Review of the Ecological Effects of Herbicide Usage in Forestry

Environment Canada
Forestry Service

Environnement Canada
Service des Forêts

J.P. Kimmins

REVIEW OF THE ECOLOGICAL EFFECTS OF HERBICIDE USAGE IN FORESTRY

J. P. Kimmins

This review was prepared by Dr. Kimmins, Associate Professor of the Faculty of Forestry, University of British Columbia, Vancouver, B.C., under contract to the Pacific Forest Research Centre, Canadian Forestry Service, Environment Canada. The work is a part of the Centre's environmental research program. Comments of readers are welcomed and should be directed to:

M.H. Drinkwater, Director
Pacific Forest Research Centre
Canadian Forestry Service
506 West Burnside Road
Victoria, B.C.

Information Report No. BC-X- 139

July, 1975

ABSTRACT

The ecological effects of herbicides in forestry practice are reviewed using as a base a recently co-authored bibliography (1973) of 1,614 references on the subject and subsequent papers published up to early 1975. The effects of herbicides are discussed both in terms of individual biotic components of forest ecosystems and in terms of the structure and functioning of complete ecosystems. The mechanisms of herbicide entry, translocation and degradation for plants, animals, soil and water are outlined. A forecast is made on the manner and degree to which herbicides will be used in forestry in the future and alternatives to such use. Finally, a list of research areas is provided which is aimed at filling information gaps and reducing any adverse ecological effects of herbicide application.

RÉSUMÉ

On vient de revoir les effets écologiques de l'emploi des herbicides en foresterie, ayant comme point de départ une récente bibliographie de 1,614 références à ce sujet, écrite en collaboration en 1973, et aussi des articles publiés par la suite jusqu'au début de 1975. Les effets des herbicides sont considérés à deux points de vue: d'abord en rapport avec les constituantes biotiques individuelles des écosystèmes forestiers et aussi en relation avec la structure et le fonctionnement des écosystèmes entiers. Les mécanismes d'entrée et de mobilité des herbicides et de dégradation des plantes, des animaux, du sol et de l'eau, sont exposés leurs grandes lignes. On a prévu comment et jusqu'à quel point les herbicides seront à l'avenir employés en foresterie et les moyens de remplacement. Enfin on y donne une liste des zones de recherche nécessaires pour combler les manques d'information et pour réduire tout effet écologique négatif provenant de l'emploi des herbicides.

Table of Contents

Table of Contents (Continued)

Chapter 1 INTRODUCTION

1.1 Growth of Concern Over the Use of Herbicides

When she embarked on her indictment of all unthinking and irrational use of insecticides, Rachael Carson set in motion forces that have subsequently made it increasingly difficult for resource managers to employ a variety of management chemicals.* Not only did she sensitize a significant segment of both the public and the scientific community to the excessive and ill-advised use of insecticides; she also stimulated a general concern over the undesirable consequences that may attend the use of such chemicals as herbicides, rodenticides and fertilizers.

The initial focus of, and reaction to **Silent Spring** (Carson 1962), concerned broad spectrum insecticides such as D.D.T. Although the principles presented in **Silent Spring** and its sequels (e.g. Rudd 1964) apply to biocides in general, the brunt of the attack was borne by those involved with insect pest problems. The environmental consequences of using such chemicals as herbicides and fertilizers received far less attention, the energetic efforts of such people as Egler (1948, 1950, 1952, 1958, 1964) notwithstanding. The focus on insecticides in the 1950's and early 1960's was so complete that even today the term pesticide is still commonly used as synonymous with insecticide. The situation has changed dramatically in the past five years, however, and the class of management chemicals referred to collectively as herbicides now occupies centre stage.

The transfer of attention from insecticides to herbicides is the result of several factors:

1. A certain amount of success in regulating and restricting the use of insecticides has reduced somewhat the urgent attention that this class of management chemicals has received. This has released some of the energies and attentions of the antagonists of biocides and enabled them to be refocused on the biocide of current popular concern: herbicides.

2. The development of new classes of herbicides, such as picloram, that are far more persistent than such "traditional" herbicides as 2,4-D and 2,4,5-T. This has raised, in some peoples minds, the spectre of long-term residue accumulation and biological concentration, two of the most undesirable characteristics of those chlorinated hydrocarbon insecticides that were the genesis of **Silent Spring.**

3. The use of herbicides as an agent of biological warfare by the U.S. armed forces in the Vietnam war theatre in the 1960's. The repeated blanket application of heavy dosages of herbicides to tropical vegetation (Whiteside 1970) had all the ingredients of D.D.T. spray projects in the U.S. in the late 1950's that generated Rachael Carson's crusade.

4. The increasing use of herbicides in the U.S. during the 1960's for large scale spray projects that were reminiscent of and elicited the same public response as did the D.D.T. spray projects of the 1950's (Shoecraft 1971).

5. The recent realization that the assurances of herbicide manufacturers, users, and proponents that most herbicides are "safe" and have low mammalian toxicity may be unreliable; that they have frequently been based on almost total ignorance of the toxicological, carcinogenic and teratogenic characteristics and potentials of the chemicals. The discovery of highly toxic impurities in 2,4,5-T, evidence of teratogenicity of these impurities, and allegations of increased birth defects in Vietnamese populations in areas affected by U.S. herbicide applications (MacLeod <u>et al.</u> 1971, Norman 1974) have resulted in a public protest of significant proportion. A significant credibility gap has been created concerning the safety of herbicides and the qualifications of those who advocate and use them. Fears that political, military, or economic pressures may be affecting the accuracy of public pronouncements on herbicides has created doubts in the minds of some as to the credibility of much of the herbicide literature.

These and other similar considerations have changed the public and scientific response to the use of herbicides from one of general indifference to one of considerable suspicion and caution. As a result, there is a small but nevertheless real possibility that herbicides could be completely lost as a vegetation management option in very many situations. In some cases this may well be warranted, but it is unlikely that blanket condemnation of herbicide use is any more justifiable than blanket commendation. It is time to take stock of what we do and do not know about the ecological effects of herbicide use, and reviews are needed to assess the ecological aspects of specific uses of herbicides for agriculture, range management and forestry. These reviews are essential if both the proponents and opponents of herbicide use are to understand what is known about such usage, and where and how serious the information gaps are. Only on this basis can rational decisions be reached as to whether or

---

* Chemicals used in the management of biotic resources, including biocides, fertilizers, attractants, repellents and growth regulators.

not a specific herbicide application conflicts with either public or private objectives for a particular region.

The reader might have inferred from the comments so far that there is a general paucity of knowledge on the ecological aspects of using herbicides. On the contrary, there exists an extensive literature on many aspects of this topic, and entry into this literature may be made via one of the many bibliographies (e.g. Anon. 1971a, Anon. 1971b, Grover 1963, Kimmins and Fraker 1973) or using either a general or a specific review (e.g. Anon. 1961, 1963, 1970a, 1970b, McQuilkin 1960, Byrnes 1960, Crafts 1953, 1961, Fletcher 1960, Galston 1968, Geier and Clark 1960, Goodrum and Reid 1956, Grant 1972, Hayes 1960, House et al. 1967, Kearney and Kaufman 1969, Klingman 1961, Kuenen 1960, Martin 1966, Newton 1967, Norman et al. 1950, Norris 1971, Pimentel 1971, Radeleff and Bushland 1960, Robbins et al. 1952, Rudd 1964, Sobieszczanski 1969, Tschirely 1969, Tucker and Crabtree 1970, Weber 1972, Westoff 1960, Woodford et al. 1958). As in many fields of endeavor the problem is not totally one of an absolute lack of knowledge. To a considerable extent it is a failure to use and apply the knowledge that we already have. For example, the presence of 2,3,7,8-tetrachlorodibenzo-p-bioxin (hereinafter referred to simply as dioxin) in 2,4,5-T and some aspects of its toxicology have been known since 1957 (MacLeod et al. 1971), and concern amongst certain scientists and government agencies was voiced in the mid-1960's. Yet, the potential environmental and health hazards of dioxin were not made public until the information was "leaked" in 1969. Egler (1964) was the recipient of the public ire of many members of the professional and scientific community in the mid-1960's for his exposure of the institutional and industrial impediments to the communication and applications of existing knowledge on herbicides and vegetation management. However, events over the past ten years have vindicated many of his basic conclusions. Perhaps his critics were able to recognize their own contribution to the problem and were reacting defensively. There are certainly many aspects of the ecology of herbicide use that do require further research and documentation, but paralleling that need is the urgent need to make those responsible for the use of herbicides fully aware of existing information. This review attempts to contribute to this effort. There is no attempt to make the review exhaustive, and most of the non-English literature is excluded. Rather, it attempts to give the reader a general overview of what is known about the broad ecological effects of herbicide usage in forestry. Detailed specific information must involve the reader in his own reading of the literature. Readers interested in general review articles are referred to the paper by House et al. (1967) entitled "Assessment of Ecological Effects of Extensive or Repeated Use of Herbicides".

The form of the review will be to examine briefly the historical development of herbicide use in agriculture, forestry and other types of resource management, followed by a consideration of the effects of herbicides on biological systems at different levels of biological integration (Rowe 1961). Starting with a consideration of the effects on cells, physiology and genetics, the review will go on to examine effects on specific types of organisms. Attention then turns to a consideration of the effects of herbicides on the overall structure and functioning of ecosystems followed by a discourse on the behavior and fate of herbicides in ecosystems. The review will conclude with a contrast between the ecological effects of herbicides and some alternative methods of vegetation manipulation and an assessment of major information gaps.

1.2 Historical Development of Herbicides and Their Use on Forest Lands

The earliest reported herbicide usage dates from 1878 (Freed 1961) when carbon bisulphide was used as a soil fumigant to kill plants. However, herbicide usage did not really develop until after 1896 when the phytotoxicity of copper sulphate was discovered. This stimulated a search for other inorganic salts and acids suitable for selective weed control. Iron and other sulphates (including sulphuric acid) were tested on annual weeds, while arsenic and its compounds were tested on perennial weeds. By 1920, the phytotoxicity of chlorate and borate salts to perennial plants had been added to the list of inorganic herbicides.

The following two decades saw increased activity screening a wide range of inorganic and readily available organic chemicals, but few major discoveries resulted. However, this period of testing did reveal the phytotoxic potentials of oils, phenols and various petroleum products, and the discovery of the selectivity of several herbicides represented a major advance.

The 1940's was a turning point in the history of herbicides, marked by the development of the dinitro and chlorinated phenols. As early as 1880, Darwin had suggested that plant processes might be controlled by chemical substances. Verification of his theory led to extensive physiological work on chemical regulators of plant growth in the first three decades of this century, which culminated in the isolation, identification and characterization in the 1930's of three growth hormones (auxins, including idole-3-acetic acid). This stimulated research into the biological activity of various other acetic acids, and by 1938 work had begun on the phenoxy acetic acids, including 2,4-D. Early interest in 2,4-D was not for its phytotoxic properties, however; it was initially developed for its potential as a hormone to promote root growth in cuttings. In spite of warnings in the literature concerning the inherent phytotoxicity of 2,4-D, its potential as a herbicide was not given much attention until the war when recognition of its potential as a military weapon delayed its introduction as a general plant management chemical. In fact, public dissemination of information on the biological activity of the phenoxy acetic acids was not made until 1944 when the value of 2,4,5-T in controlling woody plants was announced. Interest reverted to 2,4-D, however, probably because of economic considerations, and within two years it had been widely tested and applied. However, discovery of the relative ineffectiveness of 2,4-D against woody plants led to a growing interest in 2,4,5-T, and before long it was also widely accepted.

2,4-D and 2,4,5-T were not the only phenoxy-acetic acids developed during the late pre-World War II years. MCP was developed at about the same time and within a few years a large number of new phytotoxic chemicals were produced, including CIPC, amitrole, the triazines, and many others. Recent developments have involved the addition of many new and important organic herbicides, including the picolinic acid herbicides (picloram), characterized by their effectiveness against woody vegetation and their persistence. Besides new discoveries, there have also been major advances in methods of using existing herbicides more effectively. The addition of surfactants, stickers, activators and the development of more effective application techniques has greatly improved herbicide efficiency, and recent attention has been drawn to combinations of herbicides, mixtures of herbicides and fertilizers, and mixtures of herbicides with agents to deactivate them after application in order to minimize residue problems (Foy and Bingham 1969). Increasing knowledge of the mode of action and behavior of herbicides in the environment has also led to increasingly efficient herbicide use.

Herbicide usage in forestry post-dates agricultural and horticultural usage by many decades. Most usage dates from the development for military purposes of a variety of organic herbicides and growth regulators during the second World War (Whiteside 1970). The development of herbicides for the control of woody vegetation opened up possibilities for mechanized manipulation of vegetation that were previously not available.

An examination of the literature on herbicides reveals that most of the earlier papers come from the 1940's, with a marked increase in the publication of research and empirical experience in the 1950's. A great surge in herbicide literature occurred in the 1960's, with research interests being broadened to include many topics that were ignored in the earlier years. The current decade has seen a continuation of this research interest, but with increasing interest on health aspects (toxicology, carcinogenicity and teratogenicity) and on broad ecological effects. The military use of herbicides in tropical ecosystems has provided a focus for this interest (Boffey 1971, Galston 1968, House et al. 1967, Norman 1974, Orians and Pfeiffer 1970, Tschirley 1969, Whiteside 1970).

References Cited in Chapter 1

Anon. 1961. Herbicides and their use in forestry. Proc. Symp. Oregon State Univ., Corvallis, 127 p.

Anon. 1963. Pesticide Wildlife Studies. Fish and Wildlife Service. U.S. Dept. of Interior, Circ. 199.

Anon. 1970a. International herbicide symposium, Eberswalde, June 1969. Arch. Forstw. 19: 206-345.

Anon. 1970b. Weed Control. Z. Pflkrankh. Spec. No. 5, 247 p.

Anon. 1971a. Bibliography on persistence and decomposition of herbicides in soils and plants (1968-1967). Serial No. 1478, Commonwealth Bureau of Soils, Harpenden, England, 19 p.

Anon. 1971b. Bibliography on persistence and decomposition of herbicides in soils and plants (1971-1969). Serial No. 1477, Commonwealth Bureau of Soils, Harpenden, England, 27 p.

Boffey, P.M. 1971. Herbicides in Vietnam: AAAS study finds widespread devastation. Science 171: 43-47.

Byrnes, W.R. 1960. Woody brush control. p. 90-96. In Herbicides and Their Use in Forestry. R.E. McDermott and W.R. Byrnes, eds., Pa. State University For. Symp.

Carson, R. 1962. Silent Spring. Houghton-Mifflin Co., Boston, 368 p.

Crafts, A.S. 1953. Herbicides. Annu. Rev. Plant Physiol. 4: 253-282.

Crafts, A.S. 1961. The Chemistry and Mode of Action of Herbicides. Interscience Publishers, New York, 269 p.

Egler, F.E. 1948. 2,4-D Effects in Connecticut Vegetation. Ecology 29: 386-387.

Egler, F.E. 1950. Herbicide effects in Connecticut vegetation, 1949. Bot. Gaz. 112: 76-85.

Egler, F.E. 1952. Blanket vs. selective spraying for brush control on right-of-ways. South. Weed Conf. Proc. 5: 141-142.

Egler, F.E. 1958. Science, industry, and the abuse of rights-of-way. Science 127: 573-580.

Egler, F.E. 1964. Pesticides in our Ecosystem: Communication II. Bioscience 14: 29-36.

Fletcher, W.M. 1960. The effect of herbicides on soil microorganisms. p. 20-62. In Herbicides and Their Use in Forestry. Proc. Symp., Sch. For., Oregon State University, Corvallis, Oregon.

Foy, C.L. and S.W. Bingham. 1969. Some research approaches toward minimizing herbicidal residues in the environment. Residue Rev. 29: 105-135.

Freed, V.H. 1961. The development and characteristics of herbicides. p. 5-14. In Herbicides and Their Use in Forestry. Proc. Symp., Sch. For., Oregon State University, Corvallis, Oregon.

Galston, A.W. 1968. Defoliants. p. 62-75. In Chemical and Biological Warfare. S. Rose, ed., G.G. Harrap & Co. Ltd., London.

Geier, P.W. and L.R. Clark. 1960. An ecological approach to pest control. p. 10-18. In Symp.: The ecological effects of biological and chemical control of undesirable plants and animals. 8th Tech. Meeting, I.U.C.N.

Goodrum, P.D. and V.H. Reid. 1956. Wildlife implications of hardwood and brush control. Trans. 21st North Amer. Wildl. Conf., Washington, D.C.: 127-141.

Grant, W.F. 1972. Book review of: Chemical mutagens: Environmental effects on biological systems and chemical mutagens. Principles and methods for their detection. Can. Field-Naturalist 86: 106-107.

Grover, R. 1963. Bibliography of weed control research in tree and woody ornamental nurseries and forest and shelterbelt plantation. Publ. Forest Nursery Stn., Indian Head, Sask. No. 26, 36 p.

Hayes, W.J., Jr. 1960. Agricultural chemicals in relation to human health. p. 14-15. In The Nature and Fate of Chemicals Applied to Soils, Plants, and Animals. U.S.D.A., ARS 20-9.

House, W.B., L.H. Goodson, H.M. Godberry and K.W. Dockter. 1967. Assessment of ecological effects of extensive or repeated use of herbicides. Adv. Research Projects Agency ARPA Order No. 1086, U.S. Dept. Defense, 369 p.

Kearney, P.C. and D.D. Kaufman, eds. 1969. Degradation of Herbicides. Marcel Dekker Inc., N.Y., 394 p.

Kimmins, J.P. and P.N. Fraker. 1973. Bibliography of Herbicides in Forest Ecosystems. Envir. Canada. For. Serv. Info. Rept. No. BC-X-81, 1614 refs., 261 p.

Klingman, G.C. 1961. Weed Control as a Science. John Wiley & Sons, Inc., New York, 421 p.

Kuenen, D.J. 1960. General Introduction. p. 1-9. In Symp: The ecological effects of biological and chemical control of undesirable plants and animals. 8th Tech. Meeting, I.U.C.N.

MacLeod, C.M. et al. 1971. Report on 2,4,5-T. A report of the Panel on Herbicides of the President's Science Advisory Committee. Exec. Off. of the Pres., Off. Sci. & Tech. March 1971, 68 p.

Martin, J.P. 1966. Influence of pesticides on soil microbes and soil properties. In Pesticides and Their Effects on Soils and Water. Wisconsin Soil Sci. Soc. Amer., ASA Special Publ. 8:95-108.

McQuilkin, W.E. 1960. Silvicultural applications of herbicides: General problems and a short historical review. p. 74-83. In Herbicides and Their Use in Forestry. R.E. McDermott and W.R. Byrnes, eds., Pa. State University For. Symp.

Newton, M. 1967. Vegetation management -- a system of operation. p. 8-11. In Proc., Symp. Herbicides and Vegetation Management, Oregon State University, Corvallis, Oregon.

Norman, A.G., C.E. Minarik and R.L. Weintraub. 1950. Herbicides. Annu. Rev. Plant Physiol. 1: 141-168.

Norman, C. 1974. Academy reports on Vietnam herbicide damage. Nature 248: 186-188.

Norris, L.A. 1971. The behavior of chemicals in the forest. p. 90-106. In Pesticides, Pest Control and Safety on Forest Range Lands. Oregon State University, Sch. Agric., Corvallis, Oregon.

Orians, G.H. and E.W. Pfeiffer. 1970. Ecological effects of the war in Vietnam. Science 168: 544-554.

Pimentel, D. 1971. Ecological effects of pesticides on non-target species. Executive Office of the Pres., Off. Sci. & Tech. 4106-0029.

Radeleff, R.C. and R.C. Bushland. 1960. The toxicity of pesticides for livestock. p. 134-160. In The Nature and Fate of Chemicals Applied to Soils, Plants and Animals. U.S.D.A., ARS 20-9.

Robbins, W.W., A.S. Crafts and R.N. Raynor. 1952. Weed Control: a textbook and manual, 2nd Ed., McGraw-Hill Book Co., Inc., Toronto, 503 p.

Rowe, S.J. 1961. Level-of-integration concept and ecology. Ecology 42: 420-427.

Rudd, R.L. 1964. Pesticides and the Living Landscape. Univ. Wisc. Press, Madison, 320 p.

Shoecraft, B. 1971. "Sue the Bastards". Franklin Press, Phoenix, 460 p.

Sobieszczanski, J. 1969. Herbicides as factor changing the biological equilibrium of soil. 1st Nat. Congr. Soil Sci. Soc., Sofia.

Tschirley, F.H. 1969. Defoliation in Vietnam: the ecological consequences of the defoliation program in Vietnam are assessed. Science 163: 779-786.

Tucker, R.K. and D.G. Crabtree. 1970. Handbook of toxicity of pesticides to wildlife. U.S. Fish Wildl. Serv., Bur. Sport Fish Wildl. Resource Publ., No. 84, 131 p.

Weber, J.B. 1972. Interaction of organic pesticides with particulate matter in aquatic and soil systems. p. 55-120. In Fate of Organic Pesticides in the Aquatic Environment. R.F. Gould, ed. Adv. Chem. Ser. III, Amer. Chem. Soc., Washington, D.C.

Westoff, V. and P. Zonderwijk. 1960. The effects of herbicides on the wild flora and vegetation in the Netherlands. p. 67-78. In Symp.: The ecological effects of biological and chemical control of undesirable plants and animals, 8th Tech. Meeting, I.U.C.N.

Whiteside, T. 1970. Defoliation. Ballantine/Friends of the Earth: New York, 168 p.

Woodford, E.K., K. Holly and C.C. McCready. 1958. Herbicides. Annu. Rev. Plant Physiol. 9: 311-358.

Chapter 2 EFFECTS OF HERBICIDES ON INDIVIDUAL BIOTIC COMPONENTS OF THE FOREST ECOSYSTEM

In this section of the paper I will examine some of the known effects of herbicides on individual biotic components of the forest ecosystem that we are managing. Following the concept of "levels of biological integration" (Rowe 1961), the chapter will start with the lower levels of biological integration by examining sub-cellular and cellular systems. It will then move to more complex and integrated biological systems by considering the effects of herbicides on entire organisms of various types. Levels of biological integration above the individual organism will be discussed in the next section. Nearly all the works referred to deal with plants. Very few papers dealing with comparable animal studies were encountered, although there is growing evidence of some similarity of response at the genetic and physiological level.

2.1 Effects of Cellular and Sub-Cellular Systems: Genetic, Physiological, Cytological, and Cellular Effects

2.11 Genetic effects:

Recognition of the mutagenic potential of pesticides dates from 1931 when it was discovered that the reduced seed set of tobacco following fumigation with nicotine sulphate resulted from the induction of chromosome abnormalities (Grant 1972). This aspect of herbicide usage received relatively little attention until the last few decades, and it is only very recently that major concern has developed over the teratogenic, carcinogenic and mutagenic characteristics of herbicides.

Herbicides have classically been thought of as being "safe" chemicals. This attitude can be traced partly to the assurances of the herbicide manufacturing and using industries, and partly to a general failure until recent times to test any toxicological aspects of herbicides other than acute toxicity (Grant 1971a). There has been a conspicuous failure to test the more subtle and insidious toxicological characteristics of herbicides (Grant 1971b). Work over the past two decades, exemplified by the writing of Grant (1971a, 1972) and Wuu and Grant (1966, 1967a, 1967b, 1967c), has made it abundantly clear that a far more rigorous analysis of the health and environmental toxicology of herbicides is required before we can accept bland assurances of their "safety". The discovery of the extreme acute toxicity and teratogenicity of dioxin, an impurity of 2,4,5-T, stands as a warning that these chemicals are not "safe" and that they require great care and strict control on their usage.

It has been well-known for some time that certain types of radiation can be mutagenic, but it is rather less generally recognized that a wide variety of chemical mutagens can induce rather similar chromosome aberrations (Doxey and Rhodes 1949, Fishbein et al., 1970, Vogel and Rohrborn 1970, Hollaender et al. 1971, Grant 1972). Wuu and Grant (1966) showed that all pollen mother cells in barley plants grown from seed soaked in the herbicide 'Lorox' exhibited nuclear abnormalities including chromosome stickiness, chromosome clumping, cytoplasmic furrowing, unequal distribution of chromatin material between daughter cells, and asynchronous, multiple cell divisions. Grant and Harney (1960) showed that cytological abnormalities persist in tomato plants to at least the third generation following the treatment of seeds with maleic hydrazide. Unrau (1953, 1954) and Unrau and Larter (1951) have reported irregular meiotic behavior in barley and wheat plants sprayed with 2,4-D at any of several different stages of growth and that abnormal cell division persists for a considerable length of time after spraying.

Wuu and Grant (1967b) noted that the propensity of species to the induction of chromosomal aberrations by mutagenic pesticides appears to be related to nuclear size and the DNA content per diploid nucleus or per chromosome. Sensitivity of plants to radiation damage and induction of chromosome aberrations by radiation is also related to these characteristics of the nucleus (Sparrow et al. 1965), and these authors (Wuu and Grant 1967a, 1967c) went on to confirm Doxey and Rhodes' (1949) observation that chromosomal aberrations induced by chemical mutagens (in this case, herbicides) are essentially similar to those induced by X-rays. However, in a study of the cytological effects of 2,4-D and amitrole, Mohandas and Grant (1972) failed to demonstrate a correlation between nuclear volumes and the number of cytological abnormalities. These authors went on (Mohandas and Grant 1973) to state that there appears to be a general relationship between herbicide susceptibility and nuclear volumes, with sensitive species having lower nuclear volumes than resistant ones, although the relationship is confounded by perennial vs annual growth habits of the test plants. If the relationship exists, it is the inverse of the relationship between radiation-induced mutation and nuclear volume.

Dävring and Surner (1971) noted that some stages in the development of an organism are more sensitive to teratological factors (those that induce malformation and abnormal growth in fetuses) than others. During premeiosis and meiosis, teratogens may cause mutations, chromosome aberrations or the total collapse of cell division mechanisms. Fertilization can be affected and effects on mitosis can perturb the normal development of organs.

These effects have been demonstrated in a variety of higher plants, but relatively little work has been conducted on animals until recently. Concern over the teratogenic and carcinogenic potential of pesticides led to a considerable amount of work over the past decade by the U.S. National Cancer Institute (Courtney et al. 1970). One finding of this survey was that birth defects could be provoked in rats and mice by the administration of 2,4,5-T (MacLeod et al. 1971). Subsequent work has shown that the 2,4,5-T used was contaminated with dioxin and that both pure 2,4,5-T and dioxin are teratogenic and also cause increased fetal mortality in some test organisms. The initial conclusions concerning the teratogenicity in rats was probably the result of the dioxin (Courtney and Moore 1971). 2,4,5-T has been shown to produce teratogenic effects in mice, but not in rats. It thus appears that the mutagenic and chromosomal aberration effects induced by herbicides in plants may also occur in animals. This idea is supported by the findings of Dåvring and Surner (1971) who found that low levels of 2,4,5-T fed to fruit flies in aqueous solution caused drastic reductions in fertility, very high egg mortality, and total mortality of the few first instar larvae that were produced. While there were no apparent external morphological changes, ovaries were diminished and rendered abnormally fragile, oogenesis was interferred with, and chromosome aberrations were induced.

The question of the genetic effects and mutagenicity of herbicides as they are applied in forest ecosystems is not an easy one to answer. Much of the work on chemical mutagenicity has involved studies of the effects of candidate mutagens on isolated DNA. However, some mutagens that are very effective on isolated DNA may exert little or no mutagenic influence on intact cells in living organisms, either because the mutagen cannot get inside the cell, because once inside the cell it is deactivated by enzymes, or because the cell is able to repair the damage induced by the mutagen. Alternatively, compounds that are relatively ineffective at inducing mutations in isolated DNA may significantly alter hereditary material in field populations if they are activated inside a cell or if their effect on DNA cannot be repaired. For example, humans can suffer from a hereditary disease in which cells are unable to repair UV-induced genetic alterations. Such individuals are sensitive to UV which tends to induce skin cancer (Freese 1971).

A further problem results from the fact that the mutagenicity of a compound to one type of organism may not be observed in a different type of organism. Enzymatic inhibition of mutagenicity will vary from one species to another because of the variable enzyme complement of their cells. Similarity of the response of different species to a particular chemical mutagen will be greatest in closely related species because of the similarity of their biochemistry, reflecting their close evolutionary relationship.

New and synthetic substances are more likely to be mutagenic than old or natural ones since organisms are less likely to be biochemically prepared to deal with the former than the latter.

Concern over mutagenic chemicals in our food, medicine and environment has been growing over the past two decades. It received a great boost from the tragic case of Thalidomide, and subsequently from claims of increases in birth defects in areas of South Vietnam subject to heavy and repeated applications of herbicides by U.S. military forces. The increased rate of mutation imposed on the human population by mutagenic agents such as radiation, pesticides, and certain other synthetic chemicals poses a great threat to the future of mankind. In a natural population the process of natural selection constantly removes non-adaptive mutants from the population. In our sheltered, well-fed and doctored society, this process has been eliminated, increasing the so-called "genetic load": the percentage of the population carrying deleterious but sublethal genetic mutations. This load will ultimately cost society far more in terms of both human suffering and health-care dollars than the short-term benefits that the mutagenic agents provide. We may indeed be "living on borrowed time".

In concluding this section it must, unfortunately, be concluded that we do not know and are currently not in a position to accurately assess the genetic consequences of applying herbicides for either human, other animal or plant populations. The danger is perhaps greatest for the human population since we have removed the protective mechanism of natural selection, and while the relationship between increasing rate of mutation and increasing susceptibility to disease is not precisely understood, it is a relationship that cannot be ignored (Newcombe 1971). A conservative approach to the presently unanswerable question of the genetic consequences of herbicide application would appear to be prudent.

2.12 Physiological (biochemical) effects:

There is a great variety of physiological reactions induced in living cells by herbicides. These vary according to the type and formulation of the herbicide, the species and age of the plant, the method and timing of the application, and the condition of the environment. In this review of physiological effects, I have generally omitted detailed referencing of information sources since these are so adequately summarized in Ashton and Crafts (1973, Chapters 3 and 6) to which reference should be made for original papers. The influence of hormones on nucleic acid metabolism was reviewed by Key (1969).

The earliest of the herbicides, such as salts and acids acted by destroying protoplasmic structure by virtue of high acidity and osmotic potential, and their ability to precipitate proteins. However, the exact nature and sequence of the biochemical reactions involved in their herbicidal activity are poorly understood. Oils operate by destroying the semipermeable nature of living membranes by solubilizing them, and by interpolation of oil molecules into the protein layer of the membrane, resulting in loss of its function and integrity.

The physiological effects of the more recent organic herbicides were summarized by Ashton and Crafts (1973) under three headings: 1. respiration and mitochondrial electron transport, 2. photosynthesis and the Hill reaction, and 3. nucleic acid metabolism and protein synthesis.

2.121 Respiration and mitochrondrial electron transport

Mitochondria are thought to be the cellular components responsible for respiration. Many herbicides have been shown to interfere with the functioning of mitochondria by blocking the synthesis of ATP (adenosine triphosphate, a high energy molecule involved in photosynthesis, respiration and other vital metabolic processes), and by interfering with electron and energy exchange processes within the mitochondria. In some cases oxygen uptake is reduced with a subsequent suppression of metabolism, while in others it is increased reflecting increased respiration and metabolism. Many herbicides are known to induce these reactions, but only in certain cases does this represent the major physiological effect of a herbicide.

2.122 Photosynthesis and the "Hill reaction"

Many herbicides are known to be more effective in the light than in the dark, and with high rather than with low rates of transpiration. This has been shown to be related to inhibition by herbicides of the "Hill reaction" (reduction of ferricyanide by chloroplasts) and of photosynthesis: inhibitions that can take place at extremely low herbicide concentrations. This can cause a phytotoxic depletion of carbohydrates in the treated plant, and the herbicidal symptoms were experimentally alleviated in one plant species by supplying the treated plant with glucose.

For some herbicides, interference with photosynthesis, as detected by inhibition of the Hill reaction, may represent a major component of their phytotoxicity. For many others, interference with photosynthesis may be a very secondary factor in plant death. However, herbicides do affect a variety of functions of chloroplasts that are vital for normal cellular activity, such as energy transfer and ATP synthesis, and therefore herbicidal effects on chloroplasts are of considerable significance.

2.123 Nucleic acid metabolism and protein synthesis

The effects of herbicides on nucleic acid metabolism and protein synthesis are very complex, but, stated simply, there can be either a stimulation or a depression of these two metabolic processes.

Several herbicides increase nucleic acid metabolism and protein synthesis. There is a net transfer of RNA from the nucleus to the ribosomes and an increase in messenger RNA, suggesting renewed nuclear activity and a reversion from normal to meristematic metabolism. The increases in these two metabolic processes tend to be marked in herbicide-sensitive species while herbicide-tolerant species exhibit little increase or even a decrease. There may be a marked increase at high concentrations of herbicides such as 2,4-D while low concentrations result in a depression of these processes. Other herbicides have the effect of interfering with RNA synthesis, and blocking ATP and amino acid incorporation. Direct lethality resulting from interference with nucleic acid metabolism and protein synthesis has never been proven, however. Both chloroplasts and mitochondria contain DNA which is involved in the synthesis of RNA and proteins. Many herbicides which inhibit O_2 uptake or evolution by mitochondria and chloroplasts, respectively, also interfere with nucleic acid metabolism and protein synthesis and it is still not clear exactly what is the proportional contribution of each of these various effects of herbicides to the death of the plant.

The metabolism of cells and their sub-cellular components involves uptake and transfer of solutes: processes that involve ATP energy. Protein synthesis alone accounts for 90% of the expenditure of ATP energy. Thus, herbicides that interfere in ATP production will greatly effect general cellular metabolism and protein synthesis. It has been found that all the herbicides that are strong inhibitors of RNA and protein synthesis all reduce ATP levels, and a reduction of protein synthesis may be a major aspect of the phytotoxic action of the many herbicides that depress these metabolic functions.

Various other aspects of the biochemical make-up of cells and their sub-cellular components are also subject to change under the influence of herbicides. For example, Smith and Wilkinson (1973) found that simazine and atrazine treatments increased the free fatty acid content of isolated chloroplasts of spinach. Sharma and vanden Born (1972) reported that picloram markedly reduced the chlorophyll content of soybean and Canada thistle plants, while RNA and protein contents increased 30%. On the other hand, levels of these biochemicals in barley were virtually unaffected. Volynets and Kornelyuk (1973) found that 2,4-D and TCA induced accumulations of phenols (phenolic aglycones) in flowers of yellow lupin. They reported that these accumulations had a high physiological activity based on the effects of these accumulated phenols on growth of lupin seedlings and on root formation in French bean cuttings. Panić and Franke (1971) found that 2,4-D produced a variable reduction in ascorbic acid in wheat and bean plants, while the levels of ascorbic acid oxidase rose in wheat and decreased in beans.

2.13 Cytological and cellular effects:

As in the above section, much of the following information is taken from Ashton and Crafts (1973, Chp. 3) and is not referenced in detail. The topic was also reviewed by Anderson and Thomson (1973).

Interest in the cytological effects of herbicides followed the introduction of the growth regulating herbicides. One of the earliest observations was that 2,4-D stimulated cell proliferation in bean plants starting in the endodermis of the stem and spreading to the phloem and cortex parenchyma until the phloem was completely crushed. Other herbicides cause phloem necrosis which results in the accumulation of products of photosynthesis in treated leaves rather than in underground organs.

Disturbances in cell shape, cell size, cell divisions and tissue morphology are common, as are the changes in meiosis and mitosis already discussed. Phloem, parenchyma, sieve tubes and their companion cells are destroyed or blocked by callus. Affected cells enlarge but may fail to differentiate. Cell plate and cell wall formation may be inhibited and the giant cells that result may be multinucleate. Stomatal chamber volume and pallisade air space can be reduced, cambial activity arrested, and the thickness of sieve tubes and tracheal elements decreased.

Chloroplast structure is frequently affected by herbicides. Precocious vacuolation, change in shape to a spherical form, destruction or swelling of frets, disorganization of the grana, rupturing of chloroplast envelopes, loss of starch, and change in membrane permeability have been noted in studies of the effect of various herbicides on various plant species (Ashton and Crafts 1973).

Bradley and Crane (1955) reported that 2,4,5-T applications to apricot trees causes a marked increase in fruit size. This was found to result from a 57% increase in cell volume rather than an increase in the number of cells. Even greater increases in nuclear size were observed, and there was a great increase in the frequency of high polyploid nucleae (up to 64-ploid). 2,4-D was found to cause a severe shrinkage of protoplasts in Pinus radiata needle cells within 24 hours of treatment, coupled with a loss of integrity of the plasmalemma within 4-8 hours. Both 2,4-D and picloram disrupted chloroplast structure in leaf discs and the integrity of cell membranes in stem tips of Eucalyptus viminalis (Bachelard and Ayling 1971).

Dodge and Lawes (1974) described the effects of two herbicides on the fine structure of leaf cells of several plants. Effects varied greatly between plants reflecting different sensitivities to the herbicides. In the most extreme cases chloroplasts started to swell within 3-4 hours of treatment, and continued to swell until they were spherical. Eventually the cell tonoplast and plasmalemma broke down, followed by the rupture of the chloroplast envelope. Other cell organelles disintegrated and finally all that remained of the cells were the membranous components of the chloroplasts and granular material from the nucleus.

Wu and Kozlowski (1972) reported the effect of 2,4,5-T on Pinus resinosa seeds and young seedlings. Cell division and root expansion was inhibited, followed by proliferation and expansion of parenchyma cells in the stem and cotyledons. Parenchyma cells in the upper stem became disorganized and many collapsed leading to callus formation. Development of primary leaf primordia and the expansion of primary needles were inhibited, and cotyledons were fused to the primary needles.

White and Hemphill (1972) observed that while expanding leaves from the upper portion of tobacco plants were resistant to 2,4-D, mature leaves from lower on the plant were highly sensitive. The mesophyll cells of these lower leaves underwent rapid rupturing and disintegration of the tonoplast, plasmalemma, and membranes of chloroplasts and mitochondria.

2.2 Effects on Plant Morphology

The general effects of herbicides on plant morphology were reviewed by Gorter and van der Zweep (1964) and Ashton and Crafts (1973) and the following discussion is based on their papers.

All morphological abnormalities are ultimately the result of alterations in the biochemistry and physiology of cells. These processes control morphogenesis which in turn produces a given morphology. The herbicide-induced alterations in biochemistry and physiology discussed above provide the basis for explaining the herbicide-induced morphological abnormalities described below.

Most morphological aberrations are the result of the auxin or growth regulator herbicides (e.g. 2,4-D, 2,4,5-T, picloram). Herbicides that act as direct phytotoxins produce fewer morphological changes since the plant or organ dies before such aberrations can be expressed.

The reaction of different organs to herbicides varies greatly, and the effects of a herbicide on different parts of the same plant may be independent. For example, tops may be killed while roots remain unharmed. As we have already seen, the type of morphological effect will depend very much upon the herbicide concentration. It also depends upon whether the herbicide reaches a particular organ by redistribution within the plant or by direct application.

Plant morphology is controlled by meristematic activity under the control of growth hormones that either stimulate or inhibit cell division and expansion, depending upon concentration. Each plant and each organ of each plant has a characteristic sensitivity to growth hormones (or their synthetic analogues, auxins). Roots are the most sensitive, being inhibited at concentrations much lower than stems. Buds have an intermediate sensitivity.

The great sensitivity of roots has led to use of the inhibition of elongation in primary roots as a sensitive bioassay of herbicides. Characteristic abnormalities include root thickening and the massive induction of lateral root formation and growth, root branching, and an alteration in root hairs. Elongation of root cells is inhibited while lateral growth is stimulated leading to swelling and thickening of roots. Massive development of lateral roots may occur which rupture and destroy root tissues and open the way for invasion by pathogens. In other species, lateral root growth may be inhibited. Death of the main root system may be followed by the stimulation of adventitious root formation; this is common in monocotyledons where the formation of crown roots is stimulated. Root hairs are stunted by the inhibition of cell elongation, and their walls become thickened impairing their function. Root nodulation of legumes and other plants may be inhibited by the application of herbicides (Dunigan et al. 1972).

Stems are less sensitive than roots, and inhibition of growth requires much higher concentrations of herbicides. Below such concentrations, stem elongation is stimulated, while stem thickening occurs at higher concentrations. Morphological abnormalities are due to herbicidal effects on cell extension, cell division, and on the organization of the growing point. Cell extension may be accelerated or retarded, and unequal concentrations of herbicide in different parts of the stem gives rise to twisting. Cell division may be stimulated resulting in galls or tumors which can occur in either meristematic or mature tissues and which may rupture the stem permitting entry of pathogens. Inhibition of wound periderm formation compounds the damage. General stimulation of cambial activity can occur leading to thickening of the stem and a crushing of internal tissues. Disturbance of the growing tip resulting from a change in shape of the growing tip from a dome to a ridge can lead to fasciation and the formation of a band-shaped distortion. This results in a flat or fluted stem.

The growth of leaves from an early age is the result of cell expansion rather than cell division. Auxin herbicides do not cause cell enlargement in leaves as they do in stems, but the relative development of mesophyll and veins may be altered resulting in either strap-like or crinkled leaves and abnormal vein patterns. Leaf size may be reduced and altered and twisting of petioles is common. Leaf morphology can be changed with compound leaves becoming fused into simple leaves. Production of leaves can even be completely suppressed resulting in the formation of leafless stems. Effects on leaves can be sustained for several years after treatment of perennials because of the alteration of growing tips in dormant buds. Herbicides may induce leaf abscission in some plants.

Flower and inflorescence morphology can be affected by inhibition or stimulation of cell division or enlargement. There can be a reduction or alteration in the number, size and shape of flower parts, and there may be a differential effect on organs of different sex. There can be a multiplication or reduction in flower number, and earlier flowering can be stimulated. The resulting fruit may be larger due to increase in cell volume, but the shape of the fruit may be aberrant if the concentration of herbicide within the fruit is uneven.

Much of the work on effects of herbicides on plant morphology relates to agricultural and horticultural plants. References to effects on forest trees are much less common, but the types of effects described above can be expected. For example, Peterson et al. (1974) examined the effect of picloram on the shoot anatomy of red maple and white ash and found enlargement of cortical and pith cells and xylem blockage followed by death of the shoot tip, parenchyma and phloem in red maple. White ash showed little anatomical or morphological change and was still healthy after 22 days while red maple shoots were desiccated and dead. Sterrett et al. (1974) investigated the effects of two herbicides on foliar abscission in certain deciduous and coniferous woody plants. Abscission occurred within 14 days in the deciduous species while it was absent in the coniferous species.

2.3 Effects on Wildlife

There appears to be a rather general feeling that the direct toxic effects of herbicides on wildlife are of less significance than the indirect effects of habitat and food plant modification (e.g. Lawrence 1967, Brown 1967, Keith et al. 1959, Azevedo 1973). There is almost universal agreement that compared with pesticides like DDT or the organophosphates, most commonly used herbicides have only a moderate to low acute toxicity to wildlife (Rudd and Genelly 1956, Warren 1967, Brown 1967, Drill and Hiratzka 1953, Rowe and Hymas 1954). The Mrak Commission Report on Pesticides and Their Relationship to Environmental Health (Mrak et al. 1969) concluded that ..."herbicides as presently used do not present serious and widespread hazards to non-target organisms. With few exceptions, most herbicides have a low order of toxicity to aquatic and terrestrial animals". These comments do not apply to arsenical herbicides which are normally considered to be highly toxic to wildlife (e.g. Exon et al. 1974, Dickinson 1972) and which are used as insecticides in the case of bark beetles (e.g. Chansler and Pierce 1966, Buffam 1971, Buffam and Flake 1971, Frye and Wygant 1971, Newton and Holt 1971, Stelzer 1970).

Many of the statements in the literature concerning the toxicity of herbicides date from a time when our knowledge of acute and chronic toxicity, carcinogenicity and teratogenicity was substantially less than the still rather incomplete knowledge that we now have. While this does not necessarily render these opinions invalid, it does emphasize the Mrak Commission's conclusion that we do not have adequate information at this time to either condemn or condone with confidence many of the herbicides in common use. Until evidence to the contrary is produced, it appears necessary to accept that herbicides lack the serious toxicological implications that are characteristic of many other biocides, while at the same time realizing that they are toxic, that they can produce symptoms of acute and/or chronic toxicity, and that they are known to have toxicological characteristics other than acute and chronic toxicity (see discussion of physiological and genetic effects above).

Herbicides have been widely used as a tool of habitat and food plant manipulation by wildlife biologists. Conversion of scrub forest to shrub communities (Shipman 1971, Krefting and Hansen 1969, Gysel 1957), conversion of dryland shrub communities to grassland (Wilbert 1963, Quimby 1966) and modifying aquatic vegetation (Coulter 1958, Leonard and Cain 1961, Schroeder 1972) have been widely practised as a means of improving ungulate or game bird habitat. Conversely, herbicidal control of hardwood brush for the establishment of conifer plantations may remove valuable wildlife browse species and habitat. Chemical brush control on power right-of-ways have been shown to favour wildlife (Bramble and Byrnes 1972).

Exposure of wildlife to herbicides involves either direct impact of the spray on the organism, contact with a contaminated surface, inhalation of contaminated air, and/or ingestion of contaminated food, water or other material. There is some evidence that herbicide treatment may reduce the palatability of vegetation (Grigsby and Farwell 1950, Leopold et al. 1971, p. 65), and this could reduce the exposure of wildlife to the herbicide. However, the evidence on this point is rather weak and there is counter evidence that wildlife will remain in and browse on plants in sprayed areas (Newton and Norris 1968). Ward (1973) noted that sage-brush control with 2,4-D had little observable effect on calving behavior and feeding habits of elk in Wyoming.

There have been numerous studies in which livestock and domestic animals have been fed herbicide-contaminated food and subsequently examined for toxic effects, but few comparable studies have been reported for wildlife species. Sheep and cattle appear to tolerate moderate quantities of 2,4-D and 2,4,5-T in their feed, with rapid and fairly complete elimination of the herbicide in the urine and faeces and/or detoxification in the intestine. Studies on cows have revealed little adsorption from the intestine, very little accumulation in organs or tissues, and low, transitory levels in milk (Fisher et al. 1965, Ware and Brakel 1963, Norris 1971, Bohmont 1967, Palmer and Radeleff 1964, Buck et al. 1961, Kenaga 1969, Klingman et al. 1966). Radiolabelled 2,4-D fed to sheep rapidly appeared in the blood with peak concentrations within two hours followed by a rapid decline to almost zero in 24 hours. 96% of the dose was excreted unchanged in the urine within 72 hours (Clarke et al. 1964). 2,4-D and 2,4,5-T have a higher toxicity then picloram-based herbicides and the low acute toxicity of 2,4-D and 2,4,5-T for sheep and cows is not reflected in the somewhat higher values for dogs. The low acute toxicity data for livestock have led to the feeling that many herbicides are "safe". However, this can also be misleading unless it is realized that sub-lethal dosages of various herbicides in various animals have been shown to induce a variety of pathological symptoms, including nausea, vomiting, lethargy, loss of muscular coordination, myotomia, coma, stiffness of extremities, kidney and liver damage, loss of weight, spasticity, and bleeding of the gums. Clearly, herbicides are toxic if an organism receives an adequate dose, but the levels required to produce such symptoms will rarely be experienced by wildlife in forestry spray operations (Radeleff 1958).

The effects of herbicides on the more diminutive species of wildlife has received even less attention than the larger species, laboratory test animals excepted. However, Morton and Moffett (1972) reported some effects of several herbicides on honey bees. They cited previous studies which have shown that while arsenic and paraquat herbicides are toxic, adult bees are relatively unaffected by exposure to substituted phenoxy, substituted benzoic and substituted picolinic acid herbicides through feeding or spraying. The most adverse effects of these herbicides on the bees has been attributed to loss of food plants killed by the herbicides. They found, however, that while the feeding of picloram 2,3,6-TBA and dicamba to honey bees at 1000 ppm had no adverse effects on brood development, chloramben and dalapon at this concentration reduced brood development while 2,4-D, 2,4,5-T, silvex, 2,4,DB and EPTC severely reduced or eliminated brood production. Concentrations of 10 ppm had no effect while 100 ppm reduced brood production. Brood development resumed when the herbicide was removed from the diet.

Toxicity of herbicides to birds is summarized by Heath et al. (1972). They noted that compared to other pesticides, herbicides generally have a low order of toxicity, with only paraquat and diquat consistently producing mortality in tests. Most acute toxicity LC_{50}'s were greater than 5000 ppm, and in many tests there was no mortality at that concentration (based on 8-day post-treatment mortality). Sub-lethal effects on birds have apparently received less study. However, Kopischke (1972) reported the results of spraying pheasant eggs with either aqueous 2,4-D solution or diesel fuel. The 2,4-D at concentrations comparable to those of a normal field application did not affect hatchability of eggs or cause death or deformity in hatched chicks. Diesel fuel, on the other hand, reduced hatchability to zero.

Concern over the teratogenic effects of herbicides extends to wildlife as well as man. The work on this aspect of herbicide toxicology has largely been conducted on domesticated strains of wildlife (laboratory rats and mice) and it is therefore reasonable to anticipate the potential for a teratological problem in wildlife populations. However, the levels of herbicidal teratogens to which wildlife populations will normally be exposed are likely to be substantially below the levels necessary to produce teratogenic symptoms in laboratory test animals, so the incidence of such symptoms in wildlife populations as a result of forest herbicide usage is likely to be extremely low. Because of heavy natural selection pressures in wild populations, genetic or morphological irregularities induced by herbicides will be rapidly eliminated from the population, so the long-term threat of such effects to wildlife would appear to be insignificant. Wilson (1973) appealed for a calm, dispassionate view of the problem of teratogenicity in pesticides. He pointed out that a large variety of chemicals have teratogenic potential if administered at very high dosages. Correct commercial usage of herbicides in forestry will rarely expose an organism to levels that will create serious problems. While this does not in any way denigrate the legitimate concern over the health and environmental dangers of herbicide use, it does remind us that toxicological information should not be used out of context.

Herbicides are known to affect plant chemistry, and this may render plants more or less suitable for wildlife. In some plants, nitrate concentrations may be substantially increased rendering them toxic to some animals, and there is the case of herbicide induced increases in the hydrocyanic acid content of sudan grass (Fertig 1952, Swanson and Shaw 1954) or wild cherry leaves (Lynn and Barrons 1952). However, these effects do not appear to be general (Buck et al. 1961), and the increases in plant protein and mineral content that sometimes accompany the use of herbicides will tend to improve the nutritional value of plants to wildlife and may also improve their attractiveness and palatability.

Relatively little work has been done on herbicidal residues in wildlife. Newton and Norris (1968) examined levels of 2,4,5-T and atrazine in blacktail deer feeding in recently sprayed areas and concluded that little of the herbicides was absorbed and stored in internal tissues. While their data are not conclusive, they felt that they supported the theory that ruminants are able to degrade these herbicides after ingestion and that there is little uptake and internal storage. Smith (1973) examined the effect of 2,4-D and 2,4,5-T on in-vitro digestion of forage and on growth of rumen microorganisms. He found that the microbial populations were apparently unaffected, but that they were unable to degrade these herbicides.

Norris (1971) noted that the acute toxicity of 2,4-D is fairly uniform among animals, varying from 500-1500 ppm in their feed. 2,4,5-T is slightly more toxic at 200-1200 ppm. Norris states that exposure to such concentrations is unlikely to occur in the forest environment following normal brush control projects. Chronic toxicity from 2,4-D is unlikely because of the rapid disappearance of this herbicide. Picloram is less toxic than either 2,4-D or 2,4,5-T and many animals can apparently tolerate high dosages. Since it is generally applied at much lower rates than 2,4-D and 2,4,5-T, it offers a much lower toxicity hazard than these two herbicides in spite of its greater persistence.

The toxicity of a herbicide to wildlife is highly variable and subject to modification by many factors (Cope 1971). Sometimes there is a diffference according to the sex of the organism, and generally the young are very much more sensitive than fully-grown individuals. Both the chemical form and the formulation of the herbicide are extremely important, with differences in toxicity between different forms and formulations sometimes being greater than differences between different herbicides. Increasing temperature can increase toxicity, while the type of site (climate, vegetation and soil) can affect exposure to the herbicide. Presence of other pesticides may produce synergistic effects that raise or lower the toxicity of individual substances. The type of exposure will influence toxicity: an ingested herbicide may be detoxified and have little or no effect on an organism whereas an organism that receives the herbicide directly through the skin may suffer toxic effects.

Because of these considerations, it is extremely difficult to predict toxicological problems for wildlife from herbicidal manipulation of vegetation. Careful, one-time applications of moderate to small amounts of 2,4-D, 2,4,5-T and picloram applied at a time that does not expose wildlife directly to the spray would appear to cause little reason for concern for the safety and well-being of wildlife species. On the other hand, careless application of larger dosages, that involves direct impact on animals, particularly when using the more toxic types and formulations of herbicides, could pose unacceptable problems.

2.4 Effects on Soil Organisms

Most herbicides applied in either agriculture or forestry eventually end up in the soil. Because herbicides are basically biological poisons, and because of the importance of soil organisms in soil fertility, considerable interest has been focused on the effects of herbicides on the numbers, composition and the function of soil biota. Interest has focused, in particular, on effects on microorganisms involved in nitrogen fixation, mobilization and release. Audus (1964) noted that the soil is a very complex and dynamic system, the fertility of which often depends upon the very delicate balance that exists between the various types of microorganisms involved in the various nutrient cycles. Most of the research has focused on the microflora (fungi and bacteria), and there is much less information available on the effects of herbicides on micro and macro fauna. Fletcher (1960, 1966), Bollen (1961) and Audus (1964) have reviewed the effects of herbicides on soil microorganisms and much of the following, largely unreferenced, discussion is adapted from these papers. The general conclusion reached by these reviewers is that the great majority of herbicides, particularly those commonly used in forestry, appear to have no permanent undesirable effects on soil microflora. Even the arsenical herbicides, cacodylic acid and MSMA, do not appear to have a major effect on forest floor microbes, with little or no effect at concentrations that may be expected in the field (Bollen et al. 1974).

It is generally agreed that 2,4-D and 2,4,5-T have no lasting effects on the total number of soil microorganisms, although at higher concentrations (10^3 - 10^4 ppm), 2,4-D may cause a decline in bacterial numbers. In alkaline soils this is followed by a resurgence in the bacterial population, but in acid soils the recovery is slower because of the greater toxicity of 2,4-D to soil microorganisms at low pH.

Not all soil organisms are equally affected by phenoxy herbicides. Aerobic forms are more affected than facultative anaerobic types, and gram-positive bacteria are inhibited by lower concentrations of 2,4-D and 2,4,5-T than gram-negative forms. Spore-forming bacteria are more sensitive than non-spore forming types. Actinomycetes have generally been reported as being relatively resistant to the phenoxy acetic acid herbicides, although there is a variation in sensitivity between species, while fungi are generally thought of as being more resistant than bacteria. As with higher forms of life, the chemical form of these herbicides appears to play an important role in determining the effects of herbicides on microorganisms.

The contention that many microbes are insensitive to most herbicides, at least at lower concentrations, is supported by the finding that for many herbicides there is at least one species of microbe capable of adapting or mutating to a form that can utilize the herbicide as its sole or principal source of energy and carbon. Certain forms have even been found which grow better in the presence of MCPA or 2,4-D than in the absence of this chemical, and several authors have observed the stimulation of saprophytic bacteria by 2,4-D.

Many soil fungi are stimulated by herbicidal applications, at least at lower concentrations. For example, at concentrations of less than 100 ppm, 2,4-D enhanced the formation of citric acid by Aspergillus niger, while this was inhibited at concentrations greater than 100 ppm. Several species of pathogenic fungi are inhibited by higher levels (up to 40,000 ppm) of phenoxy acetic acids, and the combination of these herbicides with fungicides has been shown to enhance the activity of the latter. However, some pathogens are stimulated by lower concentrations of certain herbicides. The effects on fungi may not be a direct inhibition or stimulation. It was found in one study, for example, that fungi may respond to phenoxy herbicides by producing an antibiotic which alters their competitive relationships with other microbes. This could be a very important indirect effect of herbicides but we do not know enough about it to be certain of its practical significance.

Production of inhibitory effects of herbicides on soil microorganisms in soil requires high concentrations: well above those encountered in normal forest herbicide applications. However, in solution cultures it is possible to elicit effects at much lower levels. For example, 2,4-D has been shown to severely retard nitrification at 3 ppm in solution culture, although the addition of soil eliminated any inhibition at normal field concentrations of up to 18 ppm (Fletcher 1960). The degree to which such inhibition is alleviated by soil will depend upon the type of soil. It has been hypothesized that the toxicity of 2,4-D is related to the quantity of exchangeable magnesium in the soil. 2,4-D is apparently particularly effective at chelating magnesium, which is required by microbes for oxydation and phosphorylation reactions, and the toxicity of 2,4-D to microbes has been shown to be alleviated by the addition of magnesium sulphate. Thus, a soil rich in exchangeable magnesium may have a greater ameliorative effect on the toxicity of 2,4-D to soil microbes than one low in exchangeable magnesium.

The effects of herbicides on nitrification processes have aroused particular interest because of the importance of this microbial activity in controlling the availability and leaching losses of nitrogen. Many studies have shown that phenoxyacetic acids at normal application rates inhibit nitrification in pure culture, but that addition of soil almost completely counteracts the effect. The inhibition is normally followed by a recovery, presumably by microbial adaptation or the development of detoxifying organisms. Otten et al. (1957) noted that it was doubtful whether any herbicide subject to rapid microbial decomposition would have any important effects on nitrification. Hale et al. (1957) reported that CIPC completely inhibited growth of nitrifying organisms in fresh field soil at 80 ppm, while up to 40 ppm of monuron had no effect. There was a 90% reduction in nitrification by CIPC at 160 ppm in soil experimentally enriched with nitrifying organisms, while monuron at 40 ppm caused a 50% reduction. However, concentrations resulting from normal field application rates were not expected to result in any detrimental effect on nitrification.

Picloram does not appear to alter nitrification (Grover 1972, Goring et al. 1967, Tu and Bollen 1969) at levels of up to 300 ppm. At 100 ppm and above, O_2 uptake is enhanced indicating some effect on microbes (Grover 1972), but a large number of soil organisms have been shown to be capable of growth at concentrations of picloram of up to 1000 ppm: of those studied only Thiobacillus thioxidans was affected at concentrations greater than 100 ppm. There were no important effects of picloram at 100 ppm on CO_2 evolution, urea hydrolysis, number of bacteria and fungi, and nitrification to nitrate or nitrite. Goring et al. (1967) concluded that if picloram does have any effects on microorganisms, the effects must be very subtle. Arnold et al. (1966) found that while Aspergillus niger proliferation was reduced by 2,4-D at 10 and 50 ppm, picloram had no effect up to 50 ppm. A. niger was able to degrade both 2,4-D and picloram, but was more effective on the former. Debona and Audus (1970) noted that Nitrosomonas is thought to be more sensitive to herbicides than Nitrobacter. There seems to be an adaptive loss of sensitivity to picloram with time, and compared with several other herbicides, picloram had little effect on the nitrification process.

Ammonification, the conversion of proteins and other complex nitrogenous compounds to ammonium compounds also appears to be largely unaffected by herbicides: indeed, nitrogen containing herbicides are ammonified by soil microorganisms.

Nitrogen fixation by the free-living aerobic Azotobacter is apparently unaffected by herbicides at normal concentrations, although high concentrations of some herbicides may inhibit growth and respiration of these microorganisms. However, some herbicides have been shown to inhibit growth and respiration while having little effect on nitrification (e.g. sodium chlorate). Inhibition of growth and respiration can be aggravated by the addition of inorganic phosphate and ameliorated by the addition of magnesium ions.

Fixation of nitrogen by bacteria of the Rhizobium genus can be affected by herbicides by direct effects on the bacteria, by affecting their invasion of the root, and by affecting the formation of nodules. The effects of 2,4-D and 2,4,5-T on rhizobia at normal concentrations are negligible, although growth in culture can be inhibited at high concentrations (300-8000 ppm). There is some evidence that rhizobia can adapt to inhibitory concentrations of several phenoxy herbicides, but not to 2,4,5-T. There have been some recorded cases of reduced nodulation in the presence of phytotoxic concentrations of phenoxy herbicides, but this is probably resulted from the effects of the herbicide on the plant rather than on the bacteria. One paper reported a significant drop in nitrogen fixation in lucern at 2,4-D and MCPA concentrations that have no effect on plant growth or degree of nodulation. This drop in nitrogen fixation might reflect a change in species balance of the bacterial populations within the nodule.

Horvath and Hunyadi (1973) reported that trifluralin inhibited the multiplication of alfalfa and tobacco mozaic viruses. Bounds and Colmer (1964) found that recommended doses of silvex, dalapon, 2,4-D and 2,4,5-T did not reduce the numbers of streptomyces, while great reductions occurred at 1000 x this dose. Zabel and O'Neil (1957) reported that several organic arsenicals demonstrated bactericidal and fungicidal activity at concentrations similar to commercial slimicides.

Besides their direct effect (or lack thereof) on soil microorganisms, herbicides can have an important indirect effect. By killing green vegetation, herbicides can greatly alter the quantity and chemical quality (which controls palatability, nutritional values and metabolic availability) of the microbial substrate. This can result in a great stimulation of soil microbial activity with important implications for biogeochemistry (e.g. Likens et al. 1969). This is discussed further in Chapter 3.

In conclusion, it would appear that at the concentrations normally used in forest practice, and with the susceptibility to biodegradation that is characteristic of many of the organic herbicides used in forestry, the risk of serious and permanent interference with normal soil microbiological processes is remote. This does not rule out the possibility of soil microbial problems in environments such as forest nurseries where repeated applications may be made, and we still do not know enough about the more subtle effects of herbicides in affecting the competitive relationships between different components of the soil microflora. These relationships have important implications for plant fertility and pathology and should be kept in mind.

Information about the effects of herbicides on soil micro and macrofauna are scarce. Some herbicides have insecticidal properties and, as already discussed, several have significant toxicological implications for higher animals. The question of the effects of herbicides on soil fauna is therefore referred to the section on wildlife.

2.5 Effects on Aquatic Organisms

The literature on effects of herbicides on aquatic organisms reveals that they are significantly more sensitive than terrestrial organisms to most herbicides. Several of the herbicides have moderate water solubility resulting in a more continuous exposure of fish and aquatic invertebrates to the herbicide than of terrestrial organisms.

Norris (1971) noted that while the "no-effect" concentration (concentration in a diet for a limited exposure period which causes no acute toxic effects) for 2,4-D was 720 ppm in birds, 1500 ppm in rodents, 2400 ppm in ruminants and 500 ppm in non-ruminant mammals, it was 0.1 ppm for fish and other aquatic organisms. For 2,4,5-T terrestrial animals exhibit 200-1200 ppm "no-effect" concentrations, while fish and aquatic organisms vary from 0.05-0.1 ppm. Comparable figures for amitrole are given as 2000 + ppm and 2-32 ppm, and for picloram as 1000-3000 ppm and 0.1-1.0 ppm.

As was the case for terrestrial wildlife, the chemical form and the formulation of the herbicide have a tremendous influence on toxicity for aquatic organisms. Median tolerance limit (Tlm) of bluegill sunfish to 2,4-D over 24 hours varied from 900 ppm acid equivalent (a.e.) for the alkanolamine (ethanol and isoproponal series) form to 0.9 ppm a.e. for the isopropyl ester. Values for 2,4,5-T vary between 144 ppm a.e. for the dimethylamine form and 1.4 for the butoxyethanol ester (Hughes and

Davis 1963). Bond et al. (1959), Juntunen and Norris (1972) and Meehan, Norris and Sears (1974) presented similar information, while Kenaga (1969) presented data on a variety of formulations of Tordon herbicides. Sensitivity (LC_{50}24 hours) of rainbow trout varied from 303 ppm a.e. for Tordon M-3088, 2,4-D TIPA salt down to 1.3 ppm a.e. for the isooctyl ester of Tordon 155 acid.

The toxicity of herbicides to aquatic organisms is highly influenced by the chemistry of their environment (Cope 1971). Availability of herbicides for uptake, uptake via the gills, and the toxicity of the herbicidal material are all subject to modification according to the pH and chemistry of the water. Streams and lakes rich in silt, clay and organic material will exhibit far less severe herbicide toxicity problems than streams of pure water flowing in clean, rocky streambeds.

In many forest streams there is a significant amount of organic debris, clay and silt which rapidly absorb herbicides out of solution. Tarrant and Norris (1967) and Norris (1967) have shown that concentrations of herbicides in streams flowing through sprayed areas decrease rapidly with time and with distance downstream. In none of the data they presented were concentrations greater than 1.0 ppm observed, and they generally fell to two orders of magnitude below this within a few hours. Thus, although aquatic organisms are much more sensitive to the direct toxic effects of herbicides than terrestrial wildlife, the environment in which they occur acts to prevent or minimize exposure to levels of herbicides sufficiently high to produce symptoms of acute toxicity. Because of the rapid drop in concentration exhibited in sprayed streams and lakes, problems of chronic toxicity probably do not occur.

There is very little published information on rate of elimination of herbicide residues from aquatic organisms. Rodgers and Stalling (1972) reported that peak residues of 2,4-D in most organs of three species of fish in an aquarium experiment occurred within 1-2 hours for fed fish and 1-8 hours for fasted fish. Fasted fish accumulated 2-5 times greater residues than fed fish. Maximum accumulations were 7-55 times (depending on organ) greater than the exposure concentrations (dissolved in water) one to six hours after exposure. It was also noted that the butoxyethanol ester of 2,4-D used was hydrolysed to 2,4-D acid very much faster in the presence of fish than in their absence. Thus, the fate of herbicides in aquatic ecosystems may be very much dependent upon the extent of biological activity. Elimination of residues following exposure was rapid, and was correlated with the rate at which the ester was hydrolysed. The authors concluded that in a natural situation, fish would be exposed to the ester for longer than was the case in their experiment, resulting in higher peak residue levels and slower elimination of the residues.

Much of the recent concern over 2,4,5-T has been related to the contaminant dioxin: a substance known to be highly toxic to animals. Little work has been done on its toxicity to aquatic organisms, but Norris and Miller (1974) reported that guppies exposed to concentrations of 0.l parts per billion or more for 120 hours died within 35 days. They noted that environmental and threshold response levels of dioxin must be determined before the hazard of dioxin to aquatic organisms can be determined. Because of the apparent extreme toxicity of this substance, great care must obviously be taken to avoid use of herbicides containing it. Another study (Miller, Norris and Hawkes 1973) involving several major classes of aquatic organisms revealed that dioxin is toxic to fish in either food or water. 24-96-hour exposures of young salmon were irreversible and resulted in death after 10-80 days. Some aquatic invertebrates showed reduced reproductive success with exposures to 0.2 parts per billion. While careful application of 2,4,5-T should not result in stream concentrations above 0.l ppb, these authors felt that the extreme sensitivity of aquatic organisms to this substance urgently requires that considerable work be done to study the movement and accumulation of dioxin in the environment.

Sub-lethal toxic effects of herbicides on fish have received much less attention. Speed, stamina and strength are important parameters for the survival and completion of the life cycle, especially in migratory (anadramous) fish and if these are significantly affected, the results for the population may be no different from fatal toxicity. Fish are also known to be very sensitive to trace levels of chemicals in water, and herbicide residues in streams could possibly influence migration from the ocean to fresh-water spawning grounds. Until better information on this question is available it would seem prudent to avoid using herbicides at a time when downstream migratory fish would experience even trace concentrations. The first fall rains are known to be important in initiating upstream migration, but they are also known to flush herbicide residues from sprayed areas.

Cope (1971) pointed out that the young developmental stages of organisms are generally much more sensitive to pesticides than mature individuals. Most fish-toxicity tests are conducted on fish of undefined age and there is little or no data on effects on developing eggs and very young fish. Many small ephemeral forest streams are important nursery areas for young salmonids and no studies were found that investigated the effects of herbicide sprays on these fish: because of the low water flows in these streams, significantly higher herbicide concentrations are probably experienced in these small streams than those published by Norris et al. (op. cit.).

In addition to direct toxic effects, herbicides can have a variety of indirect effects. They can decrease phytoplankton and aquatic macrophyte production (Butler 1965) thus affecting food webs. Diuron at 1.4 ppm has been shown to change the natural bacterial population balance existing in lake aquatic ecosystems. Aerobic, heterotrophic bacteria were reduced and chromagenic bacteria were inhibited (Guthrie et al. 1974). Herbicides can kill aquatic insects that are food for fish, and by killing streambank vegetation they can expose fish to increased water temperature, reduced input of terrestrial insects and leaf fall, increased predation, and reduced streambank stability. Thus, as with terrestrial wildlife, the indirect effects of herbicides on fish and other aquatic organisms are probably more serious than direct toxic effects.

2.6 Effects on Man

It would perhaps be appropriate to start this section with two quotations from the Mrak (1969) Report.

1. (p. 236) ..."the field of pesticide toxicology exemplifies the absurdity of a situation in which 200 million Americans are undergoing life-long exposure, yet our knowledge of what is happening to them is at best fragmentary and for the most part indirect and inferential. While there is little ground for forebodings of disaster, there is even less for complacency".

2. (p. 508) ..."A particularly subtle danger from wide scale use of pesticides lies in the possibility that some of them may be damaging to the hereditary material. If this is so, we may be unwittingly harming our descendants. Whether this is happening, and if so, what is the magnitude of the effect, is regrettably unknown. Surely one of the greatest responsibilities of our generation is our temporary custody of the genetic heritage received from our ancestors. We must make every reasonable effort to ensure that this heritage is passed on to future generations undamaged. To do less, we believe , is grossly irresponsible".

Concern over the implications of herbicides for public health received a great stimulation with the discovery of the teratogenicity of 2,4,5-T and its impurity, dioxin. With the exception of arsenicals, most commonly used herbicides have traditionally been considered relatively safe for man because of their moderate acute toxicity and their prompt elimination of urine. This designation is perhaps unwise since it refers to the acute toxicity of expected dosages rather than the inherent acute toxicity of herbicides. For example, Goldstein et al. (1959) described three cases of severe sensory and motor symptoms necessitating hospitalization of individuals who had accidentally permitted small quantities of solutions of esters of 2,4-D to contact and remain on their skin for several hours. Pain developed following exposure, followed by peresthesia and severe paralysis. Disability was protracted, recovery was incomplete after several years, and neurological damage was diagnosed. Thus, many of the herbicides that may indeed be safe (as far as acute toxicity is concerned) at low concentrations may be extremely hazardous should accidental direct exposure of humans occur.

Johnson (1971) reviewed the public health implications of phenoxy and picloram herbicides. An examination of 220 men exposed to 30-40 mg/day of 2,4-D or 2-8 mg/day of 2,4,5-T in manufacturing plants revealed no differences in 20 laboratory tests in comparison with 4600 control individuals. Ten of the 220 men were karyotyped and no changes in genetic material were found (Dow Chemical Co. unpublished data: details of tests and statistical analysis of results not presented). Johnson discussed the available literature on toxicity and teratogenicity of these herbicides and dioxin to test animals (mice, rats, hamsters, sheep and rabbits) and reviewed the production of dioxin from 2,4,5-T by burning. He concluded that dioxin is highly toxic, that 2,4-D and 2,4,5-T are only moderately toxic, and that picloram has a low acute oral toxicity to rats and guinea pigs. For example, the LD_{50} as mg/Kg body weight for rats is 8200 for picloram, 500 for 2,4,5-T, 375-666 for 2,4-D, but only 0.022-0.045 for dioxin. In guinea pigs, the LD_{50} for dioxin is only 0.0006, while it is 3000.0 for picloram. Johnson concluded "...that widespread use of phenoxy herbicides has produced no demonstrable evidence of potential harm to man. The herbicides used most widely (2,4-D and 2,4,5-T) are degraded and do not bioconcentrate. Moreover, the comparative toxicity shows these materials to be well tolerated in a variety of test systems. Man is not exposed to harmful concentrations. Impurities can be an important factor - particularly the chlorodibenzo-p-dioxins- but these can be controlled by proper manufacturing techniques. As always, care in application is an important part of safe practice...".

Montgomery and Norris (1970) evaluated the hazard of 2,4,5-T in the forest environment as low. They noted that it has a higher toxicity than several other herbicides, with an LD_{50} for rats of 300 mg/Kg body weight (a higher toxicity than that given by Johnson (1971)) and 100 mg/Kg for dogs. However, in one experiment, sheep fed 100 mg/Kg 2,4,5-T ester daily for 481 days were apparently unaffected (at least, for the parameters examined), while in another experiment 369 daily doses of this magnitude were required to kill the sheep (Palmer and Radeleff 1964). Sub-lethal, teratogenic, carcinogenic and mutagenic effects were not reported.

Hayes (1969) noted that much of our knowledge of herbicide toxicology is based on animal experiments. However, ultimate assurance about human safety must come from studies of people with intensive and prolonged exposure. In spite of this fundamental deficiency there is "strong and growing evidence that long-term adsorption of traces of one or more of the synthetic, non-metallic pesticides will not lead to illness". Mullison (1973) exemplifies the many authors who readily admit the inherent toxicity of herbicides, but who stress that "the toxicity of a substance is determined by its dosage ... toxicity, therefore, is a quantitative chemical property under a given set of conditions ... chemicals normally considered toxic or poisonous have some dosage below which they cause no harmful effects. For example, the poison arsenic is used safely as a medicine".

Rowe (1951) stressed hazard as opposed to toxicity, which will not express itself unless an organism is exposed to toxic dosages. While many herbicides are toxic, the likelihood of an organism being exposed to toxic dosages as the result of careful applications in the forest environment is small (Montgomery and Norris 1970).

Arsenical chemicals are generally considered to be more toxic than the other commonly used herbicides, and considerable attention has been given to the hazard of MSMA and cacodylic acid to applicators (e.g. Norris 1971, Tarrant and Allard 1972). It has been found that workers applying arsenical silvicides do absorb arsenic but that much of the chemical appears to be excreted from the body in a short time. There appears to be no evidence of a continuing increase in arsenic levels after the first week over a period of more than two months. However, the levels of arsenic in urine from workers applying these types of herbicides is sometimes greater than that recommended by manufacturers, implying the need for greater care in use of arsenical herbicides.

Good summaries of the public health aspects of the phenoxy and picloram herbicides can be found in Mrak (1969), Leopold et al. (1971) and Johnson (1971). Amitrole was judged positive for tumor induction in mice while monuron, 2(2,4-DP), 2,4-D isopropyl ester and 2(2,4,5-trichlorophenoxy) propionic acid were all judged to show some significant increase in tumor induction in mice. A variety of other phenoxy herbicides were found to be negative for tumor induction, but since they have only been tested in one species, they have not been given clearance from suspicion (Mrak 1969). Since mutagenicity has already been discussed above, it will not be dealt with in detail. The concerned reader is recommended to examine pages 568-572 in the Mrak Commission Report (1969) which make sober reading. This report also noted that mutagenic and/or chromosome breaking chemicals are as effective in animal test systems as in plant material, so that much of the discussion on mutagenicity of herbicides in plants earlier in this review is probably germane for human health.

In concluding this section it should be noted that herbicides may have either beneficial or detrimental effects on man. They increase his food supply and reduce the cost of its production. They aid in the growing of trees and help create habitat for desired species of wildlife. They aid in the control of undesirable vegetation in a large variety of situations. Conversely, they may eliminate rare plants, reduce the complexity and stability of ecological systems, eliminate desirable animals through the destruction of their food species or habitat, and they are responsible for the creation of new weed pests. They are toxic materials and are capable of inducing a variety of toxicological symptoms in humans under certain circumstances.

While many herbicides clearly present a potential hazard of acute and chronic toxicity, carcinogenicity, teratogenicity and mutagenicity to humans, the exposure of humans to herbicides in most forest usage will be well below the dosage thought to induce these symptoms. Much of the evidence on the toxicology of herbicides is derived from test organisms in laboratory situations, and the extrapolation of this data to humans is, at best, open to question. However, there will never be room for complacency over the human health implications of herbicides, and we cannot tolerate even low levels of mutation induction in the human population. While there are no human health grounds for the total restriction of herbicide usage in forests, there are sufficient concerns to require extreme care and caution, and adequate levels of education and training on the part of applicators. While there can be little doubt that the use of herbicides in forestry poses a far smaller threat of human exposure than does the agricultural or home use of herbicides, this does not make the risk of even small exposures from this source any more acceptable.

References Cited in Chapter 2

Anderson, J.L. and W.W. Thomson. 1973. The effects of herbicides on the ultrastructure of plant cells. Residue Rev. 47: 167-189.

Arnold, W.R., P.W. Santelmann and J.Q. Lynd. 1966. Picloram and 2,4-D effects with Aspergillus niger proliferation. Weeds 14: 89-90.

Ashton, F.M. and A.S. Crafts. 1973. Mode of Action of Herbicides. Wiley & Sons, New York, 504 p.

Audus, L.J. 1964. Herbicide behaviour in the soil. Chap. 5, In The Physiology and Biochemistry of Herbicides. L.J. Audus, ed., Academic Press, N.Y.

Azevedo, J. 1973. Safety to wildlife. Calif. Weed Contr. Conf. Proc. 25: 88-90.

Bachelard, E.P. and R.D. Ayling. 1971. The effects of picloram and 2,4-D on plant cell membranes. Weed Res. 11: 31-36.

Bohmont, B.L. 1967. Toxicity of herbicides to livestock, fish, honey bees and wildlife. West. Weed Contr. Conf., Proc. 21: 25-27.

Bollen, W.B. 1961. Interactions between pesticides and soil microorganisms. Am. Res. Microbiol. 15: 69-92.

Bollen, W.B., L.A. Norris and K.L. Stowers. 1974. Effect of cacodylic acid and MSMA in microbes in forest floor and soil. Weed Sci. 22: 557-562.

Bond, C.E., R.H. Lewis and J.L. Fryer. 1959. Toxicity of various herbicidal material to fishes. Transactions, 1959 Seminar, Biol. Probl. Water Pollution. R.A. Taft Sanitary Engineering Center. Tech. Rept. W60-3. Oregon State College, Corvallis, Oregon.

Bounds, H.C. and A.R. Colmer. 1964. Resistance of Streptomyces to herbicides. Sugar Bull. Aug. 1964, p. 274-276.

Bradely, M.V. and J.C. Crane. 1955. The effect of 2,4,5-trichlorophenoxyacetic acid on cell and nuclear size and endopolyploidy in parenchyma of apricot fruits. Amer. J. Bot. 42: 273-281.

Bramble, W.C. and W.R. Byrnes. 1972. A long-term ecological study of game food and cover on a sprayed utility right-of-way. Purdue Univ. Agr. Exp. Sta. Bul. No. 885, 20 p.

Brown, E.R. 1967. Impact of range improvement on wildlife habitat. p. 203-247. In Herbicides and Vegetation Management. Symp. Proc., Oregon State Univ., Corvallis, Oregon.

Buck, W.B., W. Burns, L. James and M.C. Williams. 1961. Results of feeding of herbicide-treated plants to calves and sheep. J. Amer. Vet. Med. Assoc. 138: 320-323.

Buffam, P.E. 1971. Spruce beetle suppression in trap trees treated with cacodylic acid. J. Econ. Entomol. 64: 958-960.

Buffam, P.E. and W.H. Flake, Jr. 1971. Roundheaded pine beetle mortality in cacodylic acid-treated trees. J. Econ. Entomol. 64: 969-970.

Butler, P.A. 1965. Commercial fishery investigations. p. 65-77. In The Effects of Pesticides on Fish and Wildlife. Fish and Wildlife Service Circ. 276. Dept. of Interior, U.S., Washington, D.C.

Chansler, J.F. and D.A. Pierce. 1966. Bark beetle mortality in trees injected with cacodylic acid (herbicide). J. Econ. Entomol. 59: 1357-1359.

Clark, D.E., J.E. Young, R.L. Younger, L.M. Hunt and J.K. McLaren. 1964. The fate of 2,4-dichlorophenoxyacetic acid in sheep. Agric. Food Chem. 12: 43-45.

Cope, O.B. 1971. Interactions between pesticides and wildlife. Annu. Rev. Entomol. 16: 325-364.

Coulter. L.L. 1958. The role of herbicides in wildlife production through creation and stabilization of habitats. Down To Earth, Spring 1958, 4 p.

Courtney, K.D., D.W. Gaylor, M.D. Hogan et al. 1970. Teratogenic evaluation of 2,4,5-T. Science 168: 864-866.

Courtney, K.D. and J.A. Moore. 1971. Teratology studies with 2,4,5-Trichlorophenoxyacetic acid and 2,3,7, 8-Tetrachlorodibenzo-p-dioxin. Toxicol. Appl. Pharmacol. 20: 296-403.

Dävring, L. and M. Surner. 1971. Cytogenetic effects of 2,4,5-trichlorophenoxyacetic acid on oogenesis and early embryogenesis in Drosophila melanogaster. Hereditas 68: 115-122.

Debona, A.C. and L.J. Andus. 1970. Studies on the effects of herbicides on soil nitrification. Weed Res. 10: 250-263.

Dickinson, J.O. 1972. Toxicity of the arsenical herbicide monosodium acid methanearsenate in cattle. Am. J. Vet. Res. 33: 1889-1892.

Dodge, J.D. and G.B. Lawes. 1974. Some effects of the herbicides diquat and morfamquat on the fine structure of leaf cells. Weed Res. 14: 45-49.

Doxey, D. and A. Rhodes. 1949. The effect of the plant growth regulator 4-chloro-2-methylphenoxyacetic acid on mitosis in the onion (Allium cepa). Ann. Bot., Ser. 2, 13: 105-111.

Drill, V.A. and T. Hiratzka. 1953. Toxicity of 2,4-dichlorophenoxyacetic acid and 2,4,5-trichlorophenoxyacetic acid. A report on their acute and chronic toxicity in dogs. Arch. Ind. Hyg. Occupational Med. 7: 61-67.

Dunigan, E.P., J.P. Frey, L.D. Allen, Jr. and A. McMahon. 1972. Herbicidal effects on the nodulation of Glycine max (L.) Merrill, Agron. J. 64: 806-808.

Exon, J.H., J.R. Harr and R.R. Claeys. 1974. The effects of long-term feeding of monosodium acid methanearsenate (MSMA) to rabbits. Nutr. Repts. Int. 9: 351-357.

Fertig, S.N. 1952. Livestock poisoning from herbicide treated vegetation. Proc. 6th Ann. N.E. Weed Contr. Conf. 6: 13-19.

Fishbein, L., W.G. Flaman and H.L. Falk. 1970. Chemical Mutagens: Environmental Effects on Biological Systems. Academic Press, New York, 364 p.

Fisher, D.E., L.E. St. John, W.H. Gutenmann et al. 1965. Fate of Banvel T, Tordon, and Trifluoralin in the dairy cow. J. Dairy Sci. 48: 1711-1715.

Fletcher, W.W. 1960. The effect of herbicides on soil microorganisms. p. 20-62. In Herbicides in the Soil, Blackwell Sci. Publ., Oxford.

Fletcher, W.W. 1966. Herbicides and the bio-activity of the soil. Landbouwk Tijdschr. 78: 274-281.

Freese, E. 1971. Molecular mechanisms of mutations. Chp. 1, In Chemical Mutagens: Principles and Methods for Their Detection, A. Hollaender, ed., Plenum Press, New York, Vol. I.

Frye, R.H. and N.D. Wygant. 1971. Spruce beetle mortality in cacodylic acid-treated Engelmann spruce trap trees. J. Econ. Entomol. 64: 911-916.

Goldstein, N.P., P.H. Jones and J.R. Brown. 1959. Peripheral neuropathy after exposure to an ester of dichlorophenoxy-acetic acid. J. Amer. Med. Assoc. 171: 1306-1309.

Goring, C.A.I., J.D. Griffith, F.C. O'Melia, H.H. Scott and C.R. Youngson. 1967. The effect of Tordon on microorganisms and soil biological processes. Down To Earth 22: 14-17.

Forter, C.J. and W. van der Zweep. 1964. Morphological effects of herbicides. p. 237-275. In The Physiology and Biochemistry of Herbicides, L.J. Audus, ed., Academic Press, New York.

Grant, W.F. 1971a. The case of mutagenic testing of chemical pollutants. Can. Field Nat. 85: 203-204.

Grant, W.F. 1971b. Book review of Chemical Mutagenesis in Mammals and Man. Can. Field Nat. 85: 203-204.

Grant, W.F. 1972. Pesticides -- subtle promoters of evolution. Symp. Biol. Hung. 12: 43-50.

Grant, W.F. and P.M. Harney. 1960. Cytogenetic effects of maleic hydrazide treatment of tomato seed. Can. J. Genet. Cytol. 2: 162-174.

Grigsby, B.H. and E.D. Farwell. 1950. Some effects of herbicides on pasture and on grazing of livestock. Mich. Agr. Exp. Sta., Quart. Bull. 32: 378-385.

Grover, R. 1972. Effects of picloram on some soil microbial activities. Weed Res. 12: 112-114.

Guthrie, R.K., D.S. Cherry, and R.N. Ferebee. 1974. The effects of diuron on bacterial populations in aquatic environments. Water Res. Bull. 10: 304-310.

Gysel, L.W. 1957. Effects of different methods of releasing pine on wildlife food and cover. Down To Earth. Fall 1957, 2 p.

Hale, M.G., F.H. Hulcher and W.E. Chappell. 1957. The effects of several herbicides on nitrification in a field soil under laboratory conditions. Weeds 5: 331-341.

Hayes, W.J., Jr. 1969. Pesticides and human toxicity. Annals N.Y. Acad. Sci. 160: 40-54.

Heath, R.G., J.W. Spann, E.F. Hill and J.F. Kreitzer. 1972. Comparative dietary toxicities of pesticides to birds. U.S. Dept. Int. Fish and Wildl. Serv. Bur. Sport. Fish and Wildl., Spec. Sci. Rep. - Wildl. No. 152, 57 p.

Hollaender, A. (ed.) 1971. Chemical Mutagens: Principles and Methods for Their Detection. Plenum Press, New York, Vol. 1, 310 p., Vol. 2, 311-605 p.

Horvath, J. and K. Hunyadi. 1973. Studies of the effect of herbicides on virus multiplication. I. Effects of trifluralin to alfalfa mosaic and tobacco mosaic viruses on Phaseolus vulgaris L. cv. Pinto. Acta Phytopath. 8: 347-350.

Hughes, J.S. and J.T. Davis. 1963. Variations in toxicity to bluegill sunfish of phenoxy herbicides. Weeds 11: 50-54.

Juntenen, E.T. and L.A. Norris. 1972. Field application of herbicides - avoiding danger to fish. Special Report 354, Agric. Exp. Sta., Oregon State Univ., Corvallis, Oregon, 26 p.

Johnson, J.E. 1971. The public health implications of widespread use of the phenoxy herbicides and picloram. Bio-Sci. 21: 899-905.

Keith, J.O., R.M. Hansen and A.L. Ward. 1959. Effect of 2,4-D on abundance and foods of pocket gophers. J. Wildl. 23: 137-145.

Kenaga, E.E. 1969. Tordon herbicides - evaluation of safety to fish and birds. Down To Earth 25: 5-9.

Key, J.L. 1969. Hormones and nucleic acid metabolism. Annu. Rev. Plant Physiol. 20: 449-474.

Klingman, D.L., C.H. Gordon, G. Yip and H.P. Burchfield. 1966. Residues in the forage and in milk from cows grazing forage treated with esters of 2,4-D. Weeds 14: 164-167.

Krefting, L.W. and H.L. Hansen. 1969. Increasing browse for deer by aerial applications of 2,4-D. J. Wildl. Manage. 33: 784-790.

Kopischke, E.D. 1972. The effect of 2,4-D and diesel fuel on egg hatchability. J. Wildl. Mgmt. 36: 1353-1356.

Lawrence, W.H. 1967. Effects of vegetation management on wildlife. p. 88-93. In Herbicides and Vegetation Management. Symp. Proc., Oregon State Univ., Corvallis, Oregon.

Leonard, J.W. and S.A. Cain. 1961. The role of herbicides in wildlife management. Rec. Adv. Bot. Sect. 12: 1422-1426.

Leopold, C.M. et al. 1971. Report on 2,4,5-T. A Report of the Panel on Herbicides of the President's Science Advisory Committee. Exec. Off. Pres., Off. Sci. Tech. 68 p.

Likens, G.E., F.H. Bormann and N.M. Johnson. 1969. Nitrification: importance to nutrient losses from a cutover forested ecosystem. Science 163: 1205-1206.

Lynn, G.E. and K.C. Barrons. 1952. The hydrocyanic acid (HCN) content of wild cherry leaves sprayed with a brush killer containing low volatile esters of 2,4-D and 2,4,5-T. Proc. 6th N.E. Weed Control Conf.: 331-333.

MacLeod, C.M. et al. 1971. Report on 2,4,5-T. A report of the Panel on Herbicides of the President's Science Advisory Committee. U.S. Govt. Printing Office, Washington, D.C. 68 p.

Meehan, W.R., L.A. Norris and H.S. Sears. 1974. Toxicity of various formulations of 2,4-D to salmonids in southeast Alaska. J. Fisheries Res. Bd. Canada, 31: 480-485.

Miller, R.A., L.A. Norris and C.L. Hawkes. 1973. Toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in aquatic organisms. Environ. Health Perspectives, Sept. 1973: 177-186.

Mohandas, T. and W.F. Grant. 1972. Cytogenetic effects of 2,4-D and amitrole in relation to nuclear volume and DNA content in some higher plants. Can. J. Genet. Cytol. 14: 773-783.

Mohandas, T. and W.F. Grant. 1973. A relationship between nuclear volume and response to auxin herbicides for some weed species. Can. J. Bot. 51: 1133-1136.

Montgomery, M.L. and L.A. Norris. 1970. A preliminary evaluation of the hazards of 2,4,5-T in the forest environment. Pac. N.W. For. Range Exp. Sta., Res. Note PNW-116, 11 p.

Morton, H.L. and J.O. Moffett. 1972. Ovicidal and larvicidal effects of certain herbicides on honey bees. Environ. Entomol. 1: 611-614.

Mrak, E.M. et al. 1969. Report of the Secretary's Commission on Pesticides and their Relationship to Environmental Health. Parts I and II. E.M. Mrak, Chairman. U.S. Dept. Health. Education and Welfare, Washington, D.C., 677 p.

Mullison, W.R. 1973. Herbicides - are they safe? Down To Earth 29: 6-11.

Newcombe, H.B. 1971. Techniques for monitoring and assessing the significance of mutagenesis in human populations. Chp. 26, In Chemical Mutagens: Principles and Methods for Their Detection, A. Hollaender, ed., Plenum Press, New York, Vol. I.

Newton, M. and H.A. Holt. 1971. Scolytid and buprestid mortality in ponderosa pines injected with organic arsenicols. J. Econ. Entomol. 64: 952-958.

Newton, M. and L.A. Norris. 1968. Herbicide residues in blacktail deer from forest treated with 2,4,5-T and atrazine. Proc. West. Soc. Weed Sci. : 32-34, Boise, Idaho.

Norris, L.A. 1971. Chemical brush control: assessing the hazard. J. For. 69: 715-720.

Norris, L.A. 1967. Chemical brush control and herbicide residues in the forest environment. p. 103-123. In Herbicides and Vegetation Management, Symp. Proc. Oregon State Univ., Corvallis, Oregon.

Norris, L.A. 1971. Studies of the safety of organic arsenical herbicides as precommercial thinning agents: a progress report. Proc. Symp. Precommercial Thinning of Coastal and Intermountain Forests in the Pacific Northwest. Wash. State Univ., Pullman, Wa., p. 63-74.

Norris, L.A. and R.A. Miller, 1974. Toxicology of 2,3,7,8-tetrachlorodibenzodioxin (TCDD) in guppies (Poecilia reticulatus Peters). Bull. Environ. Contam. and Toxicol. 12: 76-80.

Otten, R.J., J.E. Dawson and M.M. Schreiber. 1957. The effects of several herbicides on nitrification in soil. Proc. 11th N.E. Weed Contr. Conf. 11: 120-127.

Palmer, J.S. and R.D. Radeleff. 1964. The toxicological effects of certain fungicides and herbicides on sheep and cattle. Ann. N.Y. Acad. Sci. 111: 729-736.

Panič, M. and W. Franke. 1971. (The influence of 2,4-D on the ascorbic acid content and the ascorbic acid oxidase activity in bean and wheat leaves after leaf application). Angew. Bot. 45: 33-45.

Peterson, R.L., G.R. Stephenson and B.J.F. Mitchell. 1974. Effects of picloram on shoot anatomy of red maple and white ash. Weed Res. 14: 227-229.

Quimby, D.C. 1966. A review of literature relating to the effects and possible effects of sagebrush control on certain game species in Montana. 46th Annu. Conf. W. Assoc. State Game & Fish Commissioner, Butte, Montana, July 12-14, 1966, 13 p.

Radeleff, R.D. 1958. The toxicity of insecticides and herbicides to livestock. Adv. Vet. Sci. 4: 265-276.

Rowe, S.J. 1961. Level-of-integration concept and ecology. Ecology 42: 420-427.

Rowe, V.K. 1951. Health hazards associated with the handling and use of herbicides. N.C. Weed Contr. Conf. Proc. 8: 90-94.

Rowe, V.K. and T.A. Hymas. 1954. Summary of toxicological information on 2,4-D and 2,4,5-T type herbicides and an evaluation of the hazards to livestock associated with their use. Amer. J. Vet. Res. 15: 622-629.

Rudd, R.L. and R.E. Genelly. 1956. Pesticides: their use and toxicity in relation to wildlife. Calif. Dept. Fish and Game. Game Bull. No. 7, 209 p.

Rodgers, C.A. and D.L. Stalling. 1972. Dynamics of an ester of 2,4-D in organs of three fish species. Weed Sci. 20: 101-105.

Schroeder, M.H. 1972. Relationships of range and forest management with herbicides to wildlife. Proc. West. Soc. Weed Sci. 25: 11-15.

Sharma, M.P. and W.H. vanden Born. 1972. Effects of picloram on growth and on chlorophyll, RNA, and protein content of plants. Can. J. Bot. 50: 2039-2044.

Shipman, R.D. 1971. Timber, wildlife improved with fenuron herbicide. Sci. Agr. 19: 2-3.

Smith, A.E. 1973. Influence of 2,4-D and 2,4,5-T on in vitro digestion of forage samples. J. Range Manage. 26: 272-274.

Smith, A.E. and R.E. Wilkinson. 1973. Influence of simazine and atrazine on free fatty acid content in isolated chloroplasts. J. Weed Sci. Soc. Amer. 21: 57-60.

Sparrow, A.H., R.C. Sparrow, K.H. Thompson and L.A. Schairer. 1965. The use of nuclear and chromosomal variables in determining and predicting radiosensitivities. Radiat. Bot. (Suppl.) 5: 101-132.

Stelzer, M.J. 1970. Mortality of Ips lecontei attracted to ponderosa pine trees killed with cacodylic acid. J. Econ. Entomol. 63: 956-959.

Sterrett, J.P., G.R. Leather, W.E. Tozer, W.D. Foster and D.T. Webb. 1974. Foliar abscission of woody plants with combinations of endothall and ethephon. Weed Sci. 22: 608-614.

Swanson, C.R. and W.C. Shaw. 1954. The effect of 2,4-dichlorophenoxyacetic acid on the hydrocyanic acid and nitrate content of Sudan grass. Agron. J. 46: 418-421.

Tarrant, R.F. and L.A. Norris. 1967. Residues of herbicides and diesel oil carriers in forest waters: a review. p. 94-102. In Herbicides and Vegetation Management, Symp. Proc. Oregon State Univ., Corvallis, Oregon.

Tarrant, R.F. and J. Allard. 1972. Arsenic levels in urine of forest workers applying silvicides. Arch. Environ. Health 24: 277-280.

Tu, C.M. and W.B. Bollen. 1969. Effect of tordon herbicides on microbial activities in three Willamette Valley soils. Down To Earth 25: 15-17.

Unrau, J. 1953. Cytogenetic effects of 2,4-D on cereals. Can. Seed Growers' Assoc. 1952-53 Ann. Rep.: 25-28.

Unrau, J. 1954. Cytogenetic effects of 2,4-D on cereals. Can. Seed Growers' Assoc. 1953-54 Ann. Rep.: 37-39.

Unrau, J. and E.N. Larter. 1951. Cytogenetical Responses of Cereals to 2,4-D. I. A study of meiosis of plants treated at various stages of growth. Can. J. Bot. 30: 22-27.

Vogel, F. and G. Rohrborn. 1970. Chemical Mutagenesis in Mammals and Man. Springer-Verlag, New York, 519 p.

Volynets, A.P. and V.N. Kornelynk. 1973. Effects of herbicides on the phenolic complex of plants. Soviet Plant Physiol. 20: 716-722.

Ward, A.L. 1973. Sagebrush control with herbicides has little effect on elk calving behavior. Rocky Mt. For. Range. Exp. Sta., U.S.D.A., For. Serv. Res. Note RM-240, 4 p.

Ware, G.W. and W.J. Brakel. 1963. Excretion of 3-Cl-aniline and isopropyl-n-(3-chlorophenyl) carbanate (CIPC) in the milk and urine in dairy cows fed CIPC. J. Dairy Sci. 46: 120-123.

Warren, L.E. 1967. Residues of herbicides and impact on uses by livestock. p. 227-242. In Herbicides and Vegetation Management. Symp. Proc., Oregon State Univ., Corvallis, Oregon.

White, J.A. and D.D. Hemphill. 1972. An ultrastructural study of the effects of 2,4-D on tobacco leaves. Weed Sci. 20: 478-481.

Wilbert, D.E. 1963. Some effects of chemical sagebrush control on elk distribution. J. Range Manage. 16: 74-78.

Wilson, J.G. 1973. Teratological potential of 2,4,5-T. Indust. Veg. Manage. 5: 10-13.

Woodford, E.K. and G.R. Sogar, eds. 1960. Herbicides in the Soil. Blackwell Sci. Publ., Oxford.

Wu, C.C. and T.T. Kozlowski. 1972. Some histological effects of direct contact of Pinus resinosa seeds and young seedling with 2,4,5-T. Weed Res. 12: 229-233.

Wuu, K.D. and W.F. Grant. 1966. Induced abnormal meiotic behavior in a barley plant (Hordeum vulgare L.) with the herbicide Lorax. Phyton 23: 63-67.

Wuu, K.D. and W.F. Grant. 1967a. Chromosomal aberrations induced by a plant growth retarding chemical (B-995) in barley (Hordeum vulgare). Bot. Bull. Academia Simica 8: 191-198.

Wuu, K.D. and W.F. Grant. 1967b. Chromosomal aberrations induced in somatic cells of Vicia faba by pesticides. The Nucleus 10: 37-46.

Wuu, K.D. and W.F. Grant. 1967c. Chromosomal aberrations induced by pesticides in the meiotic cells of barley. Cytologia 32: 31-41.

Zabel, R.A. and F.W. O'Neil. 1957. The toxicity of arsenical compounds to microorganisms. Tappi 40: 911-914.

Chapter 3 EFFECTS OF HERBICIDES ON THE STRUCTURE AND FUNCTIONING OF ECOSYSTEMS

Considering the level of concern over the environmental implications of herbicide use in forestry, there has been remarkably little research on the effects of such use on the structure and dynamics of ecosystems. In fact, there is very little information on the effects of herbicides on any biological systems more complex than the individual organism (c.f. Rowe 1961). Thus, while there are modest quantities of information on the genetic, physiological-biochemical, morphological, and toxicological effects of herbicides on a variety of types of organism, there is little direct information on the behavioral and other sub-lethal effects on individual organisms, on the integrated consequences of all these effects for populations and communities, and even less for ecosystem structure and functional processes.

While there is no great shortage of general commentaries on the potential ecological effects of herbicides, most of these are merely extrapolations of basic ecological theory, much of which itself has not yet stood the test of time and rigorous testing or which only holds true for certain types of ecological systems. In the virtual absence of data from experiments and studies specifically designed to elucidate the broad ecological effects of herbicides, the reviewer is obliged to integrate available knowledge concerning the simpler biological systems described in the preceding sections with existing theory concerning populations, communities and ecosystems. This must be done with full recognition that these conclusions may subsequently be proven to be erroneous to a greater or lesser extent.

Perhaps the fullest available review of the ecological effects of herbicides is House et al. (1967) and the reader is recommended to this paper. These authors note at the outset that our knowledge of the biological effects of man's manipulation of the environment is still so woefully inadequate as to make a completely accurate analysis of the broad ecological effects of herbicides impossible. Other good references on some aspects of the broad ecological implications of herbicides include Harper (1956, 1957), Orians et al. (1970), Boffey (1971), Tschirley (1969), Woodwell (1970), Geier and Clark (1960), Schacht and Hansen (1963), and Kuenen (1960).

An ecosystem can be defined structurally, as the total assemblage of living organisms together with their total non-living environment (Tansley 1935 - ecosystem; Sukachaev 1945 - biogeocoenose), or functionally as any unit that includes all of the organisms in any given area interacting with the physical environment so that a flow of energy leads to clearly defined trophic structure, biotic diversity and material cycles (Odum 1971).

An ecosystem is the product of biological evolution which has produced a group of living organisms that is capable, under the physical conditions of the particular environment, of transforming chemical or sunlight energy into a variety of types of organic biomass using inorganic chemicals from the soil and atmosphere. The two most fundamental processes in the ecosystem are: 1) the entry, storage, redistribution and loss of energy (ecological energetics) and 2) the accumulation and circulation of chemical nutrients (nutrient cycling or biogeochemistry) that are essential for the capture, storage and redistribution of energy. Ecological energetics and nutrient cycling involve a complex assemblage (community) of living organisms that is subject to change in physical structure and species composition over time (ecological succession). The discussion in this chapter will deal with the effects of herbicides on ecological energetics, biogeochemistry and ecological succession, and will conclude with some thoughts on the consequences of these effects for ecosystem integrity, diversity and stability. The discussion of basic ecological theory is not referenced, and the reader requiring more details of this theory is referred to such ecological texts as Odum (1971), Daubenmire (1968), Collier et al. (1973).

3.1 Effects on Ecological Energetics

Ecosystems are organized according to how a particular organism solves its constant need for energy (food). Almost all energy comes ultimately from the sun, being captured and stored by green plants. Plant-eating animals (herbivores) satisfy their energy needs by ingesting and metabolizing plant materials, while meat-eating animals (carnivores) utilize the energy stored in the bodies of herbivores. Some animals (omnivores, such as man) can obtain their energy from a mixed diet of plants and herbivores, while yet another type of organism (saprotrophs) obtains its energy from dead organic material derived from either plants or animals, or both. This sequence of food (trophic) interdependencies is referred to as a food chain, or more correctly, a food web.

It is generally recognized that the abundance and productivity of animal populations are ultimately related to the quantity of plant biomass being produced in the ecosystem. Anything that reduces the production of plants may therefore reduce the production of animals, assuming other factors to be favourable for animal production. Herbicides, being plant poisons, have a potentially great impact on ecological energetics. Where they are used to kill all living plants in an area, all food chains, with the exception of those based on dead organic material involving saprotrophs, will temporarily be dislocated.

Total erradication of vegetation by herbicides is not common in forest practice, forest nurseries being a possible exception. Herbicides are generally applied to remove specific components of the plant community (Barrons 1969) with the express purpose of favouring some other component of the plant community by temporarily relieving it of competition. Thus, the adverse impact of forest use of herbicides on ecological energetics (sunlight capture, storage and transfer) will be a function of the degree and duration of the reduction in the biomass of photosynthetic organs. The effect on herbivores, and thus on carnivores, will also depend upon the effect of the herbicide on the relative abundance, productivity and physical availability of the food plant species of each particular herbivore; it is a serious error to consider all plants as potential food for herbivores since many of them have very definite requirements in terms of species, palatability and nutrition. The impact on omnivores and carnivores will depend upon the impact on the herbivore, while the saprotrophs will generally be favoured by the increase in dead plant material available to them (e.g. Likens et al. 1969).

The top-killing of vegetation that is physically out of reach of ground-dwelling herbivores will generally lead to a short-term increase in available plant biomass (see Section 2.3), while the killing of deciduous shrubs to favour conifers may remove a physiologically available energy supply (because it is palatable and nutritious) and replace it with a physiologically unavailable energy supply (low palatability and nutritional status) of equal or greater productivity and biomass.

The effects of herbicides on ecological energetics are thus seen to be about as complex and hard to generalize as any of the other topics already reviewed. Because herbicides are generally used in forestry for vegetation manipulation rather than vegetation erradication, the overall ecosystem energetics (total energy flow) may not be greatly altered. However, specific pathways of energy flow, affecting the numbers and productivity of individual species of plants, herbivores, carnivores, omnivores and saprotrophs will be greatly affected as the particular plant species that directly or indirectly provide their energy (food) supply are either killed, suppressed or favoured by the herbicide.

The recent "energy crises" has served to focus attention on both future sources of energy, and also the energy costs of man's activities. The use of herbicides has not escaped examination. Herbicide manufacture, distribution and application all require large expenditures in fossil fuel energy, and there is growing concern that the energy requirements of alternative weed control practices may become a major factor in the decision between alternatives, and an important factor influencing the choice of herbicide, its formulation and method of application (Nalewaga 1974, Kottman 1974).

3.2 Effects on Biogeochemistry

The circulation of nutrient chemicals within an ecosystem and the inputs and outputs to and from that circulation are intimately related to the magnitude and pathways of energy flow. Thus, highly productive tropical forest ecosystems are associated with large quantities of rapidly cycling nutrients, while low productivity forests in northern, high elevation or dry-land areas are associated with the slow circulation of much smaller quantities of nutrients (Rodin and Bazilevich 1967).

Forest biogeochemistry involves a number of different nutrient cycles. The geological cycle involves inputs to the ecosystem from dust, precipitation, atmospheric gases (H_20, $C0_2$, N_2, and $S0_2$), weathering of soil minerals, soil seepage and biotic imports. It involves outputs in water, wind, soil erosion, and biotic exports. The biological cycle of nutrients involves exchanges between plants, animals, microbes and the soil within the ecosystem, while the biochemical cycle involves redistribution of nutrients within individual plants. There is also the so-called direct nutrient cycle which occurs between plants and soil microbes (mycorrhizal fungi).

Mature forests are frequently found growing moderately well on soils derived from very infertile soil parent material, and mature forests growing on a variety of soils varying greatly in their nutritional status may exhibit remarkably little variation in their species composition and productivity (see Section 3.3 below). These enigmas can be explained in part by the remarkable ability of forest ecosystems to conserve, accumulate and cycle the small but continuing inputs to the biological cycle from the geological cycle, until the forest biota become almost independent of the underlying mineral soil for the supply of certain nutrients. Anything that radically alters the species composition, the productivity or the living biomass of the forest will probably alter pathways and mechanisms of nutrient cycling. Thus, herbicides, with their ability to terminate nutrient uptake by a significant proportion of the plant community and to alter the species composition of the plant community have the potential to create significant changes in forest biogeochemistry. However, where herbicide treatment only removes a small proportion of the active plant biomass, the effects on biogeochemistry will be less dramatic.

Perhaps the best known example of the effects of herbicides on forest biogeochemistry is the Hubbard Brook watershed experiment (Likens et al. 1970). All vegetation on a watershed in New Hampshire, north-eastern U.S.A., supporting a mature northern hardwood forest was cut down to a height of 1 m, and all regrowth and minor vegetation was suppressed by applications of bromacil and 2,4,5-T. The termination of nutrient uptake by vegetation, the transfer of large quantities of highly decomposable plant debris to the saprotroph food chain, a marked increase in the activity of decomposer organisms (notably nitrifying organisms that convert leaf protein into nitrate ions (Likens et al. 1969)), and an increase in the quantity of water leaving the area in the stream resulted in a great increase in outputs to the geological cycle in the form of dissolved nutrients in Hubbard Brook.

The Hubbard Brook experiment was not representative of conventional forestry use of herbicides, and there have been no comparable experiments elsewhere which could throw some light on the question of whether or not conventional herbicide usage could cause similar changes in the inorganic chemistry of streamwater. Johnson and Swank (1973) reported on the effects on the cation content of streamwater of herbicidal (atrazine and paraquat followed by atrazine and 2,4-D) removal of grass cover on a steep Appalachian watershed that had been converted from forest cover eight years earlier. Data obtained three years after the first and one year after the last herbicide treatment showed increases in losses of about 65% for Ca and 100% for Mg as compared with streamwater from a watershed carrying mature hardwoods. However, the conclusions from this study are speculative, since no pretreatment data are available. Also, the study did not start for several years after the initial herbicide treatment, and the peak response, which would have been expected within the first 24 post-treatment months, may have been missed (Kimmins, unpublished data, Fredriksen et al. 1975).

It has been noted by many authors that herbicides influence the nutrition of plants. Sub-lethal levels of several herbicides increase plant metabolism, stimulating uptake of nitrate ions and other nutrients. Experiments have shown that the increased uptake is the result not only of reduced competition for nutrients and moisture but also of a stimulation of nutrient uptake and incorporation of nitrogen in protein. In some cases there is an increase in foliar nitrogen concentrations, but because of a reduction in dry weight production there is no net increase in nitrogen uptake. Increases in nutrient uptake have been demonstrated in certain species of trees, forbs and grasses following treatment of simazine, atrazine, paraquat, picloram, terbocil, 2,4-D and other herbicides. Apparently, the addition of low levels of certain herbicides can improve the efficiency of fertilizer use (Cooke 1957, Ries 1968, Ries et al. 1963, Conner and White 1968a, 1968b, Baur et al. 1970, Freyman 1970, and other references in Kimmins and Fraker 1973).

There seems to be little doubt that herbicide treatment of forest vegetation will have significant effects on forest biogeochemistry. This will involve changes in both the biological and geological cycles. The topic has received remarkably little attention and it would be premature to draw any firm conclusions. However, further research is needed because of the importance of minor vegetation in nutrient cycling in forested ecosystems and in nutrient retention after logging and slashburning. The elimination of weeds by herbicides may involve significant short-term nutrient losses to streams, with either beneficial or detrimental consequences for the recipient aquatic ecosystems.

3.3 Effects on Plant Community Structure and Ecological Succession

The structure of a plant community is a function of the soil, geology, topography, climate, the fire and wind history of the area, and the influence of animals. It is also a function of the degree of maturity of the ecosystem.

The maturity of an ecosystem depends upon the degree to which the biota has altered the climate near the ground and the surface geology of the area. That is, the degree to which it has created a soil and microclimate that reflect the long-term equilibrium between the climate, the vegetation and the geological character of the area. Following some event that removes the biotic community and the modifying influence that it has created on climate and geological materials, an area will be invaded by pioneer organisms. By their very presence, these organisms will alter the chemistry and microclimate of the ground/

atmosphere interface creating conditions that somewhat less hardy species (i.e. those unable to tolerate or compete successfully in the pioneer physical environment) are able to tolerate. These invade and eventually replace the pioneer community, but in so doing may create changes in the environment that lead to the invasion of yet another set of plants and animals that eventually replace them. In physically moderate or favorable environments, the early sequence of plant communities that successively occupy an area may simply represent differences in the efficiency of dispersal or speed of development of different plant species. In physically extreme environments, environmental amelioration will be necessary before one community succeeds another.

The process of successive replacement of one biotic community by another, over time, is referred to as ecological succession. Where the disturbance event removes all traces of previous biotic modification, the process is referred to as a primary succession. Where the disturbance leaves some of the biotic modification of the climate and/or geological materials intact, it is referred to as a secondary succession. The process continues until a biotic community is established that either is unable to create sufficient environmental change to permit its replacement by a succeeding community, or that represents the most competitive community existing in the region even though it continues to create slow changes in certain parameters of the ecosystem. This final community is referred to as the <u>climax</u> community while the entire series of communities from the initial pioneer community to the climax is referred to as the <u>sere</u>. Clearly identifiable communities of the sere are referred to as seral stages (see Kimmins 1973a, 1973b for a fuller discussion).

The term ecological succession is most frequently used to refer to processes occurring in the plant community. However, there is also a sequence of animal and microbial communities that accompany the vegetation changes. In some cases, the fauna and microflora are partially or even wholly responsible for the process of community replacement or for the determination of the vegetative composition of the climax community. However, more frequently the fauna and microflora are dependent on the vegetation for their food supply and habitat, and therefore change as the vegetative succession occurs.

With this brief resume of the topic of ecological succession, we can examine the effects of the use of herbicides in forestry on plant community structure and the maturity of ecosystems.

Herbicides in forestry may be used to either accelerate or retard succession. Herbicidal destruction of shrub and non-commercial hardwood species to improve or permit the growth of conifers merely accelerates a natural succession that would have occurred naturally in time. Destruction of non-palatable shrubs on overgrazed or eroded dry range land to establish communities of climax grass species also represents an acceleration of succession. Accompanying this vegetative succession will be a change in wildlife (see section 2.3) that will sometimes be desirable and sometimes undesirable in terms of wildlife productivity and species composition.

Top-killing of scrub hardwoods or mature shrubs to improve wildlife habitat and browse represents a deliberate reversal of succession to an earlier stage. Similarly, erradication of weeds in a forest nursery represents a reversion to a seral stage where there is freedom from competition. These uses of herbicides lead to secondary rather than primary successions since most of the biotic modification of microclimate and soils is deliberately retained.

Relatively little has been written on the effects of herbicides on succession. However, Egler (1948, 1950, 1953, 1958, 1964) and Bramble and Byrnes (1972) described the use of herbicides to create fairly stable shrub communities on power right-of-ways and appealed for the use of herbicides as an <u>aid</u> to successional manipulation rather than as a chemical bulldozer.

Turner (1969) and Thilenius <u>et</u> <u>al</u>. (1974) described the effects of 2,4-D applications on alpine plant communities. The graminoid:forb ratio of the vegetation on a Wyoming alpine meadow was altered from approximately 3.7 to 8.2 without appreciably changing total standing crop or its digestible dry matter content. Four years after treatment, resurgence of the forb population could not be detected. Grass production increased greatly on a mountain grassland in Colorado within a short time after competition from forbs and shrubs had been reduced by herbicides.

Schacht and Hansen (1963) reported on long-term vegetation changes in northern Minnesota following aerial applications of 2,4-D and 2,4,5-T. Reductions in tree and shrub canopies were accompanied by increase in the cover percent of low shrubs, herbaceous and grass-sedge communities.

Cox (1973) argued against the conversion of fire-climax chaparral communities in southwestern U.S. to earlier succesional but more fire resistant grassland. He noted that these earlier successional grass communities were less desirable than the native chaparral in several ways.

In concluding this section, it should be pointed out that a large part of man's energies (agriculture, forestry, and horticulture) are spent manipulating vegetative succession either to favour energy flow into desired species, of herbivores (e.g. cows or deer). Herbicides represent a valuable tool in this ubiquitous practice of successional manipulation which can be used to either accelerate or revert succession. They constitute a very powerful tool that can, if wrongly used, create a variety of undesirable effects, including an undesirable seral stage. However, successional manipulation without herbicides would be much more expensive (Green 1973).

3.4 Effects on Ecosystem Integrity, Diversity and Stability

One of the several contentious concepts in ecology over the past few years has been the relationship between ecosystem diversity and stability. The idea that the stability of an ecosystem increases linearly as the diversity of organisms in the system increases has not proven to be useful in a number of situations. However, in spite of shortcomings in the theory, it frequently does appear that as the variety of sizes and kinds of organisms, and the numbers of interrelationships and interdependencies between them increases, there is a concomitant increase in the ability of the system to resist being changed by some external disturbance factor.

Cox (1973) noted that herbicides reduce the diversity of the plant community when used to convert chaparral to grassland in southern California. Chaparral communities consist of many species of woody shrubs, annual grasses and other native annual plants. Herbicides and aerial seeding are used to replace this type of community with a mixture of three perennial grasses, but these are displaced by annual grasses if the area is too dry. Cox noted that the simplified community that results is more vulnerable to pest outbreaks, less efficient at conserving nutrients, less resistant to drought, and less resistant to erosion and land slippage because of shallower roots than the native chaparral vegetation. The grass community also supports a lower diversity of animals than chaparral.

An aspect of herbicide use that has received some attention in agriculture is the question of the role of herbicides in the evolution of weeds. Harper (1956, 1957) noted that herbicides can induce resistance in target populations by either natural selection or mutation. Weed populations exhibit remarkable overall stability which renders weed control particularly problematical. The characteristics of dormancy, efficient dispersion, and prolific production of seed contribute to this stability. Development of weed resistance in forest practice is far less likely than in agriculture because of the frequency of spraying. One or two sprays per rotation will obviously be much less effective in developing resistant strains than the annual or more frequent applications of agriculture.

The effects of herbicide use in forestry on diversity and stability are complex. For example, elimination of dense stands of monoculture brush (e.g. salmonberry or red alder) to replace them with monoculture Douglas-fir in coastal British Columbia probably has relatively little effect on floral diversity, while faunal diversity may decline. However, the stability of the subsequent population is unlikely to be any different from natural mature communities in the area, since the herbicides are generally only accelerating or reverting the natural process of succession. It is also debatable as to whether a monoculture conifer crop is more or less stable than a monoculture shrub community. Where the herbicide efficiently removes early seral vegetation in the absence of a well-established succeeding community, ecosystem stability will be temporarily reduced. However, the type of site on which herbicides would be used is characteristically re-invaded very rapidly, so that this reduction in diversity and stability would be short-lived, and after a delay there might in fact be a net increase in ecosystem diversity.

Herbicide use in forestry rarely removes all vegetation, since many of the plants will be unharmed, others will resprout from undamaged underground organs, and others will re-invade the area either from ungerminated seed stored in the forest floor or from seed distributed onto the area from nearby. Thus, while herbicides definitely have the potential to decrease ecosystem diversity and stability (c.f. references on herbicide usage in Vietnam cited earlier), it is not at all clear that conventional herbicide use in forestry will produce any changes in these parameters than would not occur naturally as the result of the process of succession.

References Cited in Chapter 3

Barrons, K.C. 1969. Some ecological benefits of woody plant control with herbicides. Science 165: 465-468.

Baur, J.R., R.W. Bovey and C.R. Benedict. 1970. Effect of picloram on growth and protein levels in herbaceous plants. Agron. J. 62: 627-630.

Boffey, P.M. 1971. Herbicides in Vietnam. AAAS study finds widespread devastation. Science 171: 43-47.

Bramble, W.C. and W.R. Byrnes. A long-term ecological study of game food and cover on a sprayed utility right-of-way. Agr. Exp. Sta., Purdue Univ., Res. Bul. No. 885.

Collier, B.C., G.W. Cox., A.W. Johnson and P.C. Miller. 1973. Dynamic Ecology. Prentice-Hall, New Jersey, 563 p.

Conner, B.J. and D.P. White. 1968a. Triazine herbicides and the nitrogen nutrition of conifers. Quart. Bull. 50: 497-503.

Conner, B.J. and D.P. White. 1968b. Triazine herbicides and the mineral nutrition of conifers. p. 193-204. In Tree Growth and Forest Soils. C.T. Youngberg and C.B. Bovey, eds. Proc. 3rd N. Amer. For. Soils Conf. Oregon State Univ. Press, Corvallis, Oregon.

Cooke, A.R. 1957. Influence of 2,4-D on the uptake of minerals from the soil. Weeds 5:25-28.

Cox, G.W. 1973. Herbicides, type conversion and chaparral ecosystem. Environ. Southwest. Aug./Sept. 1973: 4-6.

Daubenmire, R. 1968. Plant Communities: A Textbook of Plant Synecology. Harper & Row, N.Y., 300 p.

Egler, F.E. 1950. Herbicide effects on Connecticut vegetation. Bot. Gaz. 112: 76-85.

Egler, F.E. 1948. 2,4-D effects on Connecticut vegetation. Ecol. 29: 386-387.

Egler, F.E. 1953. Vegetation management for rights-of-way and roadsides. Smithsonian Inst. Ann. Rept. 1953: 299-320.

Egler, F.E. 1958. Science, industry and the abuse of rights-of-way. Science 127: 573-580.

Egler, F.E. 1964. Pesticides in our ecosystem: communication II. Bio-Sci. 14: 29-36.

Fredriksen, R.L., D.G. Moore, and L.A. Norris. 1975. The impact of timber harvest, fertilization, and herbicide treatment on streamwater quality in western Oregon and Washington. p. 283-313. In Forest Soils and Forest Land Management. B. Bernier and C.H. Winget, eds. Proc. 4th N. Amer. Forest Soils Conf., Les Presses de l'Universite Laval, Quebec.

Freyman, S. 1970. Chemical curing of pine grass with atrazine and paraquat. Can. J. Plant. Sci. 50: 195-197.

Green, M.B. 1973. Are herbicides too expensive? Weed Sci. 21: 374-378.

Geier, P.W. and L.R. Clark. 1960. An ecological approach to pest control. p. 10-18. In The Ecological Effects of Biological and Chemical Control of Undesirable Plants and Animals. Symp. Proc. 8th Tech. Meet. I.U.C.N.

Harper, J.L. 1956. The evolution of weeds in relation to resistance to herbicides. Proc. 3rd Brit. Weed Contr. Conf.: 174-188.

Harper, J.L. 1957. Ecological aspects of weed control. Outlook on Agr. 1: 197-205.

House, W.B., L.H. Goodson, H.M. Gadberry and K.W. Dockter. 1967. Assessment of ecological effects of extensive or repeated use of herbicides. Advanced Research Projects Agency No. 1086. MRI Project No. 3103-B, U.S. Dept. Commerce, Clearinghouse for Federal Scientific and Technical Information, 369 p.

Johnson, P.L. and W.T. Swank. 1973. Studies of cation budgets in the southern Appalachians on four experimental watersheds with contrasting vegetation. Ecology 54: 70-80.

Kottman, R.M. 1974. The energy crisis: problems and solutions. Proc. N.C. Weed Contr. Conf. 29: 14-18.

Kimmins, J.P. 1973a. The ecology of forestry: the ecological role of man, the forester, in forest ecosystems. For. Chron. 48: 301-307.

Kimmins, J.P. 1973b. Forest ecology: the biological basis for the management of renewable forest resources. For. Chron. 49: 25-30.

Kimmins, J.P. and P.N. Fraker. 1973. Bibliography of Herbicides in Forest Ecosystems. Environ. Canada, For. Serv. Infor. Rept. BC-X-81, 261 p.

Kuenen, D.J. 1960. General Introduction. p. 1-9. In The Ecological Effects of Biological and Chemical Control of Undesirable Plants and Animals. Symp. Proc. 8th Tech. Meet., I.U.C.N.

Likens, G.E., F.H. Bormann and N.M. Johnson. 1969. Nitrification: importance to nutrient losses from a cutover forested ecosystem. Science 163: 1205-1206.

Likens, G.E., F.H. Bormann, N.M. Johnson, D.W. Fisher and R.S. Pierce. 1970. Effects of forest cutting and herbicide treatment on nutrient budgets in the Hubbard Brook watershed-ecosystem. Ecol. Monogr. 40: 23-47.

Nalewaja, J.D. 1974. Energy requirements of various weed control practices. Proc. N.C. Weed Contr. Conf. 29: 18-23.

Odum, E.P. 1971. Fundamentals of Ecology, 3rd Edit., W.B. Saunders Co., Philadelphia, 574 p.

Orians, G.H. and E.W. Pfeiffer. 1970. Ecological effects of the war in Vietnam. Science 168: 544-554.

Ries, S.K. 1968. Spray-on protein boosters. Crops and Soils 20: 15-17.

Ries, S.K., R.P. Larson and A.L. Kenworthy. 1963. The apparent influence of simazine on nitrogen nutrition of peach and apple trees. Weeds 11: 270-273.

Rodin, L.E. and N.I. Bazilevich. 1967. Production and Nutrient Cycling in the Vegetation of the World. Oliver and Boyd, London, 288 p.

Rowe, J.S. 1961. Level-of-integration concept and ecology. Ecology 42: 420-427.

Schacht, A.J. and H.L. Hansen. 1963. Long-term vegetational changes following aerial applications of 2,4-D and 2,4,5-T in northern Minnesota. Unpubl. Rept., School of For., Univ. of Minnesota, 94 + XVI p.

Sukachaev, V.N. 1945. Biogeocoenology and phytocoenology. C.R. Acad. Sci., U.S.S.R. 47: 429-431.

Tansley, A.G. 1935. The use and abuse of vegetational concepts and terms. Ecol. 16: 284-307.

Thilenius, J.F., D.R. Smith and G.R. Brown. 1974. Effect of 2,4-D on composition and production of an alpine plant community in Wyoming. J. Range Manag. 27: 140-142.

Tschirley, F.H. 1969. Defoliation in Vietnam. Science 163: 779-786.

Turner, G.T. 1969. Responses of mountain grassland vegetation to gopher control, reduced grazing and herbicide. J. Range Manag. 22: 377-383.

Woodwell, G.M. 1970. Effects of pollution on the structure and physiology of ecosystems. Science 168: 429-433.

Chapter 4 BEHAVIOR AND FATE OF HERBICIDES IN FOREST ECOSYSTEMS

Herbicides can enter forest ecosystems by a number of routes. The most important of these is the deliberate application by aerial spraying or pelleting, ground spraying or pelleting, or by manual application to individual trees. Unintentional inputs result from drift of herbicides from adjacent agricultural or forest areas that are being sprayed, by volatilization and subsequent absorption by the vegetation or rain out of the vapours, or by water transportation in streams, overland flows or sub-surface seepage. Norris and Moore (1970) provided a summary of these inputs and the subsequent fate of the herbicides in forest ecosystems.

Once herbicides have entered the forest ecosystem, they may be absorbed by plants, ingested or absorbed by animals, enter the soil, get into water bodies, or be volatilized back to the atmosphere. In this chapter I will concentrate on the entry of herbicides into, and behavior in, soils and streams. Uptake and fate in plants and animals will be given only cursory treatment since these topics are either covered elsewhere or they are not central to the purpose of the review.

4.1 Uptake and Fate of Herbicides in Plants and Animals

4.11 Plants:

Uptake of herbicides by plants occurs chiefly via the leaves or the roots. The herbicidal action of the substance may occur at the site of uptake or may involve translocation to another part of the plant. Reviews of this topic include Currier and Dybing (1959), Foy (1964), Hacskaylo (1964), Hull (1967, 1970), van Overbeek (1956), Sargent (1965), McCready (1966), Franke (1967) and Norris (1974).

Uptake through the foliage is influenced by the character of the cuticle and the stomata, and the presence of hydathodes, lenticels and leaf hairs. Leaf age, metabolic condition, and leaf condition are also important. Herbicide formulation and surfactants modify the influence of these factors. Uptake is also related to the physiological condition of the plant, with uptake being greatest at times of maximum within-plant redistribution of materials. However, toxicity is apparently inversely related to the extent of internal movement of the herbicide because of dilution effects. Poor uptake can be offset by minimal internal movement since this permits internal accumulation of toxic concentrations of the herbicide. Uptake can also occur through stems and roots.

Once inside plants, herbicides may be phytotoxic at the site of entry, thus precluding any translocation. Other herbicides act more slowly or require translocation to a particular part of the plant before their phytoxicity is expressed. Some herbicides do not become phytotoxic until metabolized inside plants. Thus, some herbicides are fast-acting, contact herbicides while others act more slowly.

Many plants are able to degrade or detoxify particular herbicides (see review in Audus (1964) and Kearney and Kaufman (1969)) and therein lies the resistance of many plants to herbicides. Norris (1967) reviewed the role of detoxification in relation to uptake and translocation as a mechanism of plant resistance and herbicide selectivity.

4.12 Animals

Animals can take up herbicides by ingestion of contaminated food or water, by absorption through the skin, or by inhalation. The former will be the most important mechanism in terrestrial animals, while skin absorption will be equally important for aquatic organisms. Licking contaminated fur or preening contaminated feathers will also be a source of intake for appropriate types of organisms. The fate and elimination of herbicides in animals has already been reviewed above. Many animals have the ability to degrade or detoxify herbicides in their alimentary canal or internal tissues, and it appears that herbicides are normally eliminated promptly in faeces or urine. For further information, the reader should consult the literature cited in Section 2.3.

4.2 Fate of Herbicides in the Soil

The ecological effects of herbicides on biological systems are intimately related to the exposure of living organisms to the toxic substance. This in turn is influenced greatly by the movement and persistence of the chemical in soils since much of the herbicide applied finds its way into the soil soon after application. Where the chemicals are rapidly degraded to non-toxic substances, or where they are rapidly immobilized in the soil, exposure of terrestrial and aquatic organisms and the probability of adverse ecological effects of the herbicide are greatly reduced. The fate of herbicides in forest soils is thus of paramount importance in determining their ecological effects on the forest ecosystem. The discussion will be broken into three parts: adsorption, degradation, and leaching. No attempt will be made to cover in detail the substantial literature on this topic. Sample references will be given, and the reader is referred to various bibliographies for guidance in further reading (Anon. 1971a, 1971b, Kimmins and Fraker 1973).

The adsorption of herbicides by soils has been the subject of a large number of studies and is reasonably well understood (see reviews by Bailey and White (1964, 1970, Weber (1972), Weed and Weber (1974), Weber and Weed (1974), Mortland (1970), and Greenland (1965). Far more work has been done on agricultural soils than on forest soils, but the general principals derived from the agricultural studies apply equally to forest soils.

4.21 Adsorption:

Bailey and White (1970) noted that seven factors were known to influence the fate and behavior of pesticides in soil systems: (1) chemical decomposition, (2) photochemical decomposition, (3) microbial decomposition, (4) volatilization, (5) movement, (6) organism uptake, and (7) adsorption. The phenomenon of adsorption-desorption directly or indirectly influences the magnitude of the other six and therefore adsorption appears to be one of the major factors affecting the fate of herbicides in soils.

Adsorption of herbicides by the soil depends upon the chemical nature of the herbicide and the type of soil. Cationic herbicides such as paraquat and diquat are strongly adsorbed by inorganic cation exchange sites and to a lesser degree by organic exchange sites. Basic compounds such as atrazine can protonate to become positively charged at certain pH's and thus become adsorbed by both inorganic and organic cation exchange sites. They can also be held by hydrogen and hydrophobic bonding. Acidic compounds such as the phenoxy acetic acids and picloram may ionize to produce organic anions which are readily adsorbed by anion exchange sites. They are weakly adsorbed by clays and organic matter, but strongly adsorbed by hydrated iron and aluminum oxides. Thus, it might be expected that organic matter would be of less importance in the adsorption of acidic herbicides than in the adsorption of cationic and basic herbicides. However, it has been widely observed that the movement and persistence of phenoxy and picolinic acid herbicides are closely related to soil organic matter content. This is because the mechanism of herbicide adsorption in organic soils appears to involve physical and hydrogen bonding in addition to any ionic bonding that occurs.

The solubility of a herbicide in soil solution also plays an important role in its adsorption. The relationship is not a simple one, however. Within any one family of herbicides, adsorption may increase with water solubility, but adsorption of chemically different herbicides of a given solubility may exhibit little similarity.

Soils that have a high cation exchange capacity (associated with high levels of organic material and/or with vermiculite and montmorillonite clays) are characteristically associated with strong adsorption of herbicides while those low in organic material, with little or no clay, or with mainly kaolinite clay tend to exhibit weaker adsorption. There is generally more adsorption in acidic soils than neutral soils, although this varies between herbicides.

Forest soils in the major coniferous forests of the world are generally acidic, have a capping of high cation exchange capacity organic material, and have a moderate to high anion exchange capacity associated with the hydrated iron and aluminum oxides of the podzolic Bf soil horizon. Norris (1970a) reported on the adsorption - desorption kinetics of 2,4-D, 2,4,5-T, picloram and amitrole in forest floor material from a red alder stand, buffered to pH 6.5. At equilibrium, 34% of the 2,4-D in solution was adsorbed, compared to figures of 61% for 2,4,5-T, 27% for picloram, and 72% for amitrole, respectively. The adsorption-desorption kinetics for 2,4-D and 2,4,5-T, were similar. Suffling _et al._ (1974) reported that very little picloram and 2,4-D were leached from a forest podsol during rainstorms and concluded that the 0-10 cm organic layer and the acidity (pH 3.5-6.0) of the surface soil contributed to adsorption and retention of nearly all the herbicides applied.

Thus, it appears that in forest soils with appreciable organic matter, low acidity or high montmorillonite clay content, adsorption of herbicides is likely to be rather complete. On the other hand, coarse soils with low organic matter and clay content are less efficient at adsorbing herbicides and greater leaching through the soils is to be expected.

4.22 Degradation:

Herbicide degradation in soils has been demonstrated under a wide variety of conditions and a good summary is given in Kearney and Kaufman (1969). Decomposition may be chemical, biological or photochemical, depending upon the soil and the herbicide with the former two being the most important. Phenoxyacetic acid herbicides are degraded primarily by soil microbes which are sometimes able to utilize the herbicide as their sole carbon and energy source. Degradation is accelerated by warm, moist conditions in the presence of organic material, and the rate of degradation appears to be correlated with the abundance of aerobic bacteria. Other herbicides are degraded by a combination of microbial and chemical mechanisms, or by the latter alone.

Microbial degradation typically occurs after a lag whose length varies according to the herbicide; for example, the lag is relatively short for 2,4-D, long for MCPA, and very long for 2,4,5-T. Subsequent additions of herbicide to the adapted microbial population result in degradation without a further lag period. This period may represent either the time taken for mutation to a form capable of degrading the herbicide, or the time required for development of appropriate enzyme systems by existing microbial forms; current information apparently supports the latter theory (Kearney and Kaufman 1969).

The rate of microbial degradation of the phenoxyacetic acids is variable. Persistence of 2,4-D under field conditions was reported to be from less than 10 days to more than 14 weeks under various conditions of soil, precipitation, temperature, application rate and formulation (Ogle and Warren 1954). Hemmett and Faust (1969) and Pimentel (1971) reported that under favorable environmental conditions and with an acclimated microflora, 2,4-D may be degraded in 12-14 days. Under less favorable conditions, such as dry soils, poorly oxygenated lake sediments or soils without acclimated microflora, herbicides may persist for many months. Norris (1970b) reported that the rate of 2,4-D degradation was very similar in forest floor material from several different shrub, deciduous and coniferous stands, and concluded that degradation is probably more affected by microsite than litter type. Formulation of the herbicide influenced degradation, with 2,4-D acid being degraded faster than the ester or amine salt. Degradation was not influenced by diesel oil, and was accelerated by the presence of DDT. Comparative studies of 2,4-D, 2,4,5-T, picloram and amitrole showed that under the particular experimental conditions used, 2,4-D and amitrole both decomposed rapidly, with 6% and 20%, respectively, remaining after 35 days. 2,4,5-T required 120 days to degrade to 13% of the original and 65% of the initial picloram remained after 180 days; 2,4,5-T degradation approached 90% after four months, while picloram degradation accelerated after 120 days, suggesting a lag period for microbial adaptation. Sterilization experiments implicated microbial degradation for 2,4-D and 2,4,5-T, while amitrole degraded at the same rate in both sterile and normal forest floor material. The conclusion that amitrole is degraded in soil by non-biological reactions is supported by references in Kearney and Kaufman (1969). Bachelard and Johnson (1969) studied the persistence of 2,4,5-T and Tordon 50D (picloram and 2,4-D) by bioassay. Emergence of Pinus radiata seedlings was affected less than survival which was reduced for two months post-treatment by 2,4,5-T, while the effect of Tordon lasted for over three months.

Gzhegotskii and Moroz (1970) stated that urea, triazine and aliphatic herbicides may retain their toxicity in the soil for between a few months and four years or more, while reports in the literature indicate preservation in the soil of intermediate products of decomposition for 4-8 years. The depth of penetration is very important, with markedly reduced degradation of herbicides leached deep into the soil where microbial activity is negligible.

Degradation of herbicides by light was reviewed by Crosby and Li (1969) who note that most of the research on this topic has been conducted under laboratory conditions. Photolysis of phenoxyacetic acids occurs rapidly in aqueous solutions but only slowly in the absence of water. Picloram is also rapidly photolyzed by UV or sunlight irradiation in aqueous solutions. This could be a significant source of herbicide degradation in areas of high sunlight intensity. However, proof from field experiments that photolysis is important is largely lacking because of the difficulty of excluding the effects of volatilization and microbial decomposition in such experiments, and many authors feel that the UV energy of sunlight is too low in most areas to result in appreciable photolysis.

4.23 Leaching:

Leaching of herbicides in soil involves many of the same factors as adsorption in soil. Water solubility, nature of the soil colloids, herbicide dissociation constant, chemical character of the herbicide, and soil pH have all been shown to be important. Soil physical properties and climate also exert an important influence since these control the movement of water through the soil. The character of soil water movement has an important role in leaching as it affects both the equilibrium between the adsorbed herbicide and the soil solution and the exposure of soil solution to adsorbing surfaces. The timing of precipitation events after the herbicide application will also influence leaching since this will determine the proportion of the herbicide that has been adsorbed by soil colloids and therefore the ease with which leaching will occur. It may take several days for an adsorption equilibrium to be established.

Suffling et al. (1974) noted that there is general agreement that picloram leaches readily in many soils but warned that the many studies that have demonstrated this should not be used to imply that it will leach readily from **all** soils. They cited studies that found greatly reduced leaching from organic soils, heavy soils and at low soil pH values. As already noted above, they detected exceedingly little leaching of picloram and 2,4-D from an acidic podzol, although their study did not account for vertical leaching of the herbicides within the profile.

Gzhegotskii and Moroz (1970) noted that the depth to which herbicides leach affects their persistence because of reduced microbial activity at depth in soil. They noted that the majority of chlorine-containing herbicides, such as 2,4-D, are very readily eluted from all soils, especially sandy and organic ones. Harris (1967) determined the relative mobility of herbicides in soil columns. Herbicides in general are more mobile than insecticides, and the phenoxy and picloram herbicides are the most mobile. However, even these materials move only short distances in soil under normal conditions.

Bailey (1966) noted that of the two main routes by which herbicides enter water courses in agricultural areas, overland flow is thought to account for much more than soil leaching and sub-surface flow. Soil leaching is only serious on light-textured soils lacking in organic material and it has frequently been found that leaching drops dramatically as soon as organic matter is added to the soil. This appears to be somewhat in contrast to Gzhegotskii and Moroz (1970) but in agreement with Stevenson (1972) who felt that organic matter content is the soil factor most directly related to herbicide behavior in soil.

In summary, it appears that soil leaching is unlikely to be a major problem in many forest soils because of the presence of an organic forest floor, a high cation exchange capacity, a moderate to high anion exchange capacity, and low pH. On the other hand, characteristically low % base saturation of many coniferous forest soils shows that leaching can and does occur while the high rainfall characteristic of many forest areas creates the potential for leaching. However, rainfall intensity is also important. Low intensity rain in areas of high rainfall will result in less leaching than high intensity rain in areas of low rainfall because of the greater importance of surface runoff in the latter situation. The transportation of herbicides by overland flow and erosion of contaminated soil particles seems to be a more serious problem than vertical soil leaching although the number of field lysimeter studies in forest soils that can substantiate this point are very limited. A final question arises because of the accumulating evidence that much of the water flow in forest soils may be rapid flow from the surface to ground water along macropores, stems and old root channels (Feller 1975). This leads surface waters rapidly past the soils major exchange sites and could have an important influence on the leaching process.

4.3 The Entry and Fate of Herbicides in Aquatic Ecosystems

Forest soils generally represent a reasonably "safe" repository for most of the commonly used organic herbicides. The presence of abundant organic material, appreciable cation and anion exchange capacities, active microbial decomposition and low pH result in the rapid adsorption and subsequent degradation of these management chemicals. It would thus appear that the danger of herbicides getting into the aquatic environment from conventional herbicide usage would be minimal. Unfortunately, this is not always the case, and there has been growing concern over the past decade over the increasing entry of herbicides and other pollutants into forest streams (Tarrant 1966, Bailey 1966, Tarrant and Norris 1967).

Norris (1967) has reported on numerous studies of stream contamination by herbicides in Oregon. He noted that 60-75% of 2,4,5-T applied as low volatile esters in diesel oil in one study never reached the ground. Other studies have suggested up to 80% loss, and the drift of airborne herbicides away from the target area can lead to direct deposition onto waterbodies. Volatilization of herbicides can also result in transfer to non-target areas (Free 1965). In many forest spray operations it is impossible to avoid spraying directly over small watercourses, and herbicide contaminated vegetation or rainwater dripping from contaminated foliage frequently finds its way into streams. Overland flow of water carries contaminated soil particles into streams, and a high water table may prevent exposure of the herbicide to the adsorptive capacity of the soil. Surface runoff resulting from the first heavy post-spray rainfall can carry appreciable amounts of herbicide into streams (Norris 1969). Thus, it is common to find herbicide residues in nearby streams and lakes following herbicide applications.

Douglass et al. (1969) reported atrazine concentrations of up to 30 ppb thirty days after spraying grass in an Appalachian watershed with 2,4-D and atrazine: grass in the stream course itself was sprayed. A second spray which left a 10 ft. buffer strip of unsprayed vegetation resulted in no detectable increase in stream contamination which had dropped to about 3 ppb five weeks after the first spray.

Bovey et al. (1974) sprayed 2,4,5-T and picloram onto rangeland at six-month intervals over 2½ years and monitored herbicide residues in runoff water. Herbicide levels in the soil were low (0-238 ppb). Grass showed high residue levels shortly after treatment (50-70 ppm) but these declined rapidly. Runoff concentrations were high (400-800 ppb) if heavy rain occurred soon after treatment, but were low (less than 5 ppb) when rainfall did not occur for a month after spraying. Bovey and Scifres (1971) and Scifres et al. (1971) presented useful reviews of the movement and fate of picloram in grassland ecosystems, including movement to and persistence in water bodies.

Haas et al. (1974) detected picloram at concentrations of 55-184 ppb in surface runoff water leaving a sprayed Texas grassland. Surface runoff concentrations declined with time and no residues were found in the stream 0.8 km below the 32 ha treated area. Decline in picloram concentrations in a treated pond occurred at an initial rate of 14-18% per day but decreased to a rate of less than 1% per day after 100 days when concentrations were below 5 ppb. Most of the reduction was attributed to dilution from incoming water.

Concentrations of herbicides in streams typically drop off rapidly downstream from the treated area. Baur et al. (1972) showed how concentrations of picloram varied at various distances downstream during a 7-day storm immediately after application. From a peak concentration of 90 ppb just below the treated area, peak concentrations fell to about 13 ppb at 1.2 km, and 0.4 ppb at 1.6 km. Concentrations of less than 1 ppb were occasionally detected 1.6 km downstream 8 months after treatment.

Glass and Edwards (1974) compared concentrations of picloram in surface runoff and percolation water in silty-loam soil occupied by grass and broadleaf weeds. They found a year's delay before detectable levels of picloram reached a depth of 2.4 m. Concentrations in percolating water at this depth were maintained at 1-2 ppb for about 5 months, and were still at 0.5 ppb two years after initial treatment. Surface runoff showed peak concentrations of 14.5 ppb a month after treatment, but this declined to 6 ppb after about four months and dropped to zero after 11 months. Their results support the opinion that surface runoff is more important in transferring herbicides from soil to water bodies than percolating water, but shows that contamination of the latter is apparently more persistent than the former.

Davis and Ingebo (1973) treated a chaparral watershed in Arizona with pelleted picloram and monitored concentrations in streamwater leaving the area. Peak concentrations of 350-370 ppb occurred during the first three months after treatment in association with heavy rainfall. Residues fell to zero 14 months and 40 inches of rain after treatment; it was estimated that 4.5% of the picloram applied was lost to the stream. Fenuron applied to the same watershed (Davis and Ingebo 1970) yielded peak concentrations of 430 ppb 33 days after treatment following heavy rain. Concentrations over the first year ranged from 60-280 ppb, the higher concentrations being associated with heavy rain. 2.4% of the applied fenuron left the area in the stream.

All of these studies have come from relatively dry or non-forested ecosystems. In contrast, Suffling et al. (1974) reported that less than 1% of the picloram and less than 0.1% of the 2,4-D added to a forest podzol during rainstorms left the area in drainage water. Aldous (1967) reported results of spraying 2,4-D on Calluna vulgaris growing on deep acid peat. The peat was saturated at the time of spraying and heavy rainfall occurred for several weeks following treatment. The sprayed area was drained by numerous ditches that were directly exposed to the spray. Concentrations of 2,4-D between 1.5 and 2.0 ppm were detected in water draining out of the ditches for 7 days after spraying. A sample at 28 days after spraying yielded no detectable concentrations of 2,4-D.

Norris (1967) presented a summary of his studies of herbicide concentrations in streamwater following spray operations on Oregon forest lands. His results showed that the degree of stream contamination is proportional to the percentage of the watershed that is sprayed, that concentrations seldom exceed 0.5 ppm and that they usually decline to a few parts per billion within hours of the termination of spraying, that concentrations decline rapidly with distance downstream, and that the attainment of peak concentrations downstream is delayed for longer than can be explained by the time it takes for the contaminated water to reach the sampling spot. These results are explained in terms of rapid adsorption of the herbicides by stream colloids and little or no movement of herbicides to the streams after the spray operation. It is possible that higher concentrations would have been observed had heavy rain occurred shortly after spraying, but Norris (1968) reported that heavy fall rains failed to induce any detectable stream contamination in areas sprayed with 2,4-D and 2,4,5-T in the spring or early summer. He concluded that the major cause of stream contamination is direct application, drift, or surface runoff resulting from heavy rain immediately after spraying. However, in another study, Norris (1969) reported that

2,4-D and picloram applied to an area in late July and again in mid-August resulted in concentrations in runoff of 825 ppb 2,4-D and 78 ppb picloram 23 days after the last spray, with values of 250 and 38 ppb, respectively, after a further 19 days. Levels dropped to 1 ppb for both herbicides a little over two months and 11 inches of precipitation after spraying.

Reigner et al. (1968) reported on stream contamination with 2,4,5-T following deliberate spraying of streamside deciduous woody vegetation. Peak concentrations of 40 ppb were detected immediately after spraying, but this dropped to 10-20 ppb within four hours. No detectable concentrations (based on odor test with a reported 0.3-0.5 ppb sensitivity) were observed the following day or thereafter except following the first large rainstorm when concentrations of 10-20 ppb were detected.

Aly and Faust (1964) discussed the physical, chemical and biological factors that might influence the persistence of herbicides in natural waters. They reported that the sodium salt of 2,4-D persisted up to 120 days in lake water aerobically incubated in the laboratory but it is probable that biotic activity in a natural lake would have greatly reduced this persistence (e.g. Valentine and Bingham 1974). This idea is supported by Robson (1966) who found that water from a eutrophic, stagnant duck pond degraded 2,4-D from about 5.0 ppm to less than 0.5 ppm in about 13 days, while previously treated water required only 6 days. 2,4-D disappeared completely from untreated lake mud in 65 days, and from lake mud previously treated with 2,4-D in 35 days. By repeatedly treating with increasing concentrations of 2,4-D, an adapted population of microorganisms was obtained that could reduce 2,4-D by 81-85% within 24 hours (Aly and Faust 1964). These authors state that although ultraviolet decomposition of 2,4-D occurs in the laboratory, sunlight has insufficient UV energy to make this an important mechanism of loss in the field.

In summarizing this section, it would appear that forest herbicide sprays frequently result in contamination of streams, but that such contamination is at a low level and rapidly disappears because of adsorption onto colloidal exchange sites in the stream and subsequent degradation. Downstream contamination is a minor problem because of the rapid decline in concentrations with distance downstream from the sprayed area. However, it is likely to be more severe in those types of forest stream that have very pure water and little organic or fine inorganic particulate matter in the streambed. Contamination results chiefly from direct application to the water, from drift and volatilization problems, and from surface runoff accompanying heavy precipitation shortly after spraying. Contamination can be minimized by spraying at the beginning of a dry spell, under conditions (and using formulations and application techniques) that minimize drift and volatilization, and by leaving a buffer of unsprayed vegetation. Contamination will be more likely in topographic and climatic conditions that favour surface runoff rather than infiltration, and in soils that do not give prompt and efficient adsorption of the herbicides.

Aquatic environments appear to be generally well buffered against herbicides contamination, with prompt decreases in dissolved concentrations: repeated contamination is likely to speed the rate of degradation, but repeated applications of herbicides is unlikely in most forest usage. However, in spite of these favourable indications, our very incomplete knowledge of the sub-lethal effects of herbicide traces on aquatic organisms requires that until we have more information we should consider all herbicide contamination of forest waters undesirable and work to eliminate or minimize it. Recent work on the toxicity of dioxin to aquatic organisms justifies concern over herbicide residues in forest streams (see Section 2.5).

References Cited in Chapter 4

Aldhous, J.R. 1967. 2,4-D residues in water following aerial spraying in a Scottish forest. Weed Res. 7: 239-241.

Aly, O.M. and S.D. Faust. 1964. Studies on the fate of 2,4-D and ester derivatives in natural surface waters. Agr. Food Chem. 12: 541-546.

Anon. 1971a. Bibliography on persistence and decomposition in soils and plants (1968-1967). Comm. Bur. Soils., Harpenden, England. 19 p. 80 refs.

Anon. 1971b. Bibliography on persistence and decomposition in soils and plants (1971-69). Comm. Bur. Soils., Harpenden, England. 27 p. 107 refs.

Audus, L.J. ed. 1964. The Physiology and Biochemistry of Herbicides. Academic Press, N.Y. 555 p.

Bachelard, E.P. and M.E. Johnson. 1969. A study of the persistence of herbicides in soil. Australian Forestry 33: 19-24.

Bailey, G.W. 1970. Factors influencing the adsorption, desorption, and movement of pesticides in soil. Residue Rev. 32: 29-92.

Bailey, G.W. 1966. Entry of biocides into water courses. Proc. Symp. Agric. Waste Waters, Water Resources Center, Univ. Calif., Davis. Rept. No. 10: 94-103.

Bailey, G.W. and J.L. White. 1964. Review of adsorption and desorption of organic pesticides by soil colloids, with implications concerning pesticide bioactivity. J. Agr. Food Chem. 12: 324-332.

Baur, J.R., R.W. Bovey and M.G. Merkle. 1972. Concentration of picloram in runoff water. Weed Sci. 20: 309-313.

Bovey, R.W. and C.J. Scifres. 1971. Residual characteristics of picloram in grassland ecosystems. Texas Agric. Exp. Sta., Texas A & M Univ., College Sta., Texas. Rept. B-1111, 24 p.

Bovey, R.W., E. Burnett, C. Richardson et al. 1974. Occurence of 2,4,5-T and picloram in surface runoff water in the blacklands of Texas. J. Environ. Qual. 3: 61-64.

Crosby, D.G. and M. Li. 1969. Herbicide photodecomposition. Chapter 12, In Degradation of Herbicides, P.C. Kearney and D.D. Kaufman, eds., Marcel Dekker, N.Y.

Currier, H.B. and C.D. Dybing. 1959. Foliar penetration of herbicides - review and present status. Weeds 7: 195-213.

Davis, E.A. and P.A. Ingebo. 1970. Fenuron contamination of stream water from a chaparral watershed in Arizona. Res. Prog. Rept., Western Soc. Weed Sci., p. 22-23.

Davis, E.A. and P.A. Ingebo. 1973. Picloram movement from a chaparral watershed. Water Resources Res. 9: 1304-1313.

Davis, E.A., P.A. Ingebo and C.P. Pose. 1968. Effect of watershed treatment with picloram on water quality. Rocky Mt. For. and Range Exp. Sta., U.S.D.A., For. Serv. Res. Note RM-100, 4 p.

Douglass, J.E., D.R. Cochrane, G.W. Bailey et al. 1969. Low herbicide concentration found in streamflow after a grass cover is killed. S.E. For. Exp. Sta., U.S.D.A., For. Serv. Res. Note SE-108, 3 p.

Feller, M.C. 1975. Initial effects of clearcutting on the flow of chemicals through a forest watershed ecosystem in southwestern British Columbia. Ph.D. Thesis, Univ. of B.C., Vancouver, B.C.

Foy, C.L. 1964. Review of herbicide penetration through plant surfaces. J. Agr. Food. Chem. 12: 473-476.

Franke, W. 1967. Mechanisms of foliar penetration of solutions. Annu. Rev. Plant Physiol. 18: 281-300.

Freed, V.H. 1965. Chemicals and the control of plants. p. 159-171. In Research in Pesticides, C.O. Chichester, ed., Academic Press, N.Y.

Glass, B.L. and W.M. Edwards. 1974. Picloram in lysimeter runoff and percolation water. Bull. Environ. Contam. Toxicol. 11: 109-112.

Greenland, D.J. 1965. Interactions between clays and organic compounds in soils. 1. Mechanisms of interaction between clays and defined organic compounds. Soils and Fert. 28: 415-425.

Gzhegotskii, M.I. and A.M. Moroz. 1970. Accumulation, circulation and stability of herbicides in the external environment. Hyg. Sanit. 35: 251-255.

Harris, C.I. 1967. Movement of herbicides in soils. Weeds 15: 214-216.

Hackskaylo, J., ed. 1964. Absorption and translocation of organic substances by plants. 7th Ann. Symp. Amer. Soc. Plant Physiologists. S. Sec.

Hemmett, R.B., Jr. and S.D. Faust. 1969. Biodegradation kinetics of 2,4-dichlorophenoxyacetic acid by aquatic organisms. Residue Rev. 29: 191-207.

Hull, H.M. 1967. Uptake and movement of herbicides in plants. p. 49-55. In Herbicides and Vegetation Management. Proc. Symp. Oregon State Univ., Corvallis, Oregon.

Hull, H.M. 1970. Leaf structure as related to absorption of pesticides and other compounds. Residue Rev. 31, 155 p.

Kearney, P.C. and D.D. Kaufman, eds. 1969. Degradation of Herbicides. Marcel Dekker, N.Y., 394 p.

Kimmins, J.P. and P.N. Fraker. 1973. Bibliography of Herbicides in Forest Ecosystems. Environ. Canada, Forestry Service, Info. Rept. No. BC-X-81, 261 p., 1614 refs.

McCready, C.C. 1966. Translocation of growth regulators. Annu. Rev. Plant Physiol. 17: 283-294.

Mortland, M.M. 1970. Clay-organic complexes and interactions. Adv. Agron. 22: 75-117.

Norris, L.A. 1967. The physiological and biochemical bases of selective herbicide action. p. 56-66. In Herbicides and Vegetation Management, Symp. Proc. Oregon State Univ., Corvallis, Oregon.

Norris, L.A. 1967. Chemical brush control and herbicide residues in the forest environment. p. 103-123. In Herbicides and Vegetation Management, Symp. Proc. Oregon State Univ., Corvallis, Oregon.

Norris, L.A. 1968. Stream contamination by herbicides after fall rains on forest land. Res. Prog. Rept. West. Soc. Weed Sci.: 33-34.

Norris, L.A. 1969. Herbicide runoff from forest lands sprayed in summer. Res. Prog. Rept., West. Soc. Weed Sci.: 24-26.

Norris, L.A. 1970a. The kinetics of adsorption and desorption of 2,4-D and 2,4,5-T, picloram and amitrole on forest floor material. Res. Prog. Rept. West. Soc. Weed. Sci.: 102-105.

Norris, L.A. 1970b. Degradation of herbicides in the forest floor. p. 397-411. In Tree Growth and Forest Soils. C.T. Youngberg and C.B. Davey, eds., Oregon State Univ. Press, Corvallis, Oregon.

Norris, L.A. 1974. Behaviour of pesticides in plants. P.N.W. For. Exp. Sta. U.S.D.A., For. Serv. Gen. Tech. Rept. PNW-19, 6 p.

Norris, L.A. and D.G. Moore. 1970. The entry and fate of forest chemicals in streams. p. 138-158. In Proc. Symp. For. Land Uses and Stream Environ. Oregon State Univ., Corvallis, Oregon.

Ogle, R.E. and G.F. Warren. 1954. Fate and activity of herbicides in soils. Weeds 3: 257-273.

Pimental, D. 1971. Ecological effects of pesticides on non-target species. U.S. Off. Sci. Tech., 220 p.

Reigner, I.C., W.E. Sopper and R.R. Johnson. 1968. Will the use of 2,4,5-T to control streamside vegetation contaminate public water supplies? J. For. 66: 914-918.

Robson, T.O. 1966. Some studies of the persistence of 2,4-D in natural surface waters in Britain. Proc. 8th Br. Weed Contr. Conf.: 594-597.

Sargent, A.J. 1965. Foliar adsorption of growth regulators. Annu. Rev. Plant Physiol. 16: 1-12.

Scifres, C.J., R.R. Hahn, J. Diaz-Colon and M.G. Merkle. 1971. Picloram persistence in semi-arid rangeland soils and water. Weed Sci. 19: 381-384.

Stevenson, F.J. 1972. Organic matter reactions involving herbicides in soil. J. Environ. Qual. 1: 333-343.

Suffling, R., D.W. Smith and G. Sirons. 1974. Lateral loss of picloram and 2,4-D from a forest podsol during rainstorms. Weed Res. 14: 301-304.

Tarrant, R.F. 1966. Pesticides in forest waters - symptom of a growing problem. Soc. Amer. For. Proc., Div. Watershed Mgmt.: 159-163.

Tarrant, R.F. and L.A. Norris. 1967. Residues of herbicides and diesel oil carriers in forest waters: a review. p. 94-102. In Herbicide and Vegetation Management, Symp. Proc., Oregon State Univ., Corvallis, Oregon.

Valentine, J.P. and S.W. Bingham. 1974. Influence of several algae on 2,4-D residues in water. Weed Sci. 22: 358-363.

van Overbeek, J. 1956. Absorption and translocation of plant regulators. Annu. Rev. Plant Physiol. 7: 355-372.

Weber, J.B. 1972. Interactions of organic pesticides with particulate matter in aquatic and soil systems. p. 55-120. In R.F. Gould (ed.). Fate of Organic Pesticides in the Aquatic Environment. Amer. Chem. Soc., Wa., D.C.

Weber, J.B. and S.B. Weed. 1974. Effects of soil on the biological activity of pesticides. Chapter 10, In Pesticides and Soil Water. W.D. Guenzie, ed., Soil Sci. Soc. Amer., Madison, Wisc.

Weed, S.B. and J.B. Weber. 1974. Pesticide-organic matter interactions. Chapter 3, In Pesticides in Soil and Water. Agron. Soc. Amer., Madison, Wisc.

Chapter 5 FUTURE USE OF HERBICIDES IN FORESTRY AND RESEARCH NEEDS

This chapter will summarize the major environmental effects of herbicides as they are generally used in conventional forest practice, and suggest information gaps that require further research. It will also present some thoughts on how undesirable effects of herbicide use might be ameliorated. In spite of the substantial amount of information on the effects of herbicides, some of which is reviewed above, much of our knowledge is derived from laboratory studies, and our knowledge of what happens in a field situation is generally scant and very inadequate for the purposes of making rational decisions on the use of herbicides.

5.1 Summary of the Important Adverse Effects of Herbicides on Future Wood Production and Other Forest Land Values

The major adverse effects of herbicide use in forestry can be summarized as 1) toxicological effects on non-target organisms, and 2) undesirable changes in the biotic composition and functional processes of forest ecosystems.

Toxicological effects of herbicides on non-target organisms depend greatly on exposure and the toxicology of the herbicide to the recipient organism. Terrestrial organisms generally appear to show greater tolerance than aquatic organisms, and severe acute and chronic toxicity in terrestrial organisms requires higher levels of exposure than will normally be experienced following conventional herbicide use in forestry. Elimination of the herbicide by terrestrial organisms appears to be rapid and evidence of biological concentration of herbicides is scant.

Sub-lethal toxicological effects of herbicides are much more likely than direct toxicity, although the exposures required to produce carcinogenic, teratogenic, mutagenic and other pathological symptoms are thought to be generally higher than normally encountered as the result of field use of herbicides in forestry. Sub-lethal effects may influence the survival and reproduction of terrestrial organisms, with accompanying effects on populations and food chains. However, natural selection in wild populations will eliminate any non-adaptive effects on individuals and there appears to be little chance for long-term effects on the genotypic or phenotypic character of the population.

The situation for the human population is more serious. While the exposure of humans to herbicide residues from forest spray operations is much less than the exposure of animals living in the sprayed area, humans are not subject to normal selective pressures and non-adaptive mutants may be retained within the population, courtesy of our medical successes and our attitudes towards the sanctity of life. Deformed offspring will rapidly be removed from a wild population in a normal, balanced ecosystem, but victims of thalidomide bear witness to the high cost that both individuals and the human population must suffer if we chemically interfere with human genetic and developmental processes. If the unsubstantiated increases in birth defects in areas of Vietnam that were sprayed with herbicides by the U.S. military did in fact occur, they would provide an extreme example of the consequence for humans of the thoughtless uses of herbicides of undetermined toxicological character. Many herbicides have been shown to interfere with mammalian genetic and developmental processes, so even small exposures of humans to herbicides are highly undesirable and unacceptable.

The risk of exposure of humans to herbicide residues from normal forest spray operations is much less than the risk from agricultural and home-use of these chemicals because of the behavior of herbicides in forest ecosystems and the normal remoteness of forests from human habitation. The risk is enormously less than from the military use in Vietnam and the reduction of levels of dioxin present in 2,4,5-T has rendered that herbicide somewhat less noxious. Nevertheless, problems of drift, volatilization, stream contamination, consumption of contaminated wildlife and plants, and carelessness in the planning and execution of forest sprays undoubtedly do result in some exposure of human populations to herbicides following forest spraying and this is unacceptable. However, there is little evidence to support the contention that careful planning and application of appropriate types, forms and formulations of herbicides will not minimize this risk.

Adverse effects of herbicides on aquatic organisms undoubtedly occur in those situations where high concentrations of herbicides occur in streams. From what we know of the behavior of herbicides in streams, such concentrations are probably uncommon and short-lived when they do occur. However, relatively little is known of the more subtle and sub-lethal effects of herbicide residues on aquatic organisms. The development, behavior and migration of fish and aquatic invertebrates may very well be affected, and this has received little or no study. Minor effects of low-level herbicide residues on the stamina and migratory homing mechanisms of anadramous fish could well have consequences that are ultimately the same as lethal acute toxicity.

The use of herbicides can have very adverse effects on ecosystem function and integrity. There is accumulating evidence that minor vegetation or "weeds" frequently play a vital role in forest ecosystems. Loss of soil stability and resistance to erosion and mass wasting following the death and decay of tree roots several years after logging can be greatly offset by the vigorous development of deeply rooting shrub species. Alterations in microclimate following forest harvesting may be lethal or inhibitory for regeneration of the desired crop seedlings: minor vegetation can ameliorate the microclimate until the desired seedlings become established and hardy. Minor vegetation developing on clearcuts after harvesting has been shown to act as a nutrient sponge, retaining nutrients that may otherwise be leached away. Where herbicides are used to accelerate succession, a seral stage that is necessary for the satisfactory development of the subsequent plant community may be omitted, to the detriment of the growth of the crop. For example, herbicidal removal of red alder from a soil in coastal B.C. that has been impoverished in nitrogen and organic matter may have unfavourable effects on the growth of the crop that the operation was designed to benefit.

Most herbicide use in forestry will not have any major undesirable effects on these aspects of ecosystem integrity. Total removal of all vegetation is uncommon, and roots are less commonly killed than above-ground parts. Resprouting from surviving roots will help to preserve soil stability, nutrient retention, a favourable microclimate and successional modification of the site. The degree to which adverse effects are produced will be proportional to the degree to which the vegetation is killed, and selective removal of only a portion of the vegetation will generally not result in the more dramatic consequences associated with total devegetation.

Future wood production in forestry is generally benefited by careful herbicide use. However, loss of soil stability, loss of microclimate, unfavourable alterations of hydrology and loss of nutrients can occur under particular circumstances and these can certainly influence future wood production adversely in certain situations.

Production of wildlife may be benefited or harmed by herbicide use according to whether it creates or destroys desired food supplies and habitat. The effects will generally be more beneficial than harmful. Effects on aquatic productivity will be less desirable. Loss of streambank vegetation will have deleterious consequences for aquatic food chains, stream temperature, fish cover, and streambank stability. Herbicide-killing of streamside red alders in B.C., for example, has very undesirable effects on stream ecology. However, the effects would be very similar if the alders were killed manually. Where streambank vegetation is left unsprayed, and where herbicide does not fall directly into waterbodies, direct acute and chronic toxicity effects will be minimal, although we cannot rule out sub-lethal effects and recent research on the toxicity of dioxin in aquatic ecosystems are cause for continued concern until we have much better information on the behavior of this chemical in ecosystems. Indirect effects on streams may also be of some significance; increases in mass wasting and erosion of soil, coupled with alterations of site hydrology, could aggravate sediment problems in streams, while losses of nutrients from land to water may harm eutrophic (nutrient rich) streams and lakes, but benefit oligotrophic (nutrient poor) aquatic systems. Severe local losses of anadramous fish resulting from either lethal or sub-lethal effects may not appear especially serious at the time, but can be serious because the effects of the loss may be perpetuated for many years.

Herbicide use in forestry will undoubtedly influence the recreational value of forest land. Herbicides have an offensive odour (at least, they do to the author), especially in the characteristically sweet-smelling forest ecosystem. Humans are extremely sensitive to taste and odour, and minute traces of herbicides (parts per billion) can be detected by these senses in water and air. Herbicide-killed vegetation is less attractive (to the author) than an intact green plant community, and until a sprayed area has "greened-up", the aesthetic quality of the area is reduced.

In summary, herbicides have the potential to create a number of adverse effects on future production of wood and other values, but very rarely will these adverse effects be prolonged beyond a few years. Effects on humans, on the other hand, are frequently perpetuated for decades or even indefinitely. Thus, while the effects on forest values may be undesirable, their relative impermenance must make them of less concern to humans than the albeit much smaller risk of effects on human populations. Even a small risk of contamination of human populations must be considered unacceptable until our knowledge of the effects on human genetics and health is substantially better than at present.

As with so much of our powerful technology the environmental hazards of herbicides are probably controlled more by human carelessness and thoughtlessness than by the inherent characteristics of the substances themselves. This poses a great dilemma for the future use of herbicides. Any herbicide may be safe if used safely, and unsafe if used unsafely. The safety of their use is therefore largely a function of the concern, knowledge, and understanding of the applicators. As long as the cavalier attitude of some applicators and planners persists, the risk from herbicide use may be unacceptable. As soon as the care and sincere concern of certain other applicators and planners becomes universal, herbicide usage may become relatively safe.

5.2 Ecological Assessment of Alternatives to the Use of Herbicides

There are a number of alternatives to the use of herbicides in forestry, although they probably vary greatly in their acceptability.

Firstly, it may be possible to utilize the weed species rather than the crop species that it is interfering with. For example, red alder is one of the fastest growing tree species (in its first thirty years) in British Columbia, and yet herbicides are widely used to remove it in favour of Douglas-fir. Certain woody shrub species grown on short rotations may greatly exceed the fibre productivity and yield of the trees in the favour of which they are removed. There would appear to be few adverse ecological effects of this alternative, although the more frequent harvesting may give greater rise to problems of nutrient depletion, soil compaction and erosion than the longer-rotation softwoods released with the aid of herbicides, and aesthetically such short rotations may be less pleasing than longer rotation softwoods. Increasing costs of herbicides, energy and manual labour may make the "if you can't fight them, join them" alternative increasingly attractive.

Manual control of vegetation may have many effects similar to those of herbicidal control, but the absence of toxicological effects is highly desirable. Perhaps the major drawbacks of manual control are those of cost, ineffectiveness, lack of labour and creation of a greater fire hazard through fuel concentration. Physical control of vegetation such as herbs and grasses in the immediate vicinity of seedlings can be achieved by the manual placement of such materials as cardboard or black plastic sheets, but this approach would be ineffective against shrubby weeds.

Fire has been widely used as an alternative to herbicides in the manipulation of vegetation. Slashburning following clearcutting is frequently prescribed for the purpose of weed control rather than fire hazard abatement alone. The ecological effects of slashburning are exceedingly complex and will not be covered in this report: the interested reader is referred to relevant literature or one of several available reviews. Slashburning will generally have more dramatic ecological effects than herbicides, except, as with manual control, the risk of undesirable toxicological consequences is avoided. An option that is finding increasing application is very light applications of herbicides to dessicate weed foliage followed by prescribed fire to remove the aerial biomass. By reducing application rates, some of the undesirable effects of herbicides are reduced.

Another alternative to herbicides might be the selection of a crop species that is able to compete effectively with or tolerate the competition of the weed species. Herbicides are widely used to promote the growth of early or mid seral species such as pines and Douglas-fir. These species are characterized by intolerance of shading and/or intense root competition. Climax species such as the hemlocks (<u>Tsuga</u> sp.) or the true firs (<u>Abies</u> sp.), on the other hand, are capable of surviving fairly intense light competition. The lower economic value and slower growth of these species are offset by economic and environmental savings resulting from the reduced need for weed control.

Plants have been competing with each other for millions of years and many have evolved herbicidal controls of their own. Allelopathy is the term describing the release of chemicals by plants to the atmosphere or soil to kill or reduce competition from other plant species. Allelopathy is not thought to be equally well-developed in all plants and it has been thoroughly studied in only a few. However, just as second and third generation insecticides involve synthetic analogues of highly specific insect sex attractants and host specific pathogenic organisms, the phenomenon of allelopathy offers the hope that it might be possible to solve some weed problems in the future using allelopathic nurse crops or synthetic analogues of allelopathic chemicals. This alternative would not necessarily be without its ecological complications, however. Just as the phenoxy acetic acids were developed as analogues of natural plant growth hormones, the development of synthetic allelopathic chemicals might prove to have similar toxicological problems.

Mechanical control may offer a solution in some locations. In gentle topography with well-drained soils and a lack of large rocks and stumps, it is possible to use relatively inexpensive machines for weeding plantations. However, many brushy sites in old growth forests have wet soils, large stumps, accumulations of large rotten logs or large rocks and boulders requiring an investment in site preparation before such mechanical control will be feasible. This will involve an early investment and the possibility of soil compaction, erosion and nutrient leaching accompanying the site preparation. This will be offset by easier and cheaper mechanical weeding and the avoidance of the use of herbicides.

Biological control of weeds has occasionally been used successfully in agriculture and range management, and it is not unreasonable to consider this as a possible alternative to herbicides in forestry. Successful biological control of insect pests and weeds in agriculture has typically involved pests and weeds introduced to an area outside of its natural range in the absence of its natural enemies. Importation of its natural enemies has led to the control of the pest. When dealing with an indigenous weed it is much less likely that one will be able to use biological controls, and it does not appear likely with our present knowledge that this alternative exists for most herbicide applications in forestry. An exception was provided by an aspiring young forester who solved brush problems on an alluvial flat in British Columbia by arranging for a pig farmer to raise pigs on fenced areas. Within a few months, no living plants were left in these areas, the pigs were fatter, and the areas ready for planting. Unfortunately, the pigs do not appear to offer the possibilities of selective weeding, but sheep have been used to reduce grass competition in young plantations and the idea of making money whilst solving a problem is inherently attractive. The diversion of plant energy to the herbivore food chain where it can be harvested is a more attractive solution than diverting it into unharvestable decomposer food chains by killing plants with herbicides.

Foy and Bingham (1969) reviewed ways of minimizing herbicidal residues in the environment. They listed the use of alternatives to herbicides, including biological control, increasing effectiveness and selectivity of herbicides to permit reduced application rates, and removal, inactivation or alteration of the persistence of the herbicide.

In summarizing this section, the increasing cost of energy, and hence the cost of herbicides, the increasing scarcity of certain fossil fuels, and the increasing cost of labour will make the use of herbicides an increasingly expensive, energy-requiring solution to forest weed problems. It is likely that forest managers will have to examine alternatives to herbicides more in the future than in the past. Unfortunately, we do not know enough about the potential environmental consequences of these alternatives to claim with confidence that they will necessarily be ecologically superior to the careful use of herbicides. All reasonable alternatives should be considered for any particular site and a choice made using a variety of ecological, economic and management criteria.

Tarrant et al. (1973) noted that chemical aids to vegetation management are so firmly established that it is unlikely to suffer such a great decline as the use of chemical insect pest controls. However, they predicted some decline in use as public pressure increases and foresters are increasingly asked to justify their use of vegetation management chemicals. They pointed out the urgent need for more information on the ecological effects of herbicides so that we will be in a better position to justify or curtail chemical vegetation management.

5.3 Steps That Can be Taken to Ameliorate Adverse Effects of Herbicide Use in Forestry

An obvious method of ameliorating the adverse effects of herbicide use in forestry is to avoid their use by employing some alternative solution to problems of vegetation management. However, given that herbicides are the only viable alternative, what steps can be taken to reduce problems.

Perhaps the most important and effective step would be to require all planners, managers and applicators involved in the use of herbicides to have an improved understanding of the public health and environmental aspects of herbicide use. Since careless or inappropriate use is probably a greater factor in producing adverse effects than the inherent nature of the chemicals themselves, a far better knowledge of herbicides and their effects, and greater control in their application should be mandatory. All applicators should be required to take a rigorous theoretical and practical training course in herbicide use, and their knowledge should be upgraded regularly.

Secondly, adverse effects could be reduced by careful planning, layout and control of herbicide applications. By selecting types of herbicides, formulations, application methods, rates of application and timing of application that are appropriate for a particular location, the exposure of non-target organisms and undesirable impacts of herbicides on ecosystem form and function could be significantly reduced.

Management chemicals undoubtedly have an important role to play in the management of natural resources such as forest. However, experience over the past twenty years shows that management chemicals are not a panacea to vegetation and animal management problems. Used as "chemical bulldozers", herbicides can create more problems than they solve, and their use cannot be justified. Used as a part of integrated management, recognizing the ecological character and role of weed species, and with an awareness of the behavior of herbicides in the environment and the risks that they pose, there is little justification for a blanket ban on their use. Because of the risk of human error, carelessness and thoughtlessness, there are undoubtedly environments in which herbicide use should be greatly curtailed or abandoned. While it is possible to use them safely in such environments, the human factor renders their use too risky. In many forest environments, the risk posed by the human factor may be acceptable. In such environments the use of herbicides should probably be permitted, with stringent requirements to reduce the risk of human error.

5.4 Information Gaps Concerning the Ecological Effects Of Herbicides That Inhibit the Formulation of Corrective Measures.

Attempting to identify gaps in our knowledge of the ecological effects of herbicides in forestry is an intimidating undertaking. So much of our knowledge is based on agricultural usage, on studies dealing with non-forest organisms, and on laboratory experiments that almost all pronouncements on forestry usage must be made with considerable caution. Recognizing the extensive gaps in our detailed knowledge (the gaps in general knowledge are fewer and narrower), and recognizing that the closing of these gaps will involve the efforts of all segments of the scientific community from medical doctors to engineers, the following are some lines of investigation that deserve attention in the short-run.

1. Effects on aquatic ecosystems:

    a. Sub-lethal effects of herbicide residues on aquatic organisms deserve study. In particular, the effects on stamina and behavior of aquatic organisms, and the variation of effects with organisms of different age require documentation.

    b. Further work on the chemistry, behavior, and toxicology of dioxin in aquatic environments. Recent reports suggest the need for further studies of this highly toxic substance.

    c. Effects of terrestrial herbicide use on the inorganic chemistry of streams and lakes. With the exception of the Hubbard Brook experiment, almost no work has been done on this problem. We do not know the extent to which forest biogeochemistry is altered by conventional herbicide use.

    d. Identifying stream characteristics that determine the behavior of herbicide residues in streams. We need better knowledge of how to recognize streams that are inefficient at removing herbicide residues from solution and in which herbicide residues may be expected a long way downstream from the site of application.

2. Effects on terrestrial ecosystems:

    a. Further studies are required on the leaching of herbicide residues in different types of forest soils and climates. There have been few careful studies of soil leaching and surface runoff to characterize accurately herbicide movements. In particular, lysimeter field studies of herbicide movements in soils are suggested.

    b. Effects of herbicides on forest floor decomposition, nitrification, and the leaching of inorganic nutrients require further study. These phenomena are rather poorly documented for the variety of forest floor, soil and forest types in which herbicides are used.

    c. Research is needed on application methodologies and formulations that will minimize problems of drift and volatilization. Pelleting rather than sprays might reduce these problems, but may not achieve the desired objectives of vegetation management. Further work is suggested on this topic.

    d. Timing of sprays requires further research. This will vary from one climatic region to another. Stream contamination appears to be related to post-spray precipitation, and this relationship deserves better documentation in order to select the best time for spraying in terms of herbicide effectiveness and minimizing environmental problems.

    e. Research on alternative methods of vegetation management deserve attention, including mechanical alternatives, the genetic improvement, culturing and utilization of woody weeds, and the use of alternative species and silvicultural systems. Planting large stock, the use of advanced regeneration and the use of growth stimulants require and deserve investigation. The latter alternative would seem to have considerable promise.

    f. Research on systemic herbicides that are excreted by tree roots to produce a type of allelopathic effect might yield promising results. This would control weeds only in the immediate vicinity of the crop plant thus avoiding the undesirable effects of broadcast weed removal.

Considering the potential problems of and public sensitivity to the use of herbicides in forestry, remarkably little research has been done on the environmental aspects of herbicide use in forestry, the admirable work of one or two groups (e.g. Norris and associates in Oregon) notwithstanding. This leaves the public with an agricultural and military perspective of herbicide usage in forestry that results in a very negative attitude. It leaves the forest manager with an inadequate data base with which to make either educated answers to public criticism or rational decisions about herbicide use. Investment in increased research on the environmental effects of herbicide use in forestry would appear to be long overdue and very justifiable in terms of public safety, environmental protection, and efficient forest management.

References Cited in Chapter 5

Foy, C.L. and S.W. Bingham. 1969. Some research approaches toward minimizing herbicidal residues in the environment. Residue Rev. 29: 105-135.

Tarrant, R.F., H.J. Gratkowski and W.E. Waters. 1973. The future role of chemicals in forestry. PNW For. Range Exp. Sta., U.S.D.A., For. Serv. Gen. Tech. Rept. PNW-6, 10 p.

Canadian Forestry Service
Pacific Forest Research Centre
506 West Burnside Road
Victoria, B.C.

BC-X-139 July, 1975