General Technical Report PNW-149
**February 1983**

EDITOR'S
FILE COPY

# Influence of Forest and Rangeland Management on Anadromous Fish Habitat in Western North America

## FOREST CHEMICALS

L.A. NORRIS, H.W. LORZ and S.V. GREGORY




U.S. Department of Agriculture Forest Service
Pacific Northwest Forest and Range Experiment Station

# ABSTRACT

Herbicides, insecticides, fertilizers, and fire retardants are chemicals used to protect or enhance certain forest resources. Their use may directly affect anadromous fish by exposing them to toxic amounts of the chemical. Indirect effects are also possible through chemically induced alteration of habitat, including direct effects on fish-food organisms.

Data on the movement, persistence, and toxicity of chemicals provide a basis for evaluating potential direct effects of chemical use on fish and fish-food organisms. Data are included for seven herbicides (2,4-D, picloram, atrazine, MSMA, fosamine ammonium, glyphosate, and dinoseb), five insecticides (malathion, carbaryl, azinphos-methyl, carbofuran, and acephate), urea fertilizer, and the ammonium-based fire retardants. Comparison of exposures and toxicities of these materials shows their current uses provide a reasonable margin of safety to anadromous fish if direct application to surface waters is avoided.

Much less information is available on indirect effects of chemical use on anadromous fish habitat. The potential effects are known, but few quantitative data exist to evaluate their importance. Apparently, the best way to minimize indirect effects is to minimize the effects of chemicals on all biota in the riparian zone.

Research is needed on the role of buffer strips in reducing entry and impact of forest chemicals in streams, patterns of entry of selected chemicals into streams, instream fate of most forest chemicals, toxicity characteristics of combinations of forest chemicals, the interaction between amount and duration of organism exposure to toxicants, potential latent effects from short-term exposure, effects of forest chemicals on aquatic species under field conditions, and the indirect effects of using forest chemicals on anadromous fish habitat.



**KEYWORDS:** Anadromous fish, fish habitat, herbicide, insecticide, urea fertilizer, fire retardants, 2,4-D, picloram, atrazine, MSMA, fosamine ammonium, glyphosate, dinoseb, carbaryl, acephate, malathion, azinphos-methyl, carbofuran, ammonia.

USDA FOREST SERVICE
General Technical Report PNW-149

# INFLUENCE OF FOREST AND RANGELAND MANAGEMENT ON ANADROMOUS FISH HABITAT IN WESTERN NORTH AMERICA

William R. Meehan, Technical Editor

9. Forest Chemicals

L. A. NORRIS
USDA Forest Service, Pacific Northwest Forest and Range Experiment Station, Forestry Sciences Laboratory, Corvallis, Oregon

H. W. LORZ
Research and Development Section, Oregon Department of Fish and Wildlife, Corvallis, Oregon

S. V. GREGORY
USDI Fish and Wildlife Service, Columbia National Fishery Research Laboratory, Columbia, Missouri, currently at Department of Fisheries and Wildlife, Oregon State University, Corvallis

**1983**

PACIFIC NORTHWEST FOREST AND RANGE EXPERIMENT STATION
Forest Service, U.S. Department of Agriculture, Portland, Oregon

# PREFACE

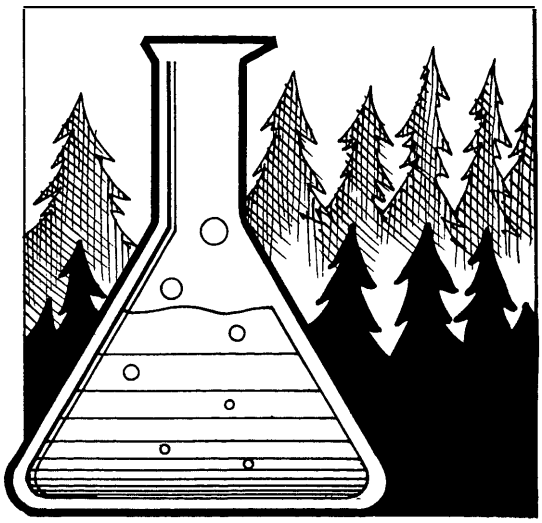
This is one of a series of publications on the influence of forest and rangeland management on anadromous fish habitat in western North America. This paper addresses the effects of forest chemicals on fish habitat. Our intent is to provide managers and users of forests and rangelands with the most complete information available for estimating the consequences of various management alternatives.

In this series of papers, we will summarize published and unpublished reports and data as well as the observations of scientists and resource managers developed over years of experience in the West. These compilations will be valuable to resource managers in planning uses of forest and rangeland resources, and to scientists in planning future research.

Previous publications in this series include:

1. "Habitat requirements of anadromous salmonids," by D. W. Reiser and T. C. Bjornn.
2. "Impacts of natural events," by Douglas N. Swanston.
3. "Timber harvest," by T. W. Chamberlin.
4. "Planning forest roads to protect salmonid habitat," by Carlton S. Yee and Terry D. Roelofs.
6. "Silvicultural treatments," by Fred H. Everest and R. Dennis Harr.
7. "Effects of livestock grazing," by William S. Platts.
8. "Effects of mining," by Susan B. Martin and William S. Platts.
11. "Processing mills and camps," by Donald C. Schmiege.
12. "Rehabilitating and enhancing stream habitat: 1. Review and evaluation," by James D. Hall and Calvin 0. Baker.
13. "Rehabilitating and enhancing stream habitat: 2. Field applications," by Gordon H. Reeves and Terry D. Roelofs.

# TABLE OF CONTENTS



# INTRODUCTION

Forest chemicals are used to protect or enhance a wide array of forest resources. Their use may have adverse effects on anadromous fish or their habitat. Forest managers, regulatory officials, and the interested public believe strongly that if forest chemicals are used, they must yield significant benefits without imposing unreasonably adverse environmental effects. We review and summarize what is known about the interaction between forest chemicals and anadromous fish and their habitat. **Our objective is to provide the reader** with a scientific basis for making informed, technically sound decisions about the use of these important management tools with respect to anadromous fish and their habitat.

# USE OF CHEMICALS IN THE FOREST

The three major categories of forest chemicals are pesticides,[1/] fertilizers, and fire retardants. Many chemicals are used in both agriculture and forestry, but the magnitude, intensity, and patterns of use are markedly different (table 1). The common, chemical, and trade names of forest chemicals used in this paper are in appendix table 18.

Table 1--Comparative annual use of chemicals in agriculture and forestry

| | Agriculture | Forestry |
|---|---|---|
| | $10^3$ kilograms | |
| Pesticides, 1980:[1/] | | |
| Insecticides | 138 924 | [2/]71 |
| Herbicides | 202 030 | [2/]169 |
| Fungicides | 22 700 | – [2/]9 |
| | $10^3$ metric tons | |
| Fertilizers, 1978:[3/] | | |
| Nitrogen | 9 636 | 55 |
| Phosphorus | 2 273 | 5 |

[1/] Agricultural data are from table 3, Pesticide Industry Sales and Usage, 1980 market estimates, U.S. Environmental Protection Agency, Washington, D.C., September 1980; forestry data are only for USDA Forest Service, National Forest Systems land, from table E1, Pesticide-Use Advisory Memorandum 284 (2150 Pesticide-Use Management and Coordination, March 12, 1981), USDA Forest Service, Washington, D.C.
[2/] USDA Forest Service, National Forest Systems land only.
[3/] Bengtson (1979).

[1/] This publication reports research with pesticides. It does not contain recommendations for their use, nor does it imply that the uses discussed here have been registered. All uses of pesticides must be registered by appropriate State and/or Federal agencies before they can be recommended. The use of trade, firm, or corporation names in this publication is for the information and convenience of the reader. Such use does not constitute an official endorsement or approval by the U.S. Department of Agriculture of any product or service to the exclusion of others that may be suitable.

# PESTICIDES

Pesticides are defined for regulatory purposes as agents used to prevent, destroy, repel, or mitigate pests. The term pesticide includes many specific chemical substances, which can be grouped according to the type of pest they are intended to control: herbicides, insecticides, fungicides, rodenticides, piscicides, and animal repellents. Although many pesticides are registered by the U.S. Environmental Protection Agency (EPA) for use in agriculture, fewer than 10 have substantial use in forestry. Forestry uses account for less than 1 percent of the total pesticides used in the United States.

Herbicides and insecticides account for more than 80 percent of the pesticides used by the USDA Forest Service (table 2). In general, these figures underestimate the total use in forestry because they do not include pesticides applied by other Federal land-managing agencies or by various State and private groups. Fumigants and fungicides account for 10 to 17 percent of the total, but their use is largely restricted to forest nurseries.

The specific herbicides and insecticides used on the National Forests or on other lands through Federal assistance projects coordinated by the USDA Forest Service are listed in tables 3 and 4. 2,4-D and picloram (alone or in combination with other herbicides) accounted for about 70 percent of the herbicides applied in the fiscal years 1979, 1980, and 1981. The use of glyphosate has increased as both registration and use-experience have expanded. The amount of insecticide used was much lower in fiscal year 1981 compared to the two previous years. Malathion, carbaryl, and acephate accounted for nearly all the insecticide used for silvicultural purposes in the fiscal years 1979, 1980, and 1981.

Table 2--Pesticides used by the USDA Forest Service

| Pesticide | Fiscal year 1981 [1] | | | Fiscal year 1980 [2] | | | Fiscal year 1979 [3] | | |
|---|---|---|---|---|---|---|---|---|---|
| | Acres [4] | Pounds [5] | Percent of total pounds | Acres [4] | Pounds [5] | Percent of total pounds | Acres [4] | Pounds [5] | Percent of total pounds |
| Herbicides | 197.043 | 380,828 [6] | 76.0 | 229,877 | 371,748 [7] | 62.1 | 184,048 | 471,184 [8] | 63.2 |
| Fumigants and fungicides | 3,618 | 85,363 | 17.0 | 2,375 | 63,015 | 10.5 | 1,334 | 81,265 | 10.9 |
| Insecticides | 49,671 | 31,595 [9] | 6.3 | 173,500 | 156,590 [10] | 26.2 | 272,420 | 173,000 [11] | 23.2 |
| Rodenticides | 51,537 | 1,570 | .3 | 29,693 | 2,063 | .3 | 44,920 | 9,066 | 1.2 |
| Repellents | 6,220 | 1,278 | .2 | 8,998 | 2,723 | .5 | 9,502 | 9,137 | 1.2 |
| Wood preservatives | -- | 255 | <.1 | -- | 1,564 | .3 | 12/ -- | 12/ -- | E/ -- |
| Piscicides | 92 | 28 | <.1 | 256 | 633 | .1 | 239 | 915 | 1 |
| Algacides | 8 | 353 | <.1 | 17 | 429 | <.1 | 54 | 408 | <.1 |
| Behavioral chemicals | 12/ -- | 12/ -- | 12/ -- | 12/ -- | 12/ -- | 12/ -- | 2,270 | 17 | <.1 |
| Total | 308.012 | 501,271 | 100 | 444,716 | 598,765 | 100 | 514,786 | 744,992 | 100 |
| Percent of 5-year average | 66 | 77 | -- | 95 | 92 | -- | 110 | 115 | -- |

-- = not applicable, unless otherwise indicated.
Note: The fiscal year begins October 1 of the previous year and runs through September 30 of the year designated.

