mortality following the addition of 6250 ppm of 2,4-D ester to nutrient syrup. However he observed that the 2,4-D solutions were disliked by the bees and quickly regurgitated. The presence of 2,4-D greatly reduced the consumption of syrup and many deaths were thought to be due to starvation rather than poisoning. The highest mortalities were in fact often associated with the lowest consumption of treated sucrose. Twinn emphasises the important point that laboratory studies generally overstate the toxicity of pesticides under practical conditions, in that bees are made to ingest doses of toxin which they would never assimilate of their own accord in the field. The hazards of most pesticides obviously relate more to the circumstances in which the products are used, than to their intrinsic toxicity.

In similar experiments, Morton and Moffett[146] fed 2,4-D, 2,4,5-T, fenoprop and a number of other herbicides to newly emerged honey bees. The herbicides were given in sucrose solution at concentrations up to 100 ppm. At these levels both 2,4-D and 2,4,5-T were relatively harmless. Other herbicides, including paraquat and cacodylic acid, were toxic at only 10 or 100 ppm. Diesel fuel and other oils used for making spray solution caused severe mortality during a period of 24 hours from spraying but thereafter lost their toxic properties, Many formulations of 2,4-D and 2,4,5-T were harmless when dissolved in water. At concentrations above 100 ppm the herbicides temporarily supressed brood production. Palmer-Jones[147], working in New Zealand, showed that normal spray applications of the butyl and butoxyethanol esters of 2,4,5-T had no effect on bees. He suggests that 2,4-D esters are normally also harmless but large scale applications such as those used in land reclamation schemes may present greater dangers.

Sprayed flowers often wilt within a few hours thereby inhibiting the secretion of nectar[148] This reduces the risk of injury very considerably. However, as a precaution, phenoxyacetic acids and other pesticides should not be applied to plants bearing open flowers. If this is not possible and there are extensive areas of flowers, for example, heather, beekeepers with bees in the vicinity should be warned.

5. The effects of 2,4-D and 2,4,5-T on other insects.

Reisch[145] has shown that the use of bark sprays containing 2,4,5-T for thinning beech sometimes increases the incidence of two beetles, *Anisandrus* (= *Xyleborus*) *dispar* and *Taphrorychus bicolor.* This is apparently due to the moist nature of the dead thinnings, which provides a suitable habitat for the beetles. Increased populations of the beetles are not found in living trees or trees which have been thinned by hand.

In laboratory study, Novy and Majumdar[150] recorded no differential genetic effects following the treatment of fruit flies *(Drosophilia)* with up to 100 mg/kg of 2,4,5-T. However, at this dosage the herbicide significantly reduced populations.

In a review, Way[92] points out that where effects on insects occur, they often take the form of population increases. For example the use of 2,4-D on cereals sometimes increases aphid populations. It is suggested that the herbicide may have toxic or repellent effects towards coccinellia beetles and other predators[92]. Ishii and Hirano[152] found that treatment of rice with 2,4-D increased the growth of stem borer larvae. This was attributed to changes in the nitrogen content of the vegetation. Davis[169] has shown that 2,4-D, 2,4,5-T and a growth regulator, maleic hydrazide, do not affect the prairie grain wireworm, *Ctenicera destructor,* when they are added to diet at huge concentrations of up to 50,000 ppm. Karg[160] observed no effects of 2,4-D, 2,4,5-T and MCPA on earthworms and enchytraeid worms.

6. The effects of 2,4-D and 2,4,5-T on fungi and soil micro-organisms.

Poppe[153] has shown that at the concentrations normally used for spraying, 2,4,5-T is toxic to mushroom mycelia. 2,4-D is however comparatively harmless. In a culture of the cultivated mushroom *Psalliota bispora,* fructification was not impaired by 2,4,5-T because the herbicide did not reach the layer of compost which contained the mycelia. However the field mushroom *Psalliota campestris* was seriously affected, because its smaller mycelial colonies occurred at shallower depths.

Many workers have found that at concentrations of up to about 100 ppm, 2,4-D and 2,4,5-T have almost no effect on soil fungi. Shennan and Fletcher[155], for example, observed only small effects from the addition of 10 ppm or 100 ppm of 2,4-D, 2,4,5-T, MCPA and other related herbicides to culture media. Very much higher concentrations, sometimes up to 10,000 ppm, were needed before growth was appreciably reduced. In Shennan and Fletcher's experiments 2,4,5-T was slightly more toxic to fungi and yeasts than 2,4-D or MCPA. Many other research workers have undertaken similar studies with a wide range of fungi, yeasts, bacteria and other soil living micro-organisms. While very high concentrations of the herbicides can undoubtedly depress the activity of particular strains or species, normal uses of 2,4-D and 2,4,5-T appear to have no permanent effects on most soil-living organisms. Populations of particular species may sometimes be temporarily reduced but recovery is usually rapid Studies in this field have been reviewed by Newman and Downey[156], Fletcher[157], Audus[158], and Edwards and Thompson[159]. Species which may be more than usually susceptible to 2,4-D or 2,4,5-T include certain nodule forming *Rhizobia*[158, 161] and *Bacillus subtilis*[160]. Smith[154] has recently reported that some mycorrhizal and root pathogenic fungi are affected by 5 ppm or 20 ppm of 2,4,5-T. However, this result is unusual. Newman and Downing[156] state that the herbicides have almost no effect on nitrifying bacteria. Gram-positive bacteria tend to be more sensitive than gram-negative types.[156] Some organisms are capable of adaption to media which contain large amounts of phenoxyacetic acids. Thus, certain *Rhizobia* can acquire an ability to tolerate high concentrations of 2,4-D and MCPA[162]. The rapid adaption of micro-organisms to media containing herbicides may explain some apparently divergent results. A *Streptomyces* species which became adapted to substances containing 2,4-D, 2,4,5-T and fenoprop (Silvex) was studied by Bounds and Colmer[163]. Moderate amounts of phenoxyacetic acid herbicides can sometimes actively enhance the growth of soil micro-organisms. For example Roberts and Bollen[164] found that 100 ppm of 2,4-D and 2,4,5-T stimulated the growth of certain moulds and Sankhla and Sharma[167] showed that 25 ppm of 2,4,5-T increased mycelial growth in *Aspergillus niger*, the fungus which causes Crown Rot of groundnuts. Audus[166] has demonstrated that a bacterium *(Arthrobacter)* from enriched soil can subsist on agar containing only 2,4-D, as the sole source of carbon.

7. The persistence of 2,4-D and 2,4,5-T in soil.

In general, 2,4-D and 2,4,5-T have little activity through the roots of plants[5] or towards most soil-living organisms. At first sight, therefore, the rate at which the herbicides disappear from soil may seem unimportant. However, the presence in the environment of any chemical with high biological activity may have unexpected consequences. The persistence of 2,4-D and 2,4,5-T in soil may therefore be very important. It has been studied extensively.

Many factors can effect the rate at which chemical substances are lost from soil, including the speed of chemical or microbial breakdown and losses by leaching or volatilization[51]. It is often difficult to separate these factors, for example to determine whether breakdown is chemical or due to living organisms. Studies of the kinetics of breakdown provide a possible means of distinguishing between these two types of degradation. The rate of disappearance of a herbicide may vary with the formulation which is used. As in plant material, the first stage of breakdown of esters is always hydrolysis to the free acid. This process is usually rapid. For example, Smith[169] reported that in a moist prairie soil at 25°C both the n-butyl and isopropyl esters of 2,4-D were almost fully hydrolysed within 1½ hours. The iso-octyl ester of 2,4-D broke down more slowly but hydrolysis was completed within 2 days. Under field conditions in Colorado, Burcar *et al*[170] found that the hydrolysis of 2,4-D iso-octyl ester occupied about 2 weeks. Hydrolysis is quickest in moist soils. It is not clear whether the degradation of esters is usually microbial or chemical. However the very rapid almost immediate disappearance of n-butyl and isopropyl esters reported by Smith[169] is more characteristic of chemical than microbial breakdown.

The free acids also have a relatively short life in soil. Klingman[51] states that when used at normal rates, i.e. at from 0.5 to 3 lb/acre (0.6 to 3.4 kg/ha), both 2,4-D and 2,4,5-T disappear within 4 or 5 weeks. By contrast, many herbicides can persist for a year or more[51]; 2,4,5-T often breaks down rather more slowly than 2,4-D[174 175] Breakdown is likely to be relatively slow under cold or dry conditions[172] or when very large doses are applied. However when normal doses of 2,4-D and 2,4,5-T are used detectible residues rarely persist for more than four or five months[159 171 173 174 175 176]. Early studies by Akamine[171] showed that the breakdown of 2,4-D in soil is favoured by alkalinity and by high populations of aerobic bacteria. As there was no correlation between the rate of breakdown and the organic matter content or absorptive capacity of soils, Akamine considered that the acids were probably degraded by microbial action. This theory is strengthened by the discovery that soil sterilization reduced the rate of herbicide loss[172]. Additionally it is found that the kinetics of 2,4-D and 2,4,5-T breakdown is typical of degradation by bacteria during a period of adaption to a new nutrient medium[175]. There is usually an initial stage during which the herbicides break down slowly. This is followed by a period of rapidly increasing loss, corresponding to a massive increase in the population of bacteria capable of attacking the herbicide. It is not known exactly how soils develop a bacterial flora which can metabolise novel chemical compounds such as 2,4-D and 2,4,5-T. However this effect, termed "enrichment", is often of great practical importance. It is a general phenomenon which occurs with many compounds besides 2,4-D and 2,4,5-T[158]. Broadly speaking, it may be said that the repeated use of any agricultural chemical is likely to lead to increases in populations of soil living bacteria which can degrade it.

Recent work on methods of disposing of surplus stocks of agent *'Orange'* shows that even massive doses of 2,4-D and 2,4,5-T can have a surprisingly short life in soil. Young *et al*[177] describe experiments in which the mixed 2,4-D and 2,4,5-T butyl esters were sprayed into trenches, at rates up to 4000 lb/acre (4400 kg/ha). 61 per cent. of the original amount was lost after 330 days. Both herbicides were broken down at about the same rate. In laboratory studies, Naidu[178] observed that most of a large application of 5 tons/ac (12000 kg/ha) of 2,4,5-T disappeared from calcareous soil during 56 days incubation. Naidu found that the respiration of micro-organisms was inhibited by pure 2,4,5-T but increased by formulated products. High concentrations of the herbicide reduced nitrification and the hydrolysis of urea but had little effect on the conversion of nitrogenous organic matter to ammonium salts.

Water soluble materials such as the amine salts of 2,4-D and 2,4,5-T might be expected to leach through soil profiles into underground water supplies. In practice however, downward movement is greatly limited by adsorption onto soil colloids and the rapid detoxifying action of soil micro-organisms. Thus, Edwards and Glass[218] observed only traces of 2,4,5-T in drainage from soil columns held in lysimeters, during a period of 14 months after applying 11.2 kg/ha of the herbicide to the soil surface. Adsorption onto soil colloids often restricts lateral, as well as downward movement. Studies in this field are discussed in Chapter 5.

8. The persistence and mobility of TCDD

Extensive research on the movement and persistence of TCDD in the environment has been carried out at the United States Department of Agriculture research centre at Beltsville, Maryland, Many results from this programme are summarised in the National Academy of Sciences Report[34]. TCDD is much more stable than either 2,4-D or 2,4,5-T. Thus, when the dioxin is incubated with soil, almost 60 per cent. of the original amount can be recovered after a year[179]. The rate of breakdown is not much affected by the soil type or the amount of TCDD which is present at the outset[179]. Downward movement through soil profiles does not occur even in sandy soils which are prone to leaching[34]. In Thailand, soil samples from land used by US forces for calibrating spray equipment in 1964 and 1965 still contained up to 0.02 ppm of TCDD in 1973[34]. This area had received altogether about 1000 kg/ha of 2,4,5-T some of which contained 30-40 ppm of TCDD. Small amounts of TCDD were also found in 1973 on an area of sandy soil in Southern USA, used for testing spraying equipment between 1962 and 1970[34].

Isensee and Jones[181] have found that plants can absorb TCDD both from culture solutions and soils. However, uptake is slow and there is at present no evidence of accumulation. There is little redistribution from older to younger parts of plants.[181] When treated material is transferred to fresh culture solutions the amount of TCDD in the foliage falls quite rapidly. Thus, TCDD was not detected in the seed of oat plants treated with the dioxin at an early growth stage. Minute amounts of TCDD were detected in the seeds of soya plants treated in this way but the quantities which were recovered were extremely small.

Matsumura and Benezet[183] have studied the bioconcentration of TCDD in model ecosystems. It appears that while transfer and accumulation of TCDD through food chains can take place, this process is much less marked than that occurring with DDT and other organochlorine insecticides. This is largely due to the low solubility of the dioxin in both water and fats and oils[34]. Of a number of organisms which have been examined, mosquito larvae are the most efficient concentrators of TCDD[183]. The rate of degradation in model ecosystems is always low[183]. Crosby *et al*[184] have shown that there is little breakdown of TCDD when suspensions in water or films on soil or glass are exposed to light. However, photodecomposition can occur in alcohol solutions, as a result of exposure to sunlight or ultraviolet radiation[184].

Jackson[185] has recently reported an effect of TCDD on plant cells which may bear on teratogenic effects in mammals. In experiments with cultured endosperm cells of the African Blood Lily *Haemanthus katherinae*, minute amounts of TCDD inhibited mitosis and caused cytological abnormalities. Purified 2,4,5-T did not have this effect. Other work[186] shows that TCDD may induce genetic mutations in strains of two bacteria, *Escherichia coli* and *Salmonella typhi*. Again, mutations were not observed when the bacteria were treated with pure 2,4,5-T.

The formation of TCDD during the preparation of 2,4,5-trichlorophenol has been described. There are, theoretically at least, several other ways in which dioxins could be produced. These include synthesis from trichlorophenol or other chlorinated phenols by the action of soil organisms, light or heat[182]. Kearney *et al*[179] found no TCDD or other dioxins following a 70 - day incubation of soils containing 2,4-D and 2,4,5-T. Similarly, irradiation of solutions of 2,4,5-T, 2,4-dichlorophenol or 2,4,5-trichlorophenol with sunlight or ultraviolet light did not lead to any detectable formation of dioxins[184 188 189]. On the other hand it is known that relatively large amounts of TCDD are produced when the sodium salt of 2,4,5-T is heated for several hours in an open tube, at 300°C - 350°C[34]. Buu-Hoi *et al*[190] have stated that TCDD may be formed when plant material which contains 2,4,5-T is burned. However, the authors of the Academy of Sciences Report regard this report as open to question[34]. The experimental methods used by Buu-Hoi and his colleagues are not fully described and there is doubt about the identity of the products which were collected. It is considered unlikely that dioxins could be formed during the combustion of material which contains 2,4,5-T in a dispersed form[34]. Johnson[191] has reported that TCDD is not produced when filter paper soaked in 2,4,5-T is burnt. He was unable to detect TCDD in the products formed by burning wood treated with 2,4,5-T, but his methods of analysis were obviously imprecise, since the limit of detection of TCDD was high, about 5 ppm. Johnson states that other combustion products interfered with the analysis.

Research now in progress at Beltsville and elsewhere may shed more light on the processes of TCDD formation and degradation. However, the evidence which is available suggests that TCDD only enters the environment as a product of side reaction which can occur during the industrial preparation of 2,4,5-trichlorophenol. This latter compound is mostly made as an intermediate for the manufacture of 2,4,5-T but is also used to a small extent for other purposes, for example, as a fungicide in cardboard and paper making. Some of these minor industrial uses of 2,4,5-trichlorophenol, which date back to before 1900, may have been a past source of TCDD contamination.

This chapter is concluded by quoting some quantitative data from the Academy of Sciences Report[34]. Firstly, it is calculated that the total amount of TCDD applied in Vietnam during the entire defoliation programme was probably about 112 kg (250 lb). Secondly, it is pointed out that the grades of 2,4,5-T now available contain well below 0.05 ppm of TCDD[34]. As the average annual production of 2,4,5-T is about 2.25 million kg (5 million lbs.) at most about 110 grammes (4 oz) of the new dioxin is being produced each year. This of course eventually becomes distributed over some millions of acres of land.

CHAPTER 5

The activity and persistence of 2,4-D and 2,4,5-T in aquatic environments

There are many ways in which herbicides can enter ponds and rivers. Sometimes, of course, these compounds are introduced deliberately to kill broadleaved water plants. Thus, 2,4-D is widely used against the very pernicious tropical weed water hyacinth[28]. It is also employed in temperate countries against emergent and floating water plants such as water lily *(Nuphar)* and *Nymphaea*[193]. While many other examples of the use of 2,4-D for controlling water weeds could be given, 2,4,5-T is now rarely used in aquatic environments. It was however formerly an approved herbicide for controlling broadleaved weeds in rice paddies[28].

In other circumstances, the herbicides may enter natural waters accidentally. For instance, when large tracts of land are sprayed from aircraft, some contamination of streams and ponds is almost inevitable. The most obvious route of entry is as stray droplets of spray solution but herbicides could obviously also reach waterways in surface run off from sprayed areas or through movement of water underground. While direct entry of herbicides into water can usually be avoided, these other types of contamination are much more difficult to prevent.

The presence of pesticides in water supplies may obviously have important direct consequences, for example where a stream is used for irrigation or for watering livestock. However, there may be other equally important but less obvious indirect effects. For instance, certain pesticides may accumulate through food chains and become concentrated in the flesh of fish. Effects on water plants could lead to changes in the flow rate of streams and rivers. By contrast with land situations, the effects of pesticides in water may extend many miles from the original point of contamination.

1. The effects of contaminated water on man and animals.

Toxicological studies with 2,4-D and 2,4,5-T have been reviewed in Chapter 3. House *et al*[32] suggest that a likely lethal dose of 2,4-D for a human may be of the order of 50 - 100 mg/kg, perhaps somewhere between 4 and 10 grammes for an adult. A simple calculation shows that wh water 0.3 m deep is exposed to spray applicatior of 3 kg/ha of pesticide, the resulting solution contains only about 1 ppm of the compound, or gramme per cubic metre. When the herbicides ar used at normal rates, therefore, contaminated drinking water probably presents almost no direc danger to humans or animals. Even water containing 100 ppm of 2,4-D or 2.4,5-T could probably be drunk without ill effects. However, where published criteria are available, very much lower levels of 2,4-D and 2,4,5-T are specified. For example, in the United States the maximum permissible concentration of either herbicide in water which is used for public supplies is only 0.1 ppm[194].

Although both 2,4-D and 2,4,5-T have a somewhat unpleasant "medicinal" taste and smel complaints of taints in water after spraying are generally due to impurities or breakdown produc rather than the herbicides themselves. For exampl off-flavours in water associated with 2,4-D spraying are generally due to 2,4-dichlorophenol. Like 2,4-D, this compound does not present any real health hazard. However it imparts an objectionable taste and smell to water even at concentrations down to 0.002 ppm. Faust *et al*[195 196 197] have found that while 2,4-dichlorophenol can be produced by the breakdown of herbicides, it is more usually introduced into water as an impurity of 2,4-D[196]. Under anaerobic conditions 2,4-dichlorophenol persists for long periods but it breaks down rapidly when water is exposed to air, particularly when organic matter is present[197]. Many other compounds can also impart unpleasant flavours to water. In particular, organochlorine products are often formed when water which contains organic matter is treated wit chlorine to destroy disease carrying micro-organisms. Taints from this cause are most commo in wet autumn weather when leaves are washed into streams and reservoirs, giving rise to unusually high organic matter contents.

2. The movement and persistence of 2,4-D and 2,4,5-T in water.

Most evidence suggests that the herbicides break down in water quite rapidly. For example, following the introduction of granular 2,4-D into a pond to provide a theoretical initial concentration of 1.3 ppm, Frank and Comes found that concentrations in the water were always low, the maximum being 0.067 ppm at 18 days. The 2,4-D content of mud at the bottom of the pond was higher, reaching 5 ppm on the day after treatment. However, as in the water, these residues declined rapidly to below 0.1 ppm at 36 days[198]. In a similar study, 4.4 kg/ha of 2,4-D amine salt was applied to the surface of a lagoon near New Orleans, to control water hyacinth[199]. Up to 1 ppm of the herbicide was recovered from parts of the lagoon on the day after spraying, but thereafter the concentration fell rapidly to 0.02 ppm after 31 days. Degradation occurred at about the same rate in samples of water from the lagoon which had been placed in open vessels[199].

Smith and Isom[200] applied 2,4-D esters at up to 110 kg acid equivalent per hectare, to control water weeds on 8000 acres of reservoir in the Tennessee Valley. Following this treatment, the highest 2,4-D concentration recorded at the intakes of water treatment plants was 0.002 ppm. In most cases, the level was much lower. In Britain, Robson[201] showed that the pattern of 2,4-D degradation in natural water resembles that in soils. He found the breakdown was at first slow but this was followed by a period of much more rapid degradation. When a second dose of the herbicide was added to the water it disappeared quickly. These results suggest that, as in soils, breakdown is mainly due to biological rather than chemical action. It is clear that "enrichment" can occur, that is, the microbial flora of ponds or rivers can become adapted to 2,4-D, which they use as a source of energy.

There have been few comparable studies with 2,4,5-T. However, many workers have examined the 2,4,5-T content of streams originating from sprayed areas. For example, Brown and Mackenzie[214] applied 2,4,5-T esters as basal bark sprays to about 30 acres of a small forested catchment in Sussex, extending in all to 68 acres. Samples of surface runoff water were collected from the start of the work until 4 days after its completion. 2,4,5-T contents of up to 0.15 ppm were recorded, peak concentrations occurring at times of heavy rain. In the United States, Sopper *et al*[203] showed that the use of 2,4,5-T to control vegetation along stream banks sometimes resulted in very high local concentrations in water, of up to 40 ppm. However these high concentrations were only recorded just after spraying in water immediately adjacent to sprayed stream banks. No serious contamination was detected downstream. It was concluded that if normal care was taken during spraying, 2,4,5-T could be safely used on catchments serving public water supplies.

Many similar studies have been carried out in the Western United States by Norris and his associates[204 205 206]. Those workers have also found that the highest concentrations in water occur just after spraying. For example, they found that aerial application of 2.2 kg/ha of 2,4,5-T or a 2,4-D/2,4,5-T mixture sometimes led to local concentrations in streams of up to 0.8 ppm. However, these concentrations occurred only within sprayed blocks of forest. Downstream herbicide contents were much lower, usually below 0.01 ppm. Norris stresses the importance of stream flow as a factor which reduces the concentration of contaminants[205]. He found that heavy rainfall during the autumn and winter after spraying did not cause any sudden increase in the herbicide content of river waters[205 206]. Small amounts of 2,4,5-T were sometimes detected as much as 15 weeks after spraying but usually the herbicides persisted for only a week or so[205]. However, relatively large amounts of 2,4-D were occasionally recorded in streams originating from marshes within sprayed areas[205]. Norris points out that marshes present unusual hazards in terms of potential stream contamination, in that a slight rise in the water table shortly after spraying can release relatively large amounts of herbicides into waterways[205]. The danger is obviously greatest with oil solution of herbicides, which float on water surfaces.

Trichell *et al*[216] examined losses of 2,4,5-T in the runoff from sprayed plots subjected to simulated rainfall. Much more 2,4,5-T was present in the runoff from grass covered plots than in runoff from bare earth. When water containing 2,4,5-T was made to flow over soil, its herbicide content was much reduced, presumably because of adsorption. In similar studies, Edwards and Glass[218] found that only about 0.05 percent of a surface application of 11.2 kg/ha of 2,4,5-T was removed in runoff. Most of the herbicide loss occurred during a period of 32 days after treatment.

In general, esters are more likely to be removed in runoff than amine salt formulations. When simulated rain was applied to sandy soil sprayed with herbicides, losses of a 2,4-D ester and amine in runoff were 27 percent and 3 percent of the total dose, respectively[219]. While esters-in-oil may be floated from soil on the surface of the water, water soluble formulations are quickly carried into the soil and absorbed. The greatest losses in runoff usually occur during the first few minutes of rainfall[218 219].

Many other workers[202 207 208 210 211 212] have examined the practical consequences of treating vegetation on catchment areas with herbicides. Normal uses of 2,4-D and 2,4,5-T rarely cause detectable contamination of water supplies. However, while only small amounts of the herbicides occur in runoff or drainage, there may be marked indirect effects on both water quality and yield. For example, the presence of large amounts of dead vegetation on catchments can increase the organic matter content of surface water while the removal of plant cover from steep slopes can lead to excessive runoff and erosion. The reduction of transpiration by trees and shrubs and the replacement of woody growth by grasses often effectively increases the yield of water for public supplies. Thus, Reinhart[211] showed that selective control of woody plants with 2,4,5-T can augment water yields from May to October by as much as 2,200,000 litres per hectare (200,000 gpa). Similarly, Pierce[221] found that removal of brush increased stream flow by a factor of about 5, in this instance without noticeably affecting water quality.

3. Contamination of water by oils.

Herbicidal spray solutions may contain up to 90 percent of oil,[208] and in the past have been applied at volume rates of up to 1100 litres/ha[25]. Tarrant and Norris[208] have reviewed field and laboratory studies on contamination of water by oils, particularly diesel fuel. In experiments in Germany with C^{14} labelled oils Böning *et al*[213] observed that spray applications of oil 20 times greater than those normally used in forestry were not leached through soils into the ground water. The oil penetrated loam or sandy loam soils only to a depth of about 50 cm and was almost completely degraded after two years, the C^{14} activity being incorporated into the soil organic matter. Soil micro-organisms were unaffected. In Britain, Brown and Mackenzie[214] were unable to detect oil in runoff after using 630 gallons (2800 litres) of diesel fuel for basal bark and cut-stump treatments on 30 acres of woodland. Similarly, no oil was observed in streams after a plantation in Germany was sprayed with 15 - 20 gpa (16 - 22 litres/ha) of diesel fuel[215]. The deliberate addition of 3 litres of diesel oil per minute to the upper reaches of a stream led to high local concentrations in the water, of up to 1 per cent However, at a point 1 mile downstream the oil content was only 1 ppm[215].

In laboratory studies Linden *et al*[217] applied diesel oil to the surface of columns of soil, at rates of up to 5000 litres/ha. The columns were then irrigated with 100 mm of water. It was found that at application rates such as are normally used in forestry, oil did not penetrate the soil at all. Only very much larger amounts of oil could be carried into the soil columns by the action of water. Thus when 5000 litres/ha of oil was applied, only 9 ppm and 1 ppm were detected at the respective depths of 0.1 m and 0.2 m.

4. The effects of 2,4-D and 2,4,5-T on aquatic plants.

As on land, the effects of 2,4-D and 2,4,5-T on water plants vary considerably. In general, the spray solutions which are used for woody plant control are likely to kill or injure most broadleaved emergent or floating aquatic species, including water lilies and watercress, *Rorippa* spp. The compounds have little effect on reeds, grasses, and most other monocotyledons. Submerged weeds, whether dicotyledons or monecotyledons, are not much affected by 2,4-D or 2,4,5-T[193]. Very dilute solutions containing one or two parts per million of the herbicides, such as might occur in ponds or streams exposed to spray drift, have little effect on most water plants. Most algae and phytoplanktons are resistant to the herbicides; this is of considerable significance because these simple plants are the food of many fish and aquatic animals. Vance and Smith[222] have shown that the green algae *Scenedesmus quadricauda, Chlamydomonas eugametes* and *Chlorella pyrenoidesa* can tolerate 2,4-D or 2,4,5-T concentrations of up to 200 ppm. Butler [227] has found that 1 ppm of 2,4-D or 2,4,5-T does not affect the growth of phytoplankton in natural waters. Pierce [228] [229] observed that plankton populations were not reduced by 2,4-D granules, when these were used at rates which provided a theoretical concentration in water of from 1.4 ppm to 6.2 ppm.

5. The effects of 2,4-D and 2,4,5-T on fish.

The effects of 2,4-D and 2,4,5-T formulations on fish have been extensively studied. In general the herbicides are only moderately toxic to fish. They are much less poisonous than many other commonly used pesticides. Individual species vary considerably in susceptibility, but many fish can tolerate remarkably high concentrations of 2,4-D or 2,4,5-T. For example, 50 percent of a stock of bluegill sunfish *Lepomis macrochirus* survived 24 hours exposure to 900 ppm of either compound formulated as amine salts[223] . Even rainbow trout, normally very sensitive to pollution, were unaffected after being placed for 48 hours in a 12 ppm solution of 2,4,5-T acid[224] . While 2,4,5-T is often rather more toxic than 2,4-D, differences between the herbicides are generally less marked than differences between individual formulations. Esters are usually much more injurious than water soluble formulations. Thus, in aquarium experiments, the propylene-glycol-butyl-ether (PGBE), butyl and butoxyethanol esters of 2,4-D were toxic to young rainbow trout at only 1.1 ppm[225] . Only slightly higher concentrations of these esters were lethal to bluegill sunfish kept in tanks. Hughes[235] suggests that the harmful effects of many esters may often be due to the materials used to formulate them, rather than the esters themselves.

There are often large differences between the results obtained by different workers. Individual samples of the same type of product often differ widely in toxicity, particularly where trials are conducted in small aquaria. Usually the herbicides are much less toxic under more natural conditions where there are opportunities for adsorption onto mud or organic matter. Thus, while 2 ppm of the PGBE ester of 2,4-D is very toxic to bluegills in aquaria [223] much higher concentrations have been found to be almost inactive towards this same species under pond conditions [226] .

Other toxicity studies involving fish have been reviewed by Way [92] and Mullison[231] . In general, it appears that under natural conditions most fish species can tolerate 2 ppm of 2,4-D or 2,4,5-T. This is the order of concentrations which would be obtained by spraying a somewhat high dose of herbicide (5 kg/ha) onto water 0.3 m deep. In practice, much higher local concentrations of 2,4-D or 2,4,5-T would probably have little effect. Under natural conditions fish can usually move away from areas of high herbicide concentration. In flowing streams, herbicides become diluted quite rapidly, often within a few metres [205]

In Britain the use of 2,4-D to control heather near lakes or trout streams has sometimes aroused concern. All the available evidence suggests that this and other forestry or agricultural uses of phenoxy herbicides are most unlikely to have direct effects on trout. In normal circumstances very little herbicide enters streams during or after spraying. In this context it should be noted that 2,4-D amine formulations are cleared under the Pesticides Safety Precautions Scheme for use in water and are often employed for weed control in lowland trout rivers.

Recently the National Academy of Sciences Report has described the effects of military uses of defoliants on Vietnamese fisheries. Observations made during and after the defoliation programme suggest that the very large quantities of 2,4-D and 2,4,5-T which were sprayed onto mangrove swamps had no adverse effect on fish catches in estuaries[34] In fact, these slightly increased during the period from 1962 to 1969[34] . The increase was attributed to the presence of decomposed organic matter in the water, which provided a better food supply for the fish.

The possibility of the herbicides being metabolised by fish does not seem to have been investigated. However it is clear that fish do not accumulate 2,4-D or 2,4,5-T. These compounds are in fact often eliminated rather rapidly. Thus, Cope *et al* [226] detected only 2 ppm of 2,4-D in bluefish on the day after the herbicide was added to pond water to provide a theoretical concentration of 10 ppm[226]. A little 2,4-D was present in the fish at 3 days after the treatment but no residues were detected at later dates. Rather unexpectedly, the addition of 2,4-D significantly increased the growth of the fish. This was thought to be due to a release of nutrients into the pond following the death of aquatic plants. In similar experiments Coakley *et al*[230] found only 0.3 ppm of 2,4-D in fish (*Lepomis* species) 3 days after applying 33 kg/ha of the herbicide to the surface of a pond.

The possibilities of indirect effects on fish have already been mentioned. In general these are likely to be much more important than direct effects. Sometimes, as in the experiments just cited,[226] fish growth may be increased by herbicide treatments. However, adverse effects are also possible, for example where vegetation control increases flooding and silting and so destroys the habitat of particular species. When large amounts of dead vegetation are present, fish and other aquatic creatures can be adversely affected by a reduction in the oxygen content of water. Deoxygenation can be caused by the death of water plants which contribute oxygen by photosynthesis but is more often due to the respiratory use of oxygen by bacteria which break down dead vegetation. In aquatic weed control, the effects of deoxygenation are usually much more serious than any direct effects of herbicides on fish.

6. The effects of 2,4-D and 2,4,5-T on other water animals.

In general, 2,4-D and 2,4,5-T acids are only mildly toxic to crustaceans, molluscs and aquatic insects. As with fish, esters are usually much more harmful than the acids or most water soluble formulations. Thus, water fleas *(Daphnia)* can tolerate 100 ppm of 2,4-D acid but are killed by 1 ppm of 2,4-D PGBE ester[232]. Similar differences have been observed with shrimps *(Cypriodopsis)*,[232] scud *(Gammerus)*,[232] sowbugs *(Asellus)*,[232] crayfish *(Orconectes)*[232] and oysters [233]. The PGBE ester appears to be unusually toxic. For example, Butler[233] reports that while 2 ppm of 2,4,5-T acid has no effect on oysters, shell growth is reduced by 0.14 ppm of the herbicide formulated as this ester. Lhoste[234] also has observed adverse effects of 2,4-D and 2,4,5-T ester on crustaceans, aquatic insects and molluscs, at concentrations between 0.1 ppm and 3.3. ppm. Unlike fish, molluscs sometimes accumulate the herbicides from their surroundings. Thus, oysters placed in water containing 0.1 ppm of 2,4-D did not suffer mortality but after 7 days contained 18 ppm of the herbicide. The 2,4-D disappeared when the oysters were transferred to uncontaminated water. A fresh water mussel, *Elliptica crassidens* is also known to be capable of accumulating 2,4-D[200]. Way [92] has discussed the activity of the herbicides towards shellfish. He suggests that the relatively greater susceptibility of many molluscs, as compared with fish, is due to their sedentary habit of existence. Under natural conditions, fish can often quickly move away from contaminated waters. However, while molluscs may be more prone to injury than fish, it appears that only ester formulations present any appreciable danger. Even in the least favourable circumstances, contamination following normal uses of esters for woody weed control would probably not have lasting effects on most species.

The effects of the phenoxyacetic acid herbicides on frogs have not been examined extensively. However Cooke[236] has shown that tadpoles are uninjured by 48 hours exposure to 50 ppm of 2,4-D. At the end of this period, the tadpoles did not contain detectable amounts of herbicide. Similarly, Western Chorus frogs have been found to be almost unaffected by 100 ppm of 2,4-D dimethylamine salt[34].

7. The incidence of TCDD in water.

The very low solubility of TCDD[34] and its immobility in soils [179] [181] greatly reduce the possibilities of the compound being transferred from sprayed areas to streams in runoff or in underground water. It is thought that measurable contamination with TCDD could occur only as a result of spray droplets containing the dioxin falling directly into waterways. This undoubtedly happened in Vietnam, when mangrove forests along rivers and estuaries were sprayed extensively from the air. However so far as is known, TCDD has never been detected in samples of natural water, either from Vietnam or elsewhere. This is surprising, in view of the fact that extremely sensitive analytical methods are now available [220].

Work on the mammalian toxicity of TCDD has been reviewed in Chapter 4. The very low solubility of the compound almost rules out acute hazards from contaminated water. Even a saturated solution contains only 0.0002 mg per litre while the lowest LD_{50} value which has been reported (for guinea pigs) is about 0.0006 mg per kg [123]. Subacute, particularly teratogenic effects from contaminated water also appear to be almost out of the question.

TCDD is very toxic to many species of fish. Thus, Miller *et al* [237] have shown that 24, 48, or 96 hours exposure to 0.000056 ppm of the dioxin kills young salmon. Exposure to one tenth of this concentration of TCDD causes about 55 per cent mortality. The duration of exposure appears to be less important than the dioxin concentration. As might be expected, small fish which have a large surface area to weight ratio are more susceptible than larger ones. Certain other aquatic creatures appear to be less sensitive. Thus, a saturated solution containing 0.0002 ppm of TCDD has no effect on the pupation of mosquito larvae and only slightly reduces the rate of reproduction of water snails[237].

By using new and extremely sensitive analytical methods, Baughman and Meselson [220] have demonstrated TCDD contents of up to 0.0008 ppm, in the body tissues of river fish collected in South Vietnam during August and September 1970, 6 months after the last recorded use of defoliants containing 2,4,5-T. This result not only shows that fish can accumulate TCDD but confirms other evidence of the slow rate of breakdown of the dioxin under natural conditions. Baughman and Meselson also found smaller amounts of TCDD in fish and prawns from coastal waters [220]. This discovery led to the impounding of large amounts of frozen shrimps which had been exported from Vietnam to Japan [34]. Further evidence of bioconcentration in aquatic situations is provided by Matsumura and Benzets studies with model ecosystems, discussed in Chapter 3. Accumulation of the compound through food chains is not as marked as that which occurs with DDT and similar organochlorine insecticides. However with a compound which is as toxic and resistant to breakdown as TCDD even inefficient concentration may be a serious matter. Further studies on the fate of TCDD in aquatic situations are in progress[34].

CHAPTER 6

The overall picture

Like all systemic herbicides 2,4-D and 2,4,5-T are compounds of high biological activity. However, this activity is very specific, the herbicides having strong effects on only one type of organism, dicotyledonous plants. While it is true that concentrated solutions can be toxic to other forms of life, there is a great difference between the very distinctive effects of minute amounts of the herbicides on dicotyledons and their much less pronounced and less specific activity towards other organisms. While sprays containing only a few parts per million of 2,4-D or 2,4,5-T are lethal to many broadleaved plants, much stronger solutions are often virtually inactive against monocotyledons, insects, birds, fish and mammals.

In discussing the possible side effects of 2,4-D and 2,4,5-T, a distinction can be drawn between hazards linked with the diluted solutions applied with conventional sprayers and those associated with undiluted concentrates and low volume applications. The latter, which are steadily becoming more important, obviously involve the use of much stronger solutions than have been generally used previously.

Diluted spray solutions containing only 1 per cent or 2 per cent of active herbicide may have unintended effects on plants but probably present almost no direct danger to other forms of life. 2,4-D and 2,4,5-T have a strong medicinal taste and smell so that tainted food or water is usually rejected. However even if contaminated material is ingested, the quantity which must be consumed before toxic symptoms develop in humans or animals is so large as to almost rule out poisoning. The herbicides are not in any sense cumulative poisons. Elimination of small doses is usually completed within a very short period. The compounds are unlikely to appear in meat or dairy produce, and are never accumulated through food chains. They break down rapidly in soil, plant material, and water. While 2,4-D and 2,4,5-T can adversely affect fish, molluscs and bees, the danger to these organisms is usually slight if normal care is taken during spraying. Fish can normally swim away from water which contains the herbicides. Bees obviously find contaminated nectar or pollen distasteful and if possible will usually avoid sprayed areas.

While dilute solutions present little if any direct danger other than to plants, concentrates must be regarded as being moderately poisonous. Some toxic effects which were attributed to 2,4,5-T were probably due to TCDD poisoning. This contaminant which is discussed separately, is rigorously kept to a very low level in current manufacture of 2,4,5-T. However setting aside any effects of TCDD, it is clear that large doses of concentrates can cause illness and death in both animals and humans. In a small proportion of people, contact with 2,4-D can bring about the condition termed peripheral neuropathy. A case of suicide following 2,4-D poisoning is on record. 2,4,5-T is probably about as poisonous, although there are virtually no documented reports of serious illness being caused by this compound. Like other poisons, 2,4-D and 2,4,5-T concentrates should always be kept in a safe place and handled by only responsible people. The greatest risks associated with the herbicides are probably those which involve accidental spillage or malicious interference with drums of concentrate. Thus, the emptying of a 40 gallon drum of 2,4-D or 2,4,5-T ester into a river would probably render the water undrinkable and kill susceptible plants, fish, and other water creatures for some distance downstream.

Very low volume techniques are steadily becoming more important. As these involve the use of relatively strong solutions or even undiluted concentrates, the potential risks to spray operations are greater than with the older medium or high volume sprays. In collaboration with the Pesticides Safety Precautions Scheme, the Forestry Commission has drawn up rules for low volume spraying which include provision for face masks and other protection[78 214]. However while low volume techniques involve potentially greater risks to operators, once the herbicides are distributed there are no real differences in the side effects associated with the two types of application. Both provide similar herbicide concentrations in vegetation, soil and water.

Although most direct side effects of the herbicide are small and easily avoidable, important changes in bird, animal and insect populations usually follow changes in plant communities. This is of course true whether the plant communities are altered by herbicides or otherwise. Thus, the elimination of heather usually reduces numbers of bees. Population changes may be in either direction; the use of 2,4,5-T and 2,4-D as management tools to make an area either more or less suitable for particular bird or animal species has been discussed. It is often impossible to predict the full consequences of herbicide applications but the important point which must be made is that indirect effects of 2,4-D and 2,4,5-T are often much greater than their direct effects. The possibility of unexpected indirect effects should be borne in mind whenever pesticides are used. In general, the practice of spraying large areas as a single operation should be avoided. Programmes which involve the sequential treatment of a number of smaller blocks are always safer.

The aspect of 2,4,5-T safety which has received most attention is undoubtedly its teratogenicity. Some early reports of 2,4,5-T causing birth defects followed the use of impure samples of the herbicide which were heavily contaminated with TCDD[46]. More recently however teratogenic effects of 2,4,5-T have been demonstrated in rodents[107 108 109]. It is important to consider this result in its proper perspective, particularly in the light of similar studies with other compounds. The effects only occur when very large doses of pure 2,4,5-T are administered to mice and rats at particular stages of pregnancy. A great many materials including antibiotics, tranquillizers, Vitamin A, caffeine, and metallic salts, have similar effects in strains of rodents used for laboratory tests[34]. Teratogenicity can be caused by exposure to adverse environmental conditions such as noise or dehydration. This effect of 2,4,5-T has never been demonstrated in other species. However if one scales up the treatments which are teratogenic in mice, the order of doses of pure 2,4,5-T which might be needed to produce similar effects in humans or domestic animals are seen to be very large indeed. The equivalent daily dose for a human would be around 5 grammes of 2,4,5-T acid, perhaps a large spoonful of a typical undiluted concentrate. Even if herbicide concentrates were handled very carelessly, accidental consumption of such large amounts would appear to be almost out of the question. It would be quite impossible for a person to ingest these quantities of 2,4,5-T unknowingly from contaminated food or water.

While all the evidence which is available shows that 2,4-D and 2,4,5-T are only mildly poisonous, there is little doubt about the extremely poisonous nature of TCDD. Other dioxins which can occur in badly made 2,4,5-T are probably also hazardous. The materials not only have very high acute toxicity but are teratogenic and extremely resistant to breakdown. They are often retained in body tissues and can be accumulated through food chains.

The broad conclusion from this survey must be that well made 2,4-D and 2,4,5-T are safe herbicides when they are used with care and forethought. Perhaps the best evidence for this statement is the fact that both compounds have been very widely used for over 25 years with very few records of injury to human or wildlife. 2,4,5-T in particular has received a great deal of adverse publicity and is still regarded as suspect by many uninformed However detailed discussion of the dangers of dioxins is now largely superfluous, since all current production of 2,4,5-T is essentially free from those impurities[34]. All 2,4,5-T which has been made during the past 5 or 6 years has contained less than 0.05 ppm of TCDD[34]. For practical purposes the amount of new dioxin which is being introduced into the world as a constituent of 2,4,5-T is insignificant, perhaps 110 g annually from the entire production of the herbicide in North America[34]. Most evidence suggests that TCDD is not formed in the environment from 2,4,5-T, 2,4-D or trichlorophenols. The residues which are present from past uses of 2,4,5-T, particularly from the defoliation programme in Vietnam, are still a matter for concern. However, it is clear that TCDD is eventually degraded to less toxic materials even though breakdown is slow. Although bioconcentration can occur, this process is not very marked. The total amount of TCDD in the world is probably much less than it was in 1970 at the conclusion of the military defoliation programme.

people. It is true that samples which contain dioxins can be dangerous. However this is the only aspect of the 2,4,5-T story, as understood by most members of the public, which has any real foundation of truth. Adverse public opinion about the properties of 2,4,5-T (and, by implication, 2,4-D) seem to have arisen from:-

1. Public opposition to the use of all chemical weapons, including defoliants, during the war in Vietnam.
2. The habit of television and newspaper commentators of treating all pesticides as a more or less uniform group of compounds, all equally poisonous and persistent. Obviously, safe compounds are not usually newsworthy.
3. Growing public awareness about the effects which human activities, including the use of pesticides, can have on wildlife.
4. The fact that reports of birth defects due to TCDD and 2,4,5-T appeared against the tragic background of the thalidomide story.

It is true that 2,4-D and 2,4,5-T like most other materials, can be dangerous if they are misused. They are always sold with a set of instructions for use, which have been carefully drawn up after discussion between the firms who market the products and representatives of several Government departments. Approved methods of use always take into account the safety of operations, the safety of crops and the possibilities of side effects on other organisms. Any material can be dangerous in the hands of fools or vandals. In the final analysis it is the care and forethought of the user which determines whether a compound has beneficial or harmful effects.

This chapter is concluded by quoting from a recent article by the editor of Weed Science, the Journal of the Weed Science Society of America.

"The 2,4,5-T story might be described as a comedy of errors, except there is nothing funny about it. It's a depressing story of how decisions about pesticides are based on emotion, political considerations and special interests rather than on scientific facts. It tells how biased views and unfounded charges might cause a valuable herbicide to be ruled unsafe and illegal."

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Abscission Leaf fall following the formation of an abscission or "separation" layer at the base of the leaf petiole.

Acid equivalent. The content of active ingredient expressed in terms of the parent acid.

Adjuvant. A substance, usually inactive when applied alone, which is added to a herbicide to increase its activity or to improve its storage or handling properties.

Agent "Orange". A mixture of equal parts by volume of the undiluted n-butyl esters of 2,4-D and 2,4,5-T, used in military operations in Vietnam.

Amines. Organic compounds which may be regarded as derivatives of ammonia, in which one or more hydrogen atoms is replaced by an alkyl radical. Like ammonia, amines are basic in character.

Amine salt. A compound formed by reacting an amine with an acid.

Auxin. A generic name for compounds characterised by their capacity to induce elongation in the cells of shoots.

Bark spraying. The application of a herbicide spray to the bark of woody species.

Basal Bark Treatment. A method of killing trees and shrubs which involves applying herbicide solution to the bark at the base of the stem.

Bioconcentration. The accumulation of a compound by plants or animals from the environment in which they live.

Broadleaved plants. Dicotyledonous plants, including both woody and herbaceous species.

Concentrate. (of a herbicide). An undiluted preparation usually containing at least 20 per cent of active ingredient.

Cosolvent. A material, usually a liquid, which improves the solvent properties of another liquid.

Cut-bark treatment. A type of treatment which involves applying herbicides to cuts made through the bark to the depth of the vascular tissue. Includes Frill girdling, Notching and Injection.

Cut-stump treatment. A treatment applied to the freshly cut surface of a stump, with the object of preventing regrowth.

Cuticle. A superficial, often more or less waterproof non-cellular layer, which covers all aerial parts of higher plants.

Defoliant. A chemical which causes leaf fall when applied to a plant.

Degradation. The breakdown of a chemical product, such as a herbicide, to simpler chemical structures.

Derivative (of a herbicide). A compound such as a salt or ester which is made by reacting the parent herbicide with another chemical. Most herbicide derivatives retain the herbicidal properties of the parent material.

Desiccant. A compound which promotes the loss of moisture from plant tissues.

Detergent. A particular type of surface active agent.

Dicotyledon, dicotyledonous plants. The larger of the two groups into which flowering plants are divided; distinguished from monocotyledons by the presence of two seed leaves (cotyledons) and by other structural features, particularly the occurrence of net-veined leaves.

Dioxins. A group of compounds which contain two benzene rings linked by adjacent oxygen bridges. The best known example is 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).

Directed sprays. Sprays applied with a boom or lance which can be angled or directed to avoid crop plants.

Dormant shoot spray. A spray applied when the shoots of a woody weed or crop species are dormant.

Drift. Movement of droplets of spray solution (spray drift) or vapour (vapour drift) caused by air currents.

Ecosystem. A community of organisms interacting with one another and with the environment.

Emulsion. A suspension of small droplets of one liquid in another, e.g. oil in water.

Emulsifying agent, emulsifier. A surface active agent which reduces interfacial tension and so promotes the formation of an emulsion.

Enrichment. The development of a particular microbial population in the presence of a continuing supply of a particular substrate.

Enzyme. A protein which promotes or catalyses a biochemical change without itself being consumed in the reaction.

Epinasty. The more rapid growth of the upper side of an organ such as a leaf or shoot, which leads to downward curling of the organ. Often incorrectly applied to describe any curling or twisting of leaf blades, stems, etc.

Ester. A group of compounds which can be made by reacting an acid and an alcohol together.

Foliage spray. A spray applied to the leaves of a weed. In practice the branches and stem are often also sprayed.

Food chain. A chain of organisms existing in a natural community through which energy and other materials may be transferred. Each link feeds on the one preceding it and is in turn eaten by the link which follows it.

Formulation. (1) The process of preparing a herbicidal compound for practical use, (2) a preparation containing a herbicide in a form suitable for practical use.

Frill girdle, Frill. A series of overlapping cuts made downwards into the bark of a tree trunk to form a girdle to which herbicide is applied.

Granule. A type of formulation for dry application, in which granules serve as carriers for the herbicide.

Hardwood. A broadleaved, dicotyledonous woody species, such as oak, ash or hawthorn.

Herbicide. A chemical which can kill or suppress the growth of certain plants.

Hydrolysis. A chemical reaction involving the uptake of water. In herbicide science, often the decomposition of an ester in the presence of water, to release the parent acid and alcohol.

Hydrophilic. Describes compounds with an affinity for and often soluble in water.

Injection A method of treating woody growth which involves the use of special equipment to inject herbicide into the vascular tissues of the trunk.

Lance, spray lance. A length of rigid tubing fitted with a spray nozzle which can be held and directed by a spray operator.

LD_{50} (50% lethal dose). A dose which kills 50 per cent of a population of test animals

Lipophilic. Describes compounds with an affinity for and often soluble in oils.

Mist blower. A machine which emits small droplets of spray solution usually up to 100 microns in diameter, entrained in a strong current of air.

Notching. The practice of making inward sloping cuts into a trunk near ground level, which are treated with herbicide solution.

Oil soluble amine. An amine whose chemical structure contains a long chain lipophilic group, which confers properties of oil solubility.

Oral toxicity. The toxicity of a compound when it is taken by mouth.

Overall spray. A spray treatment applied to both weeds and crop plants, without any attempt at avoiding the latter.

Pesticide. A chemical compound which is used to control an unwanted organism.

Phytotoxicity. Toxicity towards plants.

Pine release. The practice of applying a herbicide treatment to a mixed association of pines and broadleaved woody species, with the object of controlling the latter and promoting the growth of the conifers.

Salt. A type of chemical compound made by reacting an acid with a base.

Selectivity (of a herbicide). The property of controlling some plant species without causing serious injury to others

Substrate The material on which a micro-organism lives.

Surface active agent. A substance which has both hydrophilic and lipophilic properties and which, when added to a liquid, affects the physical properties of the liquid surface. Appropriate surfactants can increase the wetting properties of sprays and assist in the preparation of emulsions and suspensions.

Surfactant. See surface active agent.

TCDD. The usual abbreviation for the dioxin 2,3,7,8-tetrachlorodibenzo-p-dioxin.

Teratogenicity. The property of causing birth defects in unborn foetuses.

Translocation. Movement from one part of a plant to another. Translocated herbicides can affect parts of a plant remote from the point of application.

Tree injection. See injection.

Vascular system. The system of vessels which conducts water, mineral salts and food materials in higher plants.

Volume rate. The amount of liquid applied per unit area. In so far as herbicide applications are concerned this could vary from about 1 litre/ha to well over 1000 litres/ha.

Wetting Agent. A surfactant which, when added to a liquid, increases its wetting properties.

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