1/ Pesticide-Use Advisory Memorandum 316 (2150 Pesticide-Use Management and Coordination, April 5, 1982), USDA Forest Service, Washington, D.C.
2/ Pesticide-Use Advisory Memorandum 284 (2150 Pesticide-Use Management and Coordination, March 12, 1981), USDA Forest Service. Washington, D.C.
3/ Pesticide-Use Advisory Memorandum 246 (2140 Pesticide-Use Management, June 5, 1980). USDA Forest Service, Washington, D.C.
4/ 1 acre = 0.4 hectare.
5/ 1 pound = 0.45 kilogram.
6/ 30 percent by aerial application.
7/ 23 percent by aerial application.
8/ 26 percent by aerial application.
9/ 29 percent by aerial application.
10/ 84 percent by aerial application.
11/ 87 percent by aerial application.
12/ No use reported.

Table 3--Herbicides most commonly used by the USOA Forest Service

| Herbicide | Fiscal year 1981[1] | | | Fiscal year 1980[2] | | | Fiscal year 1979[3] | | |
|---|---|---|---|---|---|---|---|---|---|
| | Acres[4] | Pounds[5] | Percent of total pounds | Acres[4] | Pounds[5] | Percent of total pounds | Acres[4] | Pounds[5] | Percent of total pounds |
| 2,4-D | 72,587 | 145,474 | 38.2 | 117,174 | 205.676 | 55.3 | 73,449 | 185,323 | 39.3 |
| 2,4-D + picloram | 69,158 | 89,143 | 23.4 | 9,090 | 12.670 | 3.4 | 57.000 | 135,307 | 28.7 |
| Picloram | 15,190 | 33.722 | 8.9 | 68.001 | 35.849 | 9.6 | 15.853 | 24,947 | 5.3 |
| Atrazine | 5,968 | 21,725 | 5.7 | 7,099 | 29,772 | 8.0 | 5,299 | 18,915 | 4.0 |
| Mineral spirits | -- | -- | -- | 50 | 15.615 | 4.2 | 89 | 6,600 | 1.4 |
| Glyphosate | 12,489 | 17.622 | 4.6 | 5,667 | 9,601 | 2.6 | 3,668 | 5,841 | 1.2 |
| Dalapon | 4,286 | 12,694 | 3.3 | 4,076 | 9,084 | 2.4 | 4,240 | 10,611 | 2.3 |
| Simazine | 853 | 7,307 | 1.9 | 1.016 | 4,790 | 1.3 | 4,298 | 9,927 | 2.1 |
| Fosamine amnonium | 1,702 | 6,693 | 1.8 | 1,649 | 8.340 | 2.2 | 1,949 | 7,939 | 1.7 |
| Hexazinone | 4,550 | 6.487 | 1.7 | 2,768 | 3,900 | 1.0 | 383 | 839 | .2 |
| Dicamba | 4,208 | 4.786 | 1.3 | 3,714 | 4,207 | 1.1 | 1.060 | 1,404 | .3 |
| 2,4-D + 2.4-OP | 1.142 | 4,180 | 1.1 | 1.777 | 6,877 | 1.8 | 3.1 52 | 8,947 | 1.9 |
| Sodium metaborate + sodium chlorate | 15 | 2,410 | .6 | 11 | 3.700 | 1.0 | 11 | 793 | .2 |
| Amnonium sulfamate | 260 | 3,000 | .8 | 173 | 1,983 | .5 | 450 | 3,502 | .7 |
| Amitrole | 986 | 2,333 | .6 | 822 | 1.902 | .5 | 880 | 1,710 | .4 |
| MSMA | 940 | 617 | .2 | 1,386 | 421 | .1 | 3,558 | 18.604 | 3.9 |
| 2,4-D + dicamba | 1,612 | 3,421 | .9 | 288 | 164 | < .1 | 6,232 | 14,971 | 3.2 |

-- = no use reported.
Note: The fiscal year begins October 1 of the previous year and runs through September 30 of the year designated.

1/ Pesticide-Use Advisory Memorandum 316 (2150 Pesticide-Use Management and Coordination, April 5, 1982), USOA Forest Service, Washington, D.C.
2/ Pesticide-Use Advisory Memorandum 284 (2150 Pesticide-Use Management and Coordination, March 12, 1981), USDA Forest Service, Washington, D.C.
3/ Pesticide-Use Advisory Memorandum 246 (2140 Pesticide-Use Management. June 5. 1980), USDA Forest Service, Washington, D.C.
4/ 1 acre = 0.4 hectare.
5/ 1 pound = 0.45 kilogram.

Table 4--Insecticides most comnonly used by the USDA Forest Service

| Insecticide | Fiscal year 1981[1] | | | Fiscal year 1980[2] | | | Fiscal year 1979[3] | | |
|---|---|---|---|---|---|---|---|---|---|
| | Acres[4] | Pounds[5] | Percent of total pounds | Acres[4] | Pounds[5] | Percent of total pounds | Acres[4] | Pounds[5] | Percent of total pounds |
| Azinphos-methyl[6] | -- | 6,431 | 20.3 | -- | 17,923 | 11.4 | -- | 4,324 | 2.5 |
| Carbofuran[6] | -- | 5,511 | 17.4 | -- | 4,223 | 2.7 | -- | 5,469 | 3.2 |
| Malathion | 9,601 | 4,854 | 15.3 | 141,485 | 102,347 | 65.3 | 193,364 | 93,512 | 54.1 |
| Carbary1 | 4,984 | 4,521 | 14.3 | 26,091 | 29,687 | 19.0 | 51,178 | 50,508 | 29.2 |
| Acephate | 3,014 | 2,261 | 7.2 | -- | 3 | <.1 | 23,400 | 11,706 | 6.7 |
| Ethylene dibromide[7] | -- | 765 | 2.4 | -- | 40 | <.1 | -- | 2,521 | 1.5 |
| Toxaphene[8] | -- | 480 | 1.5 | -- | 1,607 | 1.0 | -- | 2,295 | 1.3 |
| Lindane | -- | 163 | .5 | -- | 443 | .3 | 370 | 309 | .2 |
| Diazinon[9] | -- | 162 | .5 | 64 | 81 | < .1 | 101 | 124 | < .1 |
| Tetraclorvinphos[8] | 10/ -- | 10/ -- | 10/ -- | 58 | 54 | <.I | 31 | 68 | < .1 |

-- = data not applicable, unless otherwise indicated.
Note: The fiscal year begins October 1 of the previous year and runs through September 30 of the year designated.

1/ Pesticide-Use Advisory Memorandum 316 (2150 Pesticide-Use Management and Coordination, April 5, 1982), USDA Forest Service, Washington,. D.C.
2/ Pesticide-Use Advisory Memorandum 284 (2150 Pesticide-Use Management and Coordination, March 12, 1981), USDA Forest Service, Washington,. D.C.
3/ Pesticide-Use Advisory Memorandum 246 (2140 Pesticide-Use Management, June 5, 1980), USDA Forest Service, Washington, D.C.
4/ 1 acre = 0.4 hectare. Not all applications are per acre; in projects for control of seed and cone insects, for instance, the values in the pesticide-use advisory memorandums are the number of trees treated.
5/ 1 pound = 0.45 kilogram.
6/ Control of seed and cone insects in seed-production areas.
7/ Control of bark beetles on cut logs.
8/ Control of ticks and lice on cattle.
9/ Control of insects in forest-tree nurseries.
10/ No use reported.

## FERTILIZERS

Fertilizers are applied annually to only a small portion of commercial forest land, (table 1). Several private and public land-management groups, however, have been applying forest fertilizers for over 10 years, particularly in the Pacific Northwest, where nitrogen deficiencies occur and, to a much lesser degree, in the Southeast, where phosphorous deficiencies may occur. Between 1965 and 1975, about 300 000 ha of Douglas-fir forests were fertilized in western Oregon and Washington (Moore 1975b). Bengtson (1979) wrote an excellent discussion of the use of fertilizers in American forestry.

## FIRE RETARDANTS

The use of chemical fire retardants has increased steadily since they were introduced in the 1930's (table 5). Douglas (1974) and Norris et al.[2/] summarized most of the literature on both the use and impacts of chemical fire retardants. Fire retardants are not always thought of as chemical agents, but, in fact, they are. The chemical nature of fire retardants has changed since they were first developed. Borate salts were the first chemical fire retardants to be widely used. They were effective, long-lasting retardants, but were also potent soil sterilants that retarded establishment and regrowth of vegetation. Bentonite clay suspensions in water have also been used, but they are not as effective as other materials. The chemical fire retardants in common use today are composed principally of ammonium phosphate or ammonium sulfate and small amounts of several other chemicals, such as dyes, wetting agents, thickeners, corrosion inhibitors, and bactericides.

Table 5--Fire retardant use in the United States[1/]

| Year | Quantity used | User group |
|---|---|---|
| | Liters | |
| 1956 | 87 000 | All users |
| 1961 | 28 400 000 | All users |
| 1966 | 22 500 000 | USDA For. Serv. |
| 1966 | 12 200 000 | Calif. Div. For. |
| 1966 | 3 800 000 | USDI Bur. Land Manage. |
| 1970 | 64 400 000 | All users |
| 1977[2/] | 56 669 902 | USDA For. Serv. |
| 1978[2/] | 24 371 221 | USDA For. Serv. |
| 1979[2/] | 54 795 771 | USDA For. Serv. |
| 1980[2/] | 39 348 023 | USDA For. Serv. |
| 1981[2/] | 44 712 371 | USDA For. Serv. |

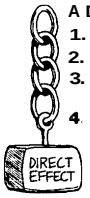
[1/] Personal communication, G. E. Cargill, U.S. Department of Agriculture, Forest Service, Washington, D.C. (December 14, 1980 and September 21, 1982 - memorandums with attachments).
/Fiscal year. The fiscal year begins October 1 of the previous year and runs through September 30 of the year designated. About 70 percent of this use is in Oregon, Washington, and California.

# RELATIQN OF USE OF CHEMICALS IN THE FOREST TO ANADROMOUS FISH HABITAT

Water is essential to the habitat of anadromous fish. The quality of the water that forested watersheds yield reflects human activities and natural processes. Forest lands comprise only one-third of the total area of the United States, but they receive more than half of the total precipitation and yield more than three-fourths of the total streamflow. Forested watersheds in the United States on the average receive more than 114 cm of precipitation and yield more than 51 cm of runoff annually, more than seven times the average amount from other lands (Storey 1965). Clearly, the possibility that chemical use in forest management may alter water quality, or some other aspect of anadromous fish habitat, deserves careful consideration.

/Unpublished report, "The behavior and impact of chemical fire retardants in forest streams," by L. A. Norris, C. L. Hawkes, W. L. Webb, D. G. Moore, W. B. Bollen, and E. Holcombe, U.S. Dep. Agric., For. Serv., Pac. Northwest For. and Range Exp. Stn., For. Sci. Lab., Corvallis, Oreg., 1978.

The chemicals used in forestry may have direct or indirect effects or no effect on anadromous fish. Direct effects require that the organism and the chemical come in physical contact. Once in contact, the chemical must be taken up by the organism and moved to the site of biochemical action where the chemical must be present in an active form at a concentration high enough to cause a biological effect (fig. 1). Direct chemical effects can be evaluated by using traditional concepts of toxicology and dose–response relationships.




**A DIRECT CHEMICAL EFFECT REQUIRES:**

1. DIRECT PHYSICAL CONTACT WITH THE CHEMICAL.
2. UPTAKE BY THE ORGANISM..
3. MOVEMENT TO THE BIOCHEMICAL SITE OF ACTION.
4. RESIDENCE AT THE SITE OF ACTION IN SUFFICIENT QUANTITY AND IN A TOXIC FORM TO CAUSE AN EFFECT.

Figure 1.––A direct chemical effect requires that a chain of events takes place, including direct contact between the organism and the chemical, uptake of the chemical and its movement to the biochemical site of action, and residence at the site of action in an active form, in sufficient quantity and long enough for a direct effect to occur.

Indirect effects result from chemically induced modification of the habitat, rather than from the direct interaction between the chemical and the organism. Examples of indirect effects are insecticide–induced decreases in the biomass of terrestrial or aquatic insects resulting in a decrease in the supply of food for anadromous fish, and reduction in cover, shade, and sources of food from riparian vegetation as a result of herbicide deposition in a streamside zone.

# DIRECT CHEMICAL EFFECTS

One of the hazards of using chemicals in the forest is the risk of direct adverse toxic effects on nontarget organisms. The two factors that determine the degree of hazard are the toxicity of the chemical and the likelihood that nontarget organisms will be exposed to toxic doses. Toxicity alone does not make a chemical hazardous, exposure to a toxic dose must also occur. Therefore, an adequate hazard assessment requires equal consideration of both the likelihood of exposure and the toxicity of the chemical (Norris 1971a, Sanders 1979).

## TOXICITY IN AQUATIC SPECIES

Acute toxicity is the fairly rapid response of organisms to a few, relatively large doses of chemical administered over a short period of time. Chronic toxicity is the slow or delayed response of organisms to many, relatively small doses of chemical administered over a long period of time. The kind of response (acute or chronic) depends on the magnitude of the dose and the duration of exposure.

## EXPOSURE IN THE AQUATIC ENVIRONMENT

Aquatic organisms may come in direct contact with a chemical in water, sediment, or food. The rate and method of application and behavior of the chemical in the environment determine both the level and the length of time any particular chemical will be in one or more of these three compartments.

## Chemicals in Water

Chemicals may enter water by one or more of the following routes: direct application, drift, mobilization in ephemeral stream channels, overland flow, and leaching. Each route of entry results in a different level and duration of entry and, therefore, a different magnitude and duration of exposure. The degree to which any particular route of entry operates depends on the nature of the application, characteristics of the chemical, and characteristics of the area treated.

Many forest chemicals are aerially applied by either fixed- or rotary-wing aircraft, although a large proportion of herbicides are applied by ground-based equipment such as hand-held nozzles fed from either high- or low-pressure pumping systems, backpack sprayers, air-blast sprayers, direct stem-injection equipment, or, occasionally, by scattering of pelletized chemical by hand (table 2). Aerial applications in or near aquatic zones present the greatest probability of introducing chemicals into the aquatic environment by either direct application or drift. Aerial applications away from aquatic zones do not offer any greater opportunity for chemical entry into water than any other type of application. Chemicals that are applied in or near aquatic zones with ground-based equipment can also enter streams by direct application and drift.

Direct application and drift are physical processes that are largely independent of the chemical properties of the material being applied. The principal variables are vertical and horizontal distance between the points of application and the exposed waters, physical characteristics of the material being applied (droplet or pellet size and characteristics of the carrier), atmospheric conditions (wind speed and direction, relative humidity, and temperature,), and type of application equipment and its operating parameters. The concepts, principles, and practice of aerial application of pesticides are presented in a series of five papers (by Maksymiuk, Jasumback, McComb, and Witt) in the proceedings of a pesticide applicators' training course (Capizzi and Witt 1971), the proceedings of a workshop on behavior and assessment of pesticide-spray application (Roberts 1976), and a USDA handbook (U.S. Department of Agriculture 1976).

### Direct Application to Surface Waters

Direct application is the route most likely to introduce significant quantities of chemicals into surface waters. It has the potential to produce the highest concentrations and, therefore, cause the most pronounced acute toxic effects. The duration of entry and the subsequent duration of exposure, however, will be brief--a few minutes to a few days (Norris and Moore 1971, Norris 1978). Concentrations that result depend on the rate of application and the ratio of stream-surface area to volume. The persistence of the chemical in the application zone depends on the length of the stream treated, the velocity of streamflow, and the hydrologic characteristics of the stream channel. The concentration of

introduced chemicals normally decreases rapidly with downstream movement because of dilution and the interaction of the chemical with various physical and biological components of the stream system.

### Drift From Nearby Spray Areas

Drift from nearby spray areas is similar to direct application except that peak concentrations are lower and the probability of impacts on stream organisms is reduced. Accidental drift of chemical from nearby spray areas to stream surfaces is a likely means of chemical entry into surface waters, but one that can be minimized through careful selection of chemical formulations, carriers, and equipment, and attention to atmospheric and operating conditions.

### Mobilization in Ephemeral Stream Channels

Ephemeral stream channels are difficult to see from the air and may be sprayed along with the rest of the area. The problem may be more acute during aerial applications, because ground applications usually provide greater opportunity for avoiding these areas. Residues remaining in ephemeral stream channels are available for mobilization by the expanding stream system (described by Hewlett and Hibbert 1967) that develops during heavy precipitation. This process probably accounts for increases in chemicals occasionally observed in streams during the first storms after application (Norris 1967, Norris et al. 1978, 1982).

### Overland Flow

Overland flow occurs infrequently on most forest lands because the infiltration capacity of the forest floor and soil is usually far greater than rates of precipitation (Rothacher and Lopushinsky 1974). Bare and heavily compacted soil may yield surface runoff, but these areas are not widespread and would seldom be treated with forest chemicals.

### Leaching

Leaching of chemicals through the soil profile is a process of major public concern, but it is the least likely to occur in forest environments. Most chemicals used in forestry are relatively immobile in soil. Intense leaching can move chemicals a few centimeters to 1 m in depth, but these distances are short in comparison to distances between treated areas and streams (Norris 1971b). Most forest chemicals do not persist long enough for significant leaching to occur.

The various routes of chemical entry into streams result in widely different degrees of exposure to aquatic organisms. Direct application and drift are likely to result in the highest concentrations of chemicals in water, but persistence is brief. Mobilization in ephemeral stream channels and overland flow are associated with periods of significant precipitation, therefore, the concentrations in the water will be substantially less than those resulting from direct applications, although the duration of exposure may be slightly longer. Leaching (if it occurs) can introduce only small amounts of chemical into the stream, although the process could be prolonged.

The degree to which any one of these routes of entry is involved depends on the properties of both the chemical and the environment. Properties of the chemical (such as vapor pressure or solubility in water) and the properties of the environment (such as temperature, moisture, and soil characteristics) interact to

produce the particular behavior (movement, persistence, and fate) we observe in the environment (fig. 2). This behavior largely determines the route of entry of chemicals into forest streams.

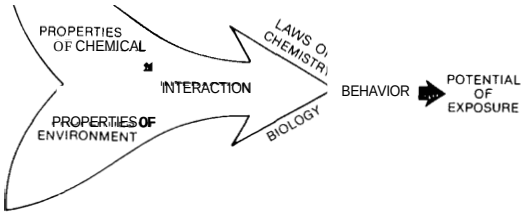



Figure 2.--The properties of the chemical interact with the properties of the environment in a manner directed by the laws of nature to produce the movement, persistence, and fate of the chemical--which determine the level and duration of an organism's exposure.

## Chemicals on Sediment

Stream sediments may be contaminated with forest chemicals by deposition of sediments carrying adsorbed chemicals from the land or by the adsorption of chemical from the water onto sediments already in place in the stream (Barnett et al. 1967).

The deposition of chemically contaminated sediments in streams requires that the chemical be present in the erodible layer of soil at the time erosion occurs. Persistence of the chemical is the predominant factor affecting its presence in the soil. This characteristic will be discussed in more detail in a later section. In general, however, nearly all chemicals are applied between March and October, and surface-erosion events are most frequently associated with intense winter storms from late November through February. Thus, appreciable quantities of a particular chemical must persist for 1 to 9 months for harmful amounts to be present in the soil at the time the first surface-erosion events are likely to occur.

Erosion is often accelerated by forest management, but the principal sources of sediment are road construction, road failure, landslides, and streambank erosion (Rice et al. 1972). Chemicals are seldom applied in such a temporal and spatial relationship with these erosion events. We believe significant movement of chemical residues to streams by this process is unlikely. The incidence of surface erosion from forest lands near anadromous fish habitat is discussed in detail in several other papers in this compendium.

Chemicals may be adsorbed from water (see earlier discussion of chemicals in water) by sediments already in the stream. Adsorption of chemical to sediments may be by chemical, physical, or a combination of these types of bonding, as determined by the physical-chemical properties of both the chemical and the sediment. The adsorption process is beyond the scope of our paper. It was reviewed in a series of symposium papers edited by Weber and Matijevic (1968). The adsorption characteristics of forest chemicals are discussed in a later section of this paper.

Norris (1969) and Norris et al. (1982) reported the discharge of pesticides in stream water is believed to represent the mobilization of chemicals in ephemeral stream channels. This research did not distinguish between pesticides in solution and those adsorbed on sediments eroded from the stream channel.

## Chemicals in the Food Chain

Chemicals may be in or on the food of anadromous fish if the food substance is sprayed directly (for instance, if terrestrial insects that are sprayed fall into the water), or if food substances bioaccumulate the chemical from the water. Residues in food from direct spraying are likely to occur primarily during or shortly after application. Few data are available on this process.

Bioaccumulation is the uptake by an organism of a chemical from its environment (for example, the uptake by fish, via the gills, of DDT from the water). Kenaga (1975, 1980a, b) and Geyer et al. (1980) provide good reviews and substantial data on bioaccumulation of organic chemicals, including many pesticides. The physical–chemical properties of the compound and the organism are the predominant factors that determine the extent of bioaccumulation. The most important properties are the level of fat in the organism and the ratio of fat solubility to water solubility of the chemical.

Bioaccumulation resulting in concentrations of chemical in an organism that are 100,000 times the concentration of the chemical in the water have been noted. The highest values occur in organisms with a high fat content that are exposed to chemicals with a high fat solubility/water solubility ratio. DDT or TCDD in fish are pertinent examples. Chemicals that are highly water soluble, like picloram or glyphosate, show little tendency to bioaccumulate. The relation of water solubility to bioaccumulation is illustrated in figure 3, and data for specific chemicals are given in table 6.

Table 6--Water solubility and bioconcentration factor[1/]

| Chemical | Water solubility | Bioconcentration factor |
|---|---|---|
| | Milligrams/liter | |
| Acephate | 650 000 | 0.3 |
| Atrazine | 33 | 86 |
| Picloram | 430 | 20 |
| 2,4-D | 900 | 13 |
| Dinoseb | 50 | 68 |
| Carbaryl | 40 | 77 |
| Malathion | 145 | 37 |
| Carbofuran | 415 | 21 |
| Glyphosate | 12 000 | 3 |
| DSMA | 254 000 | .5 |
| Chlordecone | .3 | 333 |
| Mirex | .6 | 820 |
| Methoxychlor | .003 | 16 360 |
| Urea | 1 000 000 | .5 |
| DDT | .002 | 22 500 |

1/ From Kenaga (1980b).

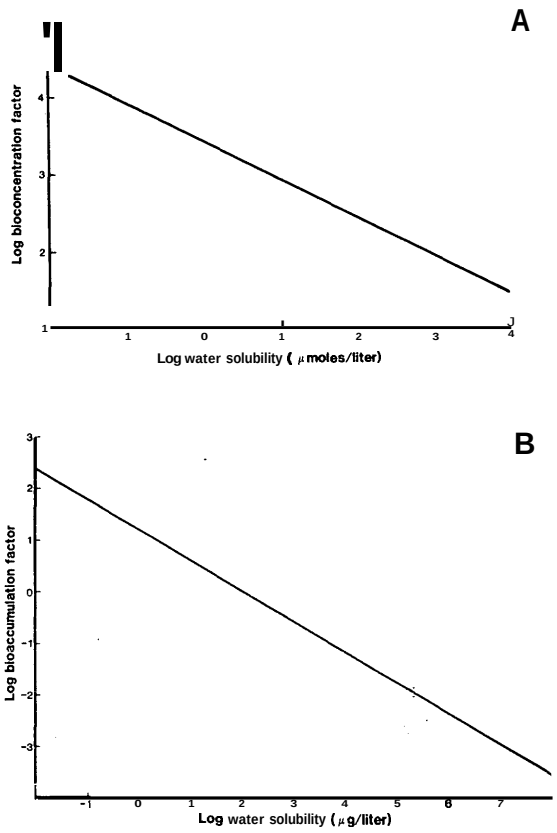



Figure 3.--The relation of water solubility to bioaccumulation.

A. Aqueous solubilities and bioconcentration factors of organic chemicals in rainbow trout. Log bioconcentration factor = 3.41 - 0.508 log water solubility, $r^2$ = 0.93. The bioconcentration factor is the concentration of a chemical in fish divided by its concentration in water (from fig. 2, Chiou et al. 1977).

B. Aqueous solubilities and bioaccumulation in adipose tissue of rats. Log bioaccumulation factor = 1.20 - 0.56 log water solubility, $r^2$ = 0.64. The bioaccumulation factor is the concentration of a chemical in adipose tissue divided by its concentration in the diet (from fig. 1, Geyer et al. 1980).

## APPROACHES TO TOXIC HAZARD ASSESSMENT

Several specific methods have been used to evaluate toxic hazards for aquatic species. Most have used a specified fraction (expressed as a decimal) of the $LC_{50}$ (or similiar measure of response) as an estimate of the no-toxic-effect exposure level (National Academy of Sciences-National Academy of Engineering 1973, U.S. Environmental Protection Agency 1976). This fraction is often called an application factor or safety factor. Where only acute exposures and survival were the primary interest, the estimates of no-toxic-effect level ranged from 0.1 to 0.05($LC_{50}$) (Sprague 1971). For compounds that were more persistent in the environment or when estimates of chronic exposures were desired, the estimate of the no-toxic-effect level ranged from 0.1 to 0.01($LC_{50}$) (Sprague 1971). These methods were popular because the concepts were easy to understand and apply. The methods relied, however, on an assumption that exposure was continuous at the specified level for a long period (usually 96 or more hours). This rationale is perhaps acceptable for large streams receiving a steady input of pollutants or for a specific pollutant point-source, but it does not work well for forest streams where the concentration of pollutant changes rapidly.

A more refined and realistic method has been published (U.S. Environmental Protection Agency 1980) that requires substantial data that define no-effect levels for a variety of aquatic species. In addition, the method provides procedures that give both an instantaneous maximum permissible concentration and a 24-hour average permissible concentration. The EPA procedure is a considerable improvement over earlier methods because it recognizes and allows for variable levels of exposure. It is hampered, however, by a paucity of well-defined, no-effect data bases for many compounds. For the purposes of hazard assessment in this paper, we have selected an approach that combines these two approaches. We have used fractional $LC_{50}$ values as the basis for estimating no-effect concentration values and integrals of the time-concentration curves of pollutants as measured in forest streams to estimate exposure. This approach is described more specifically in a later section on hazard assessment. The next section (the behavior and toxicity of commonly used forest chemicals) provides the data on toxicity and exposure that we use in a later section (hazard assessment) to relate toxicity to exposure and thereby derive estimates of the margin of safety.

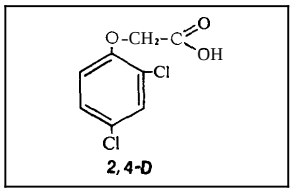
## BEHAVIOR AND TOXICITY OF COMMONLY USED FOREST CHEMICALS

The behavior (movement, persistence, and fate) of a chemical in the environment determines, in large measure, the likelihood and the nature of the exposure organisms will receive. Leonard et al. (1976) made an intensive review of this subject for many pesticides. Although their emphasis was on agriculture, many of the concepts and some of the data are relevant in forestry. In this section, we review what is known about the physical-chemical properties, movement and persistence in soil, entry and fate in forest waters, bioaccumulation, and toxicity to aquatic species of seven herbicides, five insecticides, urea fertilizer, and the ammonium-based fire retardants. These specific materials were selected for review because they are (or are likely to be) the most widely used materials in their class in forestry in the United States. The common and scientific names of organisms mentioned in this paper are in appendix tables 19 (fish) and 20 (invertebrates).

Information on rates and methods of application and carriers for pesticides are in the "Oregon Weed Control Handbook" (Whitesides 1981), the "Oregon Insect Control Handbook" (Capizzi and Fisher 1977), and "Pesticide Uses for Forestry."[3/]

## HERBICIDES

### 2,4-D



2,4-D

2,4-D is one member of a large family of phenoxy herbicides which have been reviewed by the National Research Council of Canada (1978) and Norris (1981). The most extensively used herbicide in forestry, 2,4-D is formulated as water-soluble amine salts for direct stem injection or as esters that are usually dissolved in diesel oil or emulsified in water for aerial or ground application to foliage or bark. Rates of application between 1.12 and 4.48 kg/ha are common. More specific information on the use of this herbicide is reviewed by National Forest Products Association (see footnote 3) and Newton (1981).

#### Behavior in the Environment

The physical-chemical properties of the acid, salt, and ester forms of 2,4-D are pertinent because the herbicide may be in the environment in any of these forms. It is usually applied as the ester, but is rapidly hydrolized under most circumstances to either the acid or the salt form, depending on the pH of the environment (Paris et al. 1975, National Research Council of Canada 1978, Norris 1981).

The acid of 2,4-D is soluble to about 900 mg/liter at 25 °C in water. The dimethylamine salt of 2,4-D is extremely soluble in water (300 g/100 g) and other polar solvents, such as alcohols and ketones, but it has low solubility in kerosene and diesel oil. Many 2,4-D esters are available; those commonly used in forestry are low in water solubility (less than 500 mg/liter) but are very soluble in organic solvents and oils. The acid and salt forms of 2,4-D have negligible vapor pressure, which means they are not very volatile. The vapor pressure of esters varies from $10^{-2}$ mmHg (high-volatile esters) to $10^{-6}$ mmHg (low-volatile esters).

The methyl, ethyl, propyl, isopropyl, butyl, and amyl esters are called high-volatile esters. They are not used in forestry. Propylene glycol butyl ether (PGBE), isooctyl, butoxyethyl, 2-ethyl hexyl, and propylene glycol esters (and others of similar properties) are called low-volatile esters and are commonly used in forestry. House et al.,[4/] National Research Council of Canada (1978), and Weed Science Society of America (1979) review the physical-chemical properties of 2,4-D in more detail.

2,4-D persists in soil for only short periods. Research reviewed by House et al. (see footnote 4) indicates microbial decomposition is the predominant process of 2,4-D disappearance from soil. Environmental factors that favor rapid microbial metabolism also favor the disappearance of 2,4-D from forest floor and soil. Recent research reviewed by National Research Council of Canada (1978) and Norris (1981) support these conclusions.

---

[3/] Unpublished report, "Pesticide uses for forestry," prepared by Natl. For. Prod. Assoc., Washington, D.C., 1980.

[4/] Unpublished final report, "Assessment of ecological effects of extensive or repeated use of herbicides," by W. G. House, L. H. Goodson, H. M. Gadberry, and K. W. Dockter, Contract DAHC 15-68-C-0119, Advanced Res. Proj. Agency, Dep. Defense, Midwest Res. Inst. Proj. 3103-B, Kansas City, Mo., 1967.

In laboratory studies with forest-floor material, Norris (1966, 1970a) and Norris'and Greiner (1967) report half lives for 2,4-D that range from 10 to 20 days. In the field, 2,4-D disappeared completely from an oak-forest soil in 30 days (Altom and Stritzke 1972), 90 percent in 31 days in a Minnesota forest,[5/] and 40 percent in 15 days from a southern California chaparral site (Plumb et al. 1977).

2,4-D is extensively adsorbed by soil organic matter (Norris 1970b), which tends to reduce its mobility in soil. In light, sandy soils with a high pH, however, it may show substantial mobility (see footnote 4). Forest soils are usually high in organic matter and low in pH, which will inhibit the mobility of 2,4-D. In field studies, 2,4-D residues are not normally found deeper than 20 or 30 cm, even after prolonged periods of heavy precipitation (Altom and Stritzke 1972, Plumb et al. 1977, Stewart and Gaul 1977, Norris et al. 1982, see footnote 5).

Norris (1981) reviewed the entry and fate of 2,4-D (and the other phenoxy herbicides) in forest waters. He concluded direct application and drift to surface waters are the processes most likely to produce the highest residue levels, but the persistence is brief. Mobilization of residues from ephemeral stream channels may also introduce 2,4-D to forest stream systems, but the concentrations are not likely to exceed the concentration resulting from direct application or drift.

Norris (1967) reported maximum concentrations of 2,4-D ranging from 0.001 to 0.13 mg/liter during and shortly after application. The time required to return to nondetectable levels (less than 0.001 mg/liter) varied with the nature of the area and the maximum concentration observed. Times ranging from less than 1 hour to more than 168 hours have been noted, with less than 2 days usually required.

Application to marshy areas can lead to higher than normal levels of stream contamination. In one instance, concentrations approaching 0.9 mg/liter 2,4-D were found in water flowing from a marshy area. Long-term outflow of 2,4-D has not been noted. Once the initial stream concentration declined to nondetectable levels, no 2,4-D residues were found during subsequent periods of heavy precipitation the first fall after application (Norris 1967, 1968). Norris (1969) and Norris et al. (1982) reported heavy precipitation will mobilize any surface residues of 2,4-D that are present in ephemeral stream channels.

Few quantitative studies of 2,4-D discharge for whole watersheds have been conducted. Norris et al. (1982) reported 0.014 percent of the 2,4-D (4.6 kg/ha) applied to a 7-ha watershed appeared in streamflow, all in a 12-day period during the first heavy storm after application (about 4 months after application). Suffling et al. (1974) noted less than 0.001 percent loss of 2,4-D applied to a powerline right-of-way in a similar study.

Results of monitoring operational applications of 2,4-D are largely in agreement with research findings. The U.S. Department of Agriculture, Forest Service,[6/] analyzed the results of monitoring phenoxy herbicides in streams after operational application

---

[5/] Unpublished monitoring report, FY1977 aerial herbicide project, prepared by U.S. Dep. Agric., For. Serv., Chippewa Natl. For., Cass Lake, Minn., 1977.

[6/] Memorandum, "Summary of phenoxy herbicides in water," (2150, Pesticide Use Management), from F. 3. Kopechky to the Chief, U.S. Dep. Agric., For. Serv., June 23, 1980.

in Pacific Northwest forests. Results of analyses of 680 samples taken in connection with 304 different applications in 4 years were summarized. They reported that of the 304 applications monitored, 84 percent did not result in detectable stream contamination, 14 percent had residues up to 0.005 mg/liter, 1 percent had between 0.005 and 0.01 mg/liter, and 1 percent exceeded 0.01 mg/liter (maximum residue was 0.040 mg/liter).

Few field data are available on 2,4-D levels in sediment or aquatic species in forest streams. The fate of 2,4-D in forest streams has not been determined, but we believe downstream movement, adsorption, and degradation (processes observed in other aquatic systems) result in a reduction in both the concentration and total level of 2,4-D in aquatic systems. Gangstad[7/] reviewed the fate of 2,4-D in aquatic systems where the herbicide is used to control aquatic weeds. He cited the research of several investigators in noting that the concentration of 2,4-D in hydrosoil or fish flesh did not usually exceed the concentration in water, and disappearance from the system in general was rapid (nearly 100-fold reduction in concentration in 29 days in ponds). Streit (1979) reported 2,4-D was not found on aquatic sediments at concentrations greater than in the water. The sediments in this study ranged from 2.3 to 31.9 percent organic matter. Nesbitt and Watson (1980) noted numerous factors influenced the persistence of 2,4-D in an Australian river. The number of live bacteria, nitrogen and phosphorus concentrations, sediment levels, and temperature were important.

Bioaccumulation is most likely to occur when organisms are exposed to persistent chemicals that have low water solubility and high lipid solubility. 2,4-D does not meet these criteria to the same degree as do the chlorinated hydrocarbon insecticides. Organisms exposed to phenoxy herbicides will take up some of the chemical, but generally the bioaccumulation ratio will be low and the residence time will be brief, once exposure ceases. Bohm and Mueller (1976) reported an accumulation factor of 2.2 for the alga, Scenedesmus, exposed to 0.022 mg/liter for 8 hours. Sanborn (1974) did not detect unmetabolized 2,4-D in the components of an aquatic-terrestrial model ecosystem. Schultz (1973) found less than 0.005 mg/kg 2,4-D in three species of fish exposed to 2,4-D (2.5 mg/liter) for periods of 4, 7, or 14 days, although unspecified metabolic products ranged from 0.031 to 0.122 mg/kg. As part of a widespread survey of the Swedish environment for phenoxy herbicides, Erne (1975) reported only 3 percent of 330 samples of muscles from healthy fish (several species from 120 locations) contained detectable residues of 2,4-D (residues ranged from 0.05 to 1.5 mg/kg). In an aquatic area treated wih 1.2 kg/ha 2,4-D (granule form), mussels contained 0.38 to 0.70 mg/kg 2,4-D, reflecting their consumption of algae that had adsorbed the herbicide. Fish, on the other hand, did not contain detectable residues (less than 0.04 mg/kg) (Smith and Isom 1967). Schultz and Whitney (1974) reported 2,4-D residues that ranged from undetectable to 0.162 mg/kg in a variety of fish species. About 80 percent did not contain detectable residues. A stream gastropod did not concentrate 2,4-D from water (0.0002 to 0.05 mg/liter 2,4-D in water) during a 24-hour exposure period (Streit 1979). Sigmon (1979) reported no measurable residues (less than 0.05 mg/kg) of 2,4-D in bluegills after 8 days in water containing 3 mg/liter 2,4-D (butoxyethyl ester). Rodgers and Stallings (1972) noted 2,4-D and its metabolites were rapidly eliminated from fish after exposure ceased. Extensive data from

---

7/ Unpublished draft report, "2,4-D herbicide residues in treated waters," by E. O. Gangstad, U.S. Dep. Agric.-U.S. Dep. Inter., Weed Control Comm., Washington, D.C., 1979.

Ellgehausen et al. (1980) support these findings. The lack of bioaccumulation evident in these results is consistent with the physical–chemical properties of the herbicide.

## Toxicity

A review of the toxicity to fish of 2,4-D herbicides indicates they vary (with 96-hour $LC_{50}$'s ranging from less than 1 mg/liter to more than 400 mg/liter), depending on formulation (National Research Council of Canada 1978). Most studies emphasized lethal concentration of the compounds, carried out by static bioassays; their field applicability is thus somewhat limited. The test animals used in most studies have been bluegills, a species generally considered less sensitive than salmonids.

A review of the toxicity of 2,4-D dimethylamine (DMA) herbicides indicates it is relatively low to fish. Folmar (1976) reported a 96-hour $LC_{50}$ for rainbow trout at 100 mg/liter, but he noted avoidance reaction well below the 96-hour $LC_{50}$ value. Davis and Hughes (1963) and Hughes and Davis (1963) tested the toxicity of different formulations of 2,4-D to bluegills. They found considerable variation in the toxicity of different formulations and even in the toxicity of a single formulation. The researchers believed these inconsistencies could be attributed to the different batch lots and manufacturing plants. The alkanolamine salt and the dimethylamine formulations were the least toxic (800 and 166 mg/liter, respectively, as 48-hour $LC_{50}$, depending on batch) of 11 formulations of 2,4-D tested on bluegills. The isopropyl ester and butyl ester were the most toxic (0.8 and 1.3 mg/liter as 48-hour $LC_{50}$, respectively, for bluegills). These esters are not used in forestry. Davis and Hardcastle (1959) found differences in $LC_{50}$ values for 2,4-D and other herbicides when waters from two different sources were used in toxicity tests.

Walker (1964a) compared toxicity of formulations of 2,4-D to several fish species and found that the butyl, PGBE, and butoxyethanol esters were most toxic (96-hour $LC_{50}$ less than 4 mg/liter), and the sodium salt and isooctyl ester were less toxic ($LC_{50}$ 66 and 160 mg/liter, respectively). He also reported a 96-hour $LC_{50}$ of about 90 mg/liter for a wetting agent (a sodium borate mixture).

In a comprehensive study, Meehan et al. (1974) tested the toxicity of various formulations of 2,4-D to salmonids and found that less than 50 mg/liter 2,4-D acid produced no mortality except in pink salmon fry. The butyl ester, however, was very toxic, causing almost complete mortality in all Species at concentrations greater than 1 mg/liter. The isooctyl ester was the least toxic of the ester formulations, but caused significant mortality in fry of several species at 1 and 5 mg/liter. The authors did note, however, that the butyl and PGBE ester formulations of 2,4-D posed a more significant hazard to fish than the isooctyl ester if direct application of the chemical to the water surface occurred. No-effect levels (survival) of the various formulations varied from less than 1 mg acid equivalents (a.e.) per liter for all ester formulations to 50 mg/liter for the acid.

Lorz et al. (1979) observed no mortality of yearling coho salmon exposed to 200 mg/liter of 2,4-D (DMA) for 144 hours, nor did they observe any subsequent effects when these fish were tested in seawater.

Sublethal effects of PGBE esters of 2,4-D have been demonstrated for fish (Cope 1966). Spawning of bluegills was delayed 2 weeks in ponds treated with 5 and 10 mg/liter of the herbicide. Hiltibran (1967) observed that fertilized eggs of green sunfish

developed normally when exposed to 1 mg/liter of the PGBE ester of 2,4-D under static water conditions. Bluegills, green sunfish, lake chubsuckers, and smallmouth bass fry, however, appeared to be more susceptible to the herbicide, they failed to survive the 8-day duration of the test.

Woodward/ found 96-hour $LC_{50}$ values of 0.6 to 1.0 mg/liter with PGBE ester of 2,4-D for cutthroat trout fry. He did not find that toxicity was affected by pH or water hardness. Woodward and Mayer (1978) noted that survival of cutthroat trout alevins was significantly reduced when they were continuously exposed to concentrations of 0.06 and 0.124 mg/liter 2,4-D PGBE ester. The authors suggested that the no-effect concentration for cutthroat trout was 0.03 mg/liter 2,4-D PGBE ester.

Much of the work on fish toxicity of the phenoxy herbicides concerns the PGBE esters of 2,4-D or 2,4,5-T, but little has been done on mixtures of these compounds. Hughes and Davis (1963) found both the 24- and 48-hour $LC_{50}$'s for bluegills exposed to the PGBE ester of 2,4-D were 2.1 mg/liter under static conditions in water with a mean pH of 6.9 and mean hardness of 29 mg/liter. Meehan et al. (1974) observed that the 96-hour no-effect level (survival) for coho salmon fingerlings exposed to the PGBE ester of 2,4-D was less than 1 mg/liter. A mean fry mortality of 26.7 percent occurred after the 96-hour exposure to 1 mg/liter of the herbicide in water that ranged in hardness from 10.0 to 33.6 mg/liter as calcium plus magnesium. A 48-hour $LC_{50}$ of 1.1 mg/liter PGBE ester of 2,4-D was reported by Cope (1966) for rainbow trout (no water quality given). Butler (1965) observed that the 48-hour $LC_{50}$ for the estuarine longnose killifish was 4.5 mg/liter in seawater.

Cope et al. (1970) observed bioconcentration of the PGBE ester of 2,4-D in fish tissues 1 to 3 days after treatment. No detectable residues of the herbicide were found after 4 days in bluegills exposed to 10 mg/liter PGBE ester of 2,4-D. Histological and biochemical changes were also observed in bluegills exposed to 2,4-D (PGBE ester) at and above 5 mg/liter in ponds in Oklahoma (Cope et al. 1970). The pathology involved liver, vascular system, and brain, with depletions of liver glycogen, globular deposits in the blood vessels, and stasis and engorgement of the brain circulatory system.

No deaths occurred in yearling coho salmon exposed to PGBE ester 2,4-D plus 2,4,5-T for 96 hours at equal to or less than 0.800 mg/liter (nominal concentrations, static) or 0.21 mg/liter (nominal concentration in flow-through exposure tanks (Lorz et al. 1979)). In both the static and flow-through systems, the amount of herbicide recovered was very low even though extra care was taken in mixing.

Matida et al. (1975) noted that when a mixture of 2,4-D and 2,4,5-T as the butoxyethanol ester (commercially called "Brush Killer") was aerially spread over 9.5 ha of forest at a rate of 4.05 kg/ha 2,4-D and 1.95 kg/ha 2,4,5-T active ingredient, no appreciable change was noted in the aquatic community. The authors were unable to detect the chemical in the stream during the 48-hour observation period after spraying. Similarly, fishes (cherry salmon and dace (genus not identified) fingerlings) showed no mortality or abnormal behavior, and the standing crop of invertebrates appeared unchanged. In a later laboratory study, Matida et al. (1976) found that a mixture of 2,4-D and 2,4,5-T produced toxic effects on aquatic isopods (Asellus hilgendorffii), cherry salmon fry, and dace fingerlings. The 96-hour $LC_{50}$ for the groups tested were 1.6 (isopod), 0.6 (cherry salmon), and 1.3 (dace) mg/liter. Exposures of cherry salmon fingerlings to concentrations

---

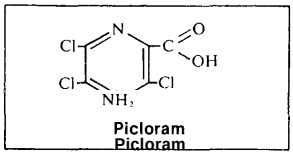
8/ Personal communication, D. Woodward, U.S. Dep. Inter., Fish and Wildl. Serv., Fish-Pestic. Res. Unit, Jackson, Wyo., 1977.

of 0.47 and 0.62 mg/liter "Brush Killer" for 96 hours caused histological changes of liver parenchyma, which the authors considered a nonspecific response to a toxic agent.

Johnson and Finley (1980) summarized the results of the studies (1965–78) of the USDI Fish and Wildlife Service, Columbia, Missouri, laboratory. They provide a useful table of acute-toxicity values of various formulations of 2,4-D to a variety of invertebrate and fish species.

Sanders (1969) studied the effect of several 2,4-D formulations on the amphipod Gammarus lacustris. The butoxyethanol ester was most toxic--with a 24-hour $LC_{50}$ of 1.4 mg/liter--followed by the PGBE ester (2.1 mg/liter), and the isooctyl ester (6.8 mg/liter). The dimethylamine salt was not toxic at 100 mg/liter (96 hours). In a later study, Sanders (1970) showed the variable toxicity of several 2,4-D formulations to various crustaceans. The PGBE esters were generally most toxic, followed by the butoxyethyl ester formulations. 2,4-D-dimethylamine (DMA) was the least toxic. Crayfish were reported to be less sensitive in this test than Daphnia, seed shrimp, glass shrimp, scuds (amphipods), and sowbugs (isopods). Schultz and Harman (1974) published an excellent review of the literature on the use of 2,4-D in fisheries as it relates to toxicity, residues, and effects on organisms.

## Picloram



Picloram
Picloram

Picloram is a broad-spectrum herbicide used for control of a wide variety of woody annual and perennial broadleaf weeds. It is available in both salt and ester formulations, but the most common used in forestry is either a potassium or amine salt. It is often applied in combination with 2,4-D (Weed Science Society of America 1979). Picloram may be applied as pellets or, more commonly, as a diluted spray mixture. Rates of application range up to 1.12 kg/ha. Picloram may also be used in stem-injection treatments. National Forest Products Association (see footnote 3) and Newton (1981) review uses of picloram in forestry.

### Behavior in the Environment

Amine and potassium salts of picloram are highly water soluble and have negligible vapor pressure (less than $10^{-6}$ mmHg). The physical-chemical properties of picloram are reviewed in detail by the National Research Council of Canada (1974) and the Weed Science Society of America (1979).

Picloram is both persisent and mobile in soil. These characteristics are reviewed in detail by House et al. (see footnote 4), Goring and Hamaker (1971), and National Research Council of Canada (1974). Norris (1970a, b) notes, however, that picloram is adsorbed by organic matter and is degraded by microbial action. In the forest environment, which is characteristically high in organic matter and low in pH, substantially less mobility and persistence have been found than in several agricultural environments.

Movement of the herbicide in the soil profile is governed by the net water flow, with maximum losses occurring under warm, humid conditions, after heavy rainfall, and in light soils that are low in organic content. The removal of picloram through rainfall and leaching is one of the major factors governing its dissipation under field conditions (National Research Council of Canada 1974). This mobility is also of environmental concern, because leached picloram may be transported to aquatic ecosystems. Residues in surface runoff have reached 2 mg/liter after application at 1.1 kg/ha (National Research Council of Canada 1974). Studies have indicated, however, that under most conditions only small proportions of picloram (less than 5 percent) applied to a watershed are transported in surface runoff.

Norris et al. (1976) determined the persistence and leaching characteristics of both picloram and 2,4-D at several sites on power transmission line rights-of-way in Oregon and Washington. Study sites ranged from zones of low to high temperature and rainfall. Both herbicides showed a rapid decline in concentration after application. Biologically significant residues were seldom present more than 12 months after application and no leaching of herbicide below 39 cm was detected (relatively little herbicide was detected below 15 cm). When a forest floor was present, nearly all the herbicide was found in this layer. At another site, Norris et al. (1982) reported picloram and 2,4-D disappeared from soil within 29 months with no significant leaching in the soil profile.

An extensive monitoring effort for picloram and 2,4-D in forest streams flowing across powerline rights-of-way treated with these herbicides failed to show measurable levels of chemicals in streams. In several cases, intensive sampling was done with automatic equipment for periods in excess of 6 months after application (Norris et al. 1976).

Where soil compaction has occurred or where ephemeral streams have been treated, surface residues of picloram may occasionally be mobilized. Norris (1969) reported a maximum concentration of 0.078 mg/liter in surface runoff water from a powerline right-of-way during the first fall rains after application. In a similar situation, Suffling et al. (1974) reported 0.038 mg/liter picloram in runoff the first day after application. In an Arizona watershed study, Johnsen (1980) found a peak concentration of picloram of 0.320 mg/liter in the first flow in an ephemeral stream 157 days after application (2.8 kg/ha to 113 ha of the 146-ha watershed). The concentration decreased with time (less than 0.1 mg/liter after 207 days, less than 0.01 mg/liter after 506 days, last detectable residue at 915 days). A total of 1.1 percent of the picloram applied was discharged in streamflow, 89 percent in the first spring runoff period after application.

In a rangeland setting where picloram was used to control streamside vegetation, residues as high as 0.37 mg/liter were found during the first two storms after application. Subsequent samples did not contain more than 0.05 mg/liter (Davis et al. 1968). Norris et al. (1982) found a similar pattern at a more extensively studied area. In both studies, the concentrations were highest with the first runoff events and decreased rapidly. At one site, 0.35 percent of the picloram applied to a 7-ha watershed was discharged in stream water in the 7 months between the time of application and the time the last sample containing herbicide was collected. All herbicide discharge occurred during the first storms, sufficient to generate streamflow, after application. Suffling et al. (1974) found about 0.22 percent of the picloram applied to a Great Lakes forest opening (a powerline right-of-way) was contained in runoff water in the first year after application.

Only negligible residues of picloram occur in streams in treated areas; apparently the herbicide is rapidly diluted (Haas et al. 1971). Field plots adjacent to a small stream were treated with 1.1 kg/ha of picloram, and water samples were collected 0, 0.8, and 1.6 km downstream from the plots after each rain for 5 months after application. Picloram was detected (0.029 mg/liter) in stream samples only during the first significant runoff. No residues were found in subsequent samples (Haas et al. 1971).

Picloram contamination in lakes has not been reported, but levels in farm ponds adjacent to plots treated with 1.1 kg/ha picloram reached 1 mg/liter (National Research Council of Canada 1974). Dissipation of the herbicide in ponds appears to be rapid. One study found an initial decline of 14 to 18 percent of the picloram per day, followed by a decline of less than 1 percent per day 15 weeks after application (Haas et al. 1971). Residues of picloram (148 µg/kg) in pond sediments immediately after application were only twice that in the water, according to Kenaga (1973, as cited in National Research Council of Canada 1974). After 75 days, 7 µg/kg picloram was detected in the pond sediments and 0.1 µg/kg picloram was found in the water. Dennis et al. (1977) measured picloram residues in water and sediment from ponds and streams after extensive use of picloram for control of woody vegetation on pastures in West Virginia. Picloram residues reached higher levels in water in ponds (up to 0.437 mg/liter) than in streams (up to 0.011 mg/liter), although the levels generally decreased with both time and distance from the treated area. Generally, residues were higher in the water than in the sediment in both streams and ponds. No picloram was detected in stream sediments despite concentrations in water that ranged from nondetectable to 0.011 mg/liter.

Johnsen and Warskow (1980) injected picloram and 2,4-D into a small stream (stream-discharge volume was 0.036 $m^3$/second) for 50 minutes to achieve a concentration of 6.26 mg/liter picloram. The highest concentration outside the treatment zone was 2.4 mg/liter at the first sampling station, 0.4 km downstream. Peak concentrations at other downstream locations were 0.94 mg/liter at 0.8 km; 0.32 mg/liter at 1.6 km; 0.014 mg/liter at 3.2 km; and 0.001 mg/liter at 6.4 km. The herbicide was not detected after 2 days. Stream water, originally containing 1.280 mg/liter picloram, contained only 0.544 mg/liter (a 57-percent reduction) after exposure to direct sunlight for 8.8 hours.

The physical-chemical properties of picloram are not compatible with extensive bioaccumulation. The high water solubility of picloram and its low lipid solubility suggest it will be rapidly excreted as exposure decreases. Residue analyses of aquatic organisms exposed to picloram indicate that this herbicide is not bioconcentrated in invertebrates or other food-chain organisms (National Research Council of Canada 1974). Daphnia exposed to 1 mg/liter of the potassium salt of picloram had whole-body residues of the herbicides equal to that present in the water (Hardy 1966). Bioconcentration of picloram (acid) was not evident in mosquitofish exposed to 1 mg/liter (a.e.) for 18 days (Youngson and Meikle 1972, as cited in National Research Council of Canada 1974). The concentration factor for these fish on a wet-weight, whole-body basis was only 0.02. The 18-day exposure to picloram was adequate to achieve a steady-state level of accumulation in the mosquitofish. Kenaga (1980a) reported a bioconcentration factor of 31 in a flowing water system compared to a factor of 0.02 in a static system.

## *Toxci*

The toxicity of picloram to fish is influenced by its formulation and the quality of the water (Sergeant et al. 1970, Woodward 1976). Technical-grade picloram (active ingredient 90 percent) was found to be more toxic under alkaline conditions (Woodward 1976) than under nonalkaline conditions. Increasing the pH from 6.5 to 8.5 increased the toxicity to cutthroat and lake trout by a factor of 2 in both species. Increasing temperature led to an increase in toxicity, but increasing hardness did not (Woodward 1976).

The acute toxicity of picloram varies considerably with the formulation and fish species. The isooctyl ester of picloram appears to be the most toxic commercial formulation (Kenaga 1969, Sergeant et al. 1970, National Research Council of Canada 1974). $LC_{50}$'s reported for this formulation are about 1 mg/liter for sensitive species. Tordon 22K (potassium salt) is considerably less toxic to several fish species (table 7).

Table 7--Mortality data for several fish species exposed to Tordon 22K for 96 hours[1/]

| Fish species | Water temperature | Concentration (acid equivalents) | Mortality/ |
|---|---|---|---|
| | Degrees C | Milligrams/liter | Percent |
| Black bullhead | 10 | 91 | 50 |
| | 10 | 69 | 0 |
| Bluegill | 18 | 5.4 | 50 |
| Brook trout | 10 | 91 | 50 |
| | 10 | 69 | 0 |
| Brown trout | 10 | 52 | 50 |
| | 10 | 22 | 0 |
| Fathead minnow | 10 | 29 | 50 |
| | 10 | 22 | 0 |
| Green sunfish | 10 | 91 | 50 |
| | 10 | 39 | 0 |
| Lake emerald shiner | 21-26 | 30 | 50 |
| Rainbow trout | 10 | 58 | 50 |
| | 10 | 22 | 0 |

[1/] Modified from Kenaga (1969).
[2/] 50-percent mortality indicates the calculated or derived 96-hour $LC_{50}$ values: 0-percent mortality indicates the highest concentration producing no mortality.

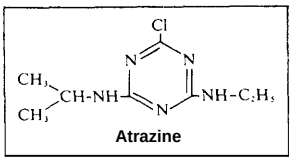
Green sunfish exposed to the 99-percent analytical-grade picloram (1.2 mg/liter a.e.) were not affected, but the technical-grade or the 22-percent commercial formulation of picloram (for up to 1 hour) caused immobilization but not death (Sergeant et al. 1970). Recovery of normal swimming response followed transfer to clean pond water. Two subsequent exposures to the herbicide shortened the recovery times; after a fourth exposure, however, many of the fish failed to recover. Analytical-grade picloram did not affect swimming behavior of green sunfish. Sergeant and coworkers suggested that technical-grade and commercial formulations of picloram might contain a toxic impurity.

Based on available information, chronic picloram toxicity to fish is not cumulative in terms of lethality (National Research Council of Canada 1974, Woodward 1976). Long-term exposures, however, have been shown to affect fish development and growth (Woodward 1976) and swimming response and liver histopathology (Sergeant et al. 1970). Woodward (1976) observed that the no-effect concentration of technical-grade picloram for lake trout was apparently less than 0.035 mg/liter, because this level of herbicide reduced fry survival and growth. Most mortalities occurred during yolk absorption, which took 4 to 5 days longer in picloram-treated fish.

In a long-term experiment (10 weeks), Hardy (1966) found that growth and reproduction of Daphnia were unaffected by 1.0 mg/liter of picloram. Lynn (1965) reported that Daphnia survived a 24-hour exposure to 380 mg/liter of Tordon 101, but 95 percent were killed at a concentration of *530* mg/liter. Sanders and Cope (1968) found the 24-hour $LC_{50}$ for stonefly nymphs (Pteronarcys californica) exposed to picloram (technical, emulsifiable concentrate) was 120 mg/liter, but the 96-hour $LC_{50}$ was 48 mg/liter. Johnson and Finley (1980), using picloram (technical material 90 to 100 percent), give the 96-hour $LC_{50}$ of Gammarus fasciatus and Pteronarcys (2d-year class) as 0.027 and 0.048 mg/liter, respectively.

Lorz et al. (1979) estimated the 24-hour $LC_{50}$ of Tordon 22K and Tordon 101 (a 4:1 mixture of 2,4-D:picloram) as 17.5 and 20 mg/liter, respectively, for yearling coho salmon. When the survivors were challenged with seawater, some of the groups that had received the lowest herbicide concentration suffered mortalities to 70 percent. Reasons for the deaths in seawater after low herbicide exposure are unknown. When coho salmon yearlings were exposed for 96 and 360 hours to Tordon 101 and then released into a small coastal stream, their downstream movement was generally inhibited except for the groups receiving the lowest concentration (0.3 mg/liter). In well-planned spray operations in forestry, similar concentrations (those that might cause inhibition of migration) are unlikely to occur in streams.

## Atrazine



Atrazine

Atrazine is one of a large group of compounds called triazine herbicides. It is widely used as a selective herbicide for control of broadleaf and grassy weeds in both agriculture and forestry at rates up to 4.48 kg/ha. At higher rates of application, it can be used for nonselective control of vegetation on noncroplands. National Forest Products Association (see footnote 3) and Newton (1981) reviewed the use of atrazine in forestry. An extensive review of the triazine herbicides is included in a special volume of "Residue Reviews'* (Gunther and Gunther 1970).

### Behavior in the Environment

Atrazine has fairly low solubility in water (33 mg/liter) with substantial solubility in several organic solvents (chloroform, 52 000 mg/liter; methanol, 18 000 mg/liter; diethyl ether, 12 000 mg/liter). Although its vapor pressure is low ($3x10^{-7}$ mmHg), it is reported to volatilize from both vegetation and soil surfaces (Kearney et al. 1964, Burt 1974).

At normal rates of application, most of the atrazine disappears within a year of application. Axe et al. (1969) found only 33 percent of the atrazine remained in soil 5 days after application. Birk and Roadhouse (1964) reported 90-percent loss of atrazine within 1 year in agricultural soils. In the same study, they found 85 percent of the atrazine was in the top 2.5-cm layer of soil and 5.7 percent in the 2.5- to 5.0-cm soil layer after 21 cm of rain had fallen. Marriage et al. (1975) reported no significant accumulation of atrazine even after annual applications of *4.5* kg/ha in nine consecutive years.

Measurable residues of atrazine were confined to the upper 15 cm of the soil profile, with the vast majority of the herbicide in the 0- to 5-cm soil layer.

Atrazine losses in runoff water and soil sediment have been measured on agricultural lands. Hall et al. (1972) reported atrazine losses in runoff ranging from 0.01 to 5.0 percent of the applied atrazine within the first season after application. About 90 percent of the loss occurred within the 1st month after application. The magnitude, frequency, and intensity of precipitation were important factors in determining the amount of atrazine in runoff. Runoff of water in this study ranged from 17 to 68 percent of the incident precipitation, resulting in loss of as much as 10 000 kg of soil/ha (silty clay loam soil, 14-percent slope). In two small (1.3- and 1.4-ha) agricultural watersheds in Georgia, 0.2 and 1.9 percent of the atrazine applied were recovered in storm-generated runoff during the first 90 days after application (1.45 and 4.03 kg/ha). The first runoff events were 6 and 24 days after application. Most of the atrazine recovered (83 and 99 percent) was in solution (Leonard et al. 1979).

Frank and Sirons (1979) monitored streams in 11 agricultural watersheds (average size 4279 ha) for atrazine in both water and sediment. The herbicide or its metabolites were found in 80 percent of the streams with a mean concentration of 0.0014 mg/liter. Peak concentration did not exceed 0.032 mg/liter. About 62 percent of the atrazine discharge was associated with storm runoff, 21 percent in base flow, and an additional 22 percent as the result of chemical spills. Atrazine was detected in stream-bottom sediments in 4 of 10 sets of samples (concentrations ranged from not detected to 20 mg/kg).

Smith et al. (1975) analyzed water samples from irrigation ditches and basins that had been sprayed with atrazine when ditches were dry. After the ditches had been filled twice, no residues of atrazine were detected in the water. These results indicate mobilization of atrazine in ephemeral stream channels is most likely to be restricted to the first few significant storms after application. Weidner (1974) noted a significant degree of atrazine degradation in ground water, although the rate of degradation was slower than would be expected for the same herbicide in soil. Maier-Bode (1972) reported that initial concentrations of atrazine of 0.5 mg/liter in water decreased by 90 percent within 2 weeks and 99 percent within 8 weeks in experimental fish ponds. Herbicide concentrations in the sediment 4 days and 8 weeks after application were 180 percent and 50 percent of the respective initial concentrations in water. Newton[2/] collected water samples from a small stream draining a Christmas tree plantation; he reported atrazine residues in water ranging from 0.42 mg/liter 10 minutes after application (3.0 kg/ha) to 0.02 mg/liter 17 days later. Heavy rain 3 days after application increased the concentration to 0.21 mg/liter on the 4th day, but it had decreased to 0.04 mg/liter by the next day, presumably reflecting decreased loading of silt with adsorbed atrazine.

---

/Personal communication, M. Newton, For. Res. Lab., Corvallis, Oreg., 1967.

Based on its physical–chemical properties, atrazine would be expected 'to show little'tendency for bioaccumulation. Böhm and Mueller (1976) noted that increasing water solubility of the herbicide resulted in a decrease in the absolute level of the herbicide in algae. The accumulation factor was 31.8. After a transfer of contaminated algae into an atrazine–free medium, the herbicide was rapidly desorbed, except for about 10 percent of the residue, which was apparently bound irreversibly to cell structures. Streit (1979) reported concentration factors ranging from less than 1 to 8 in some body parts of a stream gastropod exposed to 0.5 mg/liter atrazine for 24 hours. Paris et al. (1975) reported no measurable adsorption of atrazine by dense populations of microorganisms in aquatic cultures. Ellgehausen et al. (1980) studied the bioaccumulation, depuration, and bioconcentration of atrazine by algae, daphnids, and catfish. They reported bioaccumulation factors of about 90, 1, and 5; depuration halftimes of 0.03 hour, 9.5 hours, and 1.5 days; and biomagnification factors of less than 10. The intensive study reported by these authors indicates no significant bioaccumulation of atrazine will occur in aquatic environments in the forsst. A mollusk accumulated atrazine to a level 3 to 4 times greater than the concentration in water (0.05 mg/liter) during a 72–hour exposure period. Most of the accumulation occurred in the first 12 hours. Similar results were obtained with whitefish. Water rather than food appeared to be the major source of the herbicide (Gunkel and Streit 1980).

Douglass et al. (1969) found a peak concentration of atrazine in stream water of 0.03 mg/liter shortly after application (4 kg/ha) and during the first periods of heavy precipitation. After this time, residues did not exceed 0.010 mg/liter (the minimum quantifiable concentration). In a second application (3.36 kg/ha 2,4-D plus 5 kg/ha atrazine), an unsprayed 3-m buffer strip was left adjacent to the stream. No residues of either herbicide were detected in the water. In one test, atrazine concentrations were about 40 times higher on sediments than in water, although the concentration on the sediment seemed independent of the organic-matter content over a range from 2.3 to 31.9 percent (Streit 1979).

## Toxicity

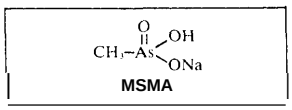
Laboratory and field tests have indicated that atrazine is moderately toxic to fish compared with other herbicides. Macek et al. (1976) investigated the effects of atrazine on survival, growth, and reproduction of three species of fish. Using soft water (hardness 33 to 40 mg/liter) and a continuous–flow apparatus, Macek et al. (1976) were able to show in acute toxicity tests that both the 96–hour and incipient $LC_{50}$ for fathead minnows were 15 mg/liter atrazine. The acute 96–hour $LC_{50}$ for bluegills was more than 8.0 mg/liter and the incipient $LC_{50}$ was 6.7 mg/liter (5.4 to 8.4), which agrees with the 96–hour $LC_{50}$ of about 6 mg/liter atrazine (wettable powder) reported by Walker (1964b) for this species. The 96–hour $LC_{50}$ for atrazine toxicity to brook trout reported by Macek et al. (1976) was 6.3 mg/liter (4.1 to 9.7) and the incipient $LC_{50}$ was 4.9 mg/liter (4.0 to 6.0).

Lorz et al. (1979) found that yearling coho salmon exposed for 144 hours to 15 mg/liter atrazine suffered only a 25-percent mortality. When the survivors of the bioassay were challenged with seawater, only the two groups that had received the highest atrazine concentration suffered any additional mortality.

Spawning, survival, and growth of bluegills and fathead minnows were not affected by exposure to 0.095 and 0.213 mg/liter atrazine, respectively (Macek et al. 1976). Hiltibran (1967) found that 10 mg/liter granular atrazine did not affect embryo development of green sunfish, or survival of bluegills and green sunfish during 8 days. Lake chubsucker fry survived 10 mg/liter wettable powder atrazine. Similarly, parental survival, egg production, and hatchability of brook trout appeared to be unaffected by exposure to 0.72 mg/liter atrazine (Macek et al. 1976). Survival and growth of brook trout fry were, however, significantly reduced after 90 days of exposure to 0.72, 0.45, and 0.24 mg/liter atrazine. Analysis of muscle tissue from bluegills, fathead minnows, and brook trout indicated that these fish did not bioconcentrate detectable amounts of atrazine (detection limits ranged from 0.2 to 1.7 mg/kg) after prolonged exposure (bluegills exposed tu 0.094 mg/liter for 78 weeks, brook trout exposed to 0.74 mg/liter for 44 weeks, and fathead minnows exposed to 0.21 mg/liter for 43 weeks) (Macek et al. 1976).

Walker (1964b) observed no fish mortality after application of 2.0 to 6.0 mg/liter atrazine to ponds infested by aquatic weeds. He suggested, however, that atrazine could affect fish in ways other than direct toxicity. A reduction in bottom fauna was observed immediately after application. Among the most sensitive were mayflies (Ephemeroptera), caddisflies (Trichoytera), leeches (Hirudinea), and gastropods (Musculium). Studies by Macek et al. (1976) on the chronic toxicity of atrazine to selected aquatic invertebrates indicated that morphological development of progeny is particularly sensitive. Exposure of two successive generations of chironomids to 0.23 mg/liter atrazine resulted in reduced hatching success, larval mortality, developmental retardation, and a reduction in the percentage of pupating larvae and emerging adults. Continuous exposure to 0.25 mg/liter atrazine significantly reduced production of Daphnia magna. Development to the seventh instar of the $F_1$ generation of gammarids exposed to 0.14 mg/liter atrazine was reduced 25 percent below that of lower concentrations and controls.

MSMA



MSMA

MSMA is a pentavalent organic arsenical herbicide. In forestry, its principal use has been stem injection, both for precommercial thinning and to aid in control of certain bark beetles. These particular patterns of use provide only limited opportunity for entry into the aquatic environment. National Forest Products Association (see footnote 3) and Newton (1981) reviewed the use of MSMA in forestry. Norris[10] reported the results of a major study of the behavior and impact of the organic arsenical herbicides in the forest environment.

## Behavior in the Environment

The water solubility of MSMA is 25 g/100 g of water at 20 °C. Although it has very little vapor pressure, MSMA may be altered by microbial action to derivatives of arsine that are volatile. The behavior of MSMA in the environment was reviewed by Ray (1975). In soils, MSMA reacts with iron, aluminum, calcium, and magnesium to form compounds of low solubility. Wauchope (1975) reported the organic arsenicals are intensively adsorbed by soils with high contents of clay, iron, and aluminum oxide. The phytotoxicity of MSMA is rapidly dissipated in soil, probably through a strong interaction between the herbicide and soil particles. Some microbial degradation of MSMA has been reported; an arsenate was the product of the metabolism (Von Endt et al. 1968). Robinson (1975) measured arsenic residues in soils over a 5-year period after annual applications of MSMA at rates ranging from 4.4 through 288 kg/ha. Elemental arsenic did not increase in any plot receiving MSMA at rates less than 36 kg/ha. The mechanisms of loss in these studies were not determined.

Dickens and Hiltbold (1967) conducted column leaching studies that showed that after 20 successive 2.5-cm increments of water were added to a loam sand, about half of the applied MSMA remained in the surface 2.5 cm of soil and none was leached below 15 cm. Using columns of forest-floor material and soil from ponderosa pine, Douglas-fir, and mixed-fir forest types, Norris (see footnote 10) determined MSMA was rapidly leached through the forest-floor material but was not leached in the three different soils by 86.4 cm of water applied over a 20-day period. In tests with 2.54 cm of undisturbed forest-floor material, as little as 2.5 cm of water delivered over an 8-day period was sufficient to move about half of the surface-applied MSMA through the forest-floor material. These results indicate that MSMA deposited on the forest floor will readily move through it to the soil--even with small amounts of precipitation. Once reaching the soil, however, MSMA is rapidly immobilized.

Norris (see footnote 10), reporting the work of Canutt and Norris, observed a decline with time in the arsenic concentration in the forest floor under stands that had been precommercially thinned with MSMA. The fate of arsenic in the forest floor was not determined, but the small increases in soil residues indicated some movement from forest floor to soil.

---

[10] Unpublished report, "The behavior and impact of organic arsenical herbicides in the forest: final report on cooperative studies," by L. A. Norris, U.S. Dep. Agric., For. Serv., Pac. Northwest For. and Range Exp. Stn., For. Sci. Lab., Corvallis, Oreg,, 1974.

Norris (see footnote 10) looked for arsenic in four different streams flowing from areas that had been precommercially thinned with MSMA. Samples were collected at various intervals after treatment, with special emphasis given to storm periods when runoff events might occur and again during spring runoff. Only five samples contained detectable quantities of arsenic; four of these five were at the minimum level of detection; the fifth sample was from an upstream site presumably containing water that had not passed through areas previously thinned with MSMA. The results of this study indicate the careful application of MSMA in thinning programs poses little or no threat of increased arsenic levels in aquatic systems.

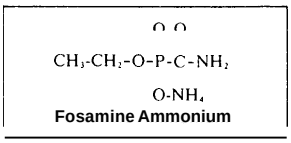
Woolson et al. (1976) determined the distribution and persistence of MSMA in two aquatic model ecosystems. One system contained sandy loam soil as the sediment, channel catfish, and crayfish; the second system contained sediment, algae, daphnids, Gambusia, and crayfish. The concentration of arsenic in the water was reduced from 2 to 10 times in 5 days. Catfish showed little tendency to bioaccumulate arsenic from MSMA (bioaccumulation ratio of 4:1) and showed substantial reduction in bioaccumulation level after 14 days in fresh water. Crayfish (Procambarus clarki) showed higher levels of accumulation (bioaccumulation ratios of 80:1 to 480:1). They also showed a 50-percent decrease in arsenic concentration after 18 days in fresh water. The second experiment was conducted similarly, and different results were obtained. Gambusia had bioaccumulation ratios of about 100:1, but crayfish showed bioaccumulation ratios of less than 10:1. Daphnids and algae had bioaccumulation ratios of 5:1 and 34:1, respectively. Although MSMA does show a slight tendency for bioaccumulation, the limited probability that it will be present in aquatic systems in the forest reduces the importance of this characteristic.

## Toxicity

Only limited data are available on toxicity effects of MSMA to fish. The 96-hour $LC_{50}$ ranges from 12 to more than 100 mg/liter, depending on species, test conditions, and the amount of active ingredient in the formulation tested (U.S. Environmental Protection Agency 1975, Johnson and Finley 1980). McCorkle et al. (1977) found less than 10-percent mortality occurred in fingerling channel catfish held for 48 hours under static conditions at 10 mg/liter of MSMA.

Sanders (1970) found that MSMA was not toxic to the scud (Gammarus fasciatus) after 96 hours of exposure at 100 mg/liter. Spehar et al. (1980) recently completed experiments on the comparative toxicity of arsenic compounds and their accumulation in invertebrates and fish. These investigators noted that concentration of 1 mg As/liter as arsenic III was lethal to amphipods within 1 week. The same concentration of arsenic supplied as arsenic V, disodium methanearsonate (DSMA), or sodium dimethyl arsonate (SDMA) did not significantly decrease the survival of amphipods (Gammarus pseudolimnaeus) and Daphnia magna after 2 weeks of exposure, or other species including stoneflies, snails, and rainbow trout after 28 days.

## Fosamine Ammonium



Fosamine Ammonium

Fosamine ammonium is a new herbicide that is expected to be increasingly used in forestry. It is registered for control of a wide variety of woody vegetation. It is usually applied as a foliar spray either by aerial or ground equipment during the 2 months before fall coloration. Rates of application of 3.36 to 6.72 kg/ha are common. National Forest Products Association (see footnote 3) and Newton (1981) discuss the use of fosamine ammonium in more detail.

### Behavior in the Environment

Fosamine ammonium is highly water soluble (179 g/100 g at 25 °C) with substantially less solubility in nonpolar organic solvents (0.02 g in 100 g n-hexane at 25 °C). It has little vapor pressure ($4 \times 10^{-6}$ mmHg at 25 °C).

Fosamine ammonium is not persistent in soils; laboratory studies indicate the mechanism of disappearance is rapid decomposition by soil microorganisms (Han 1979a). In greenhouses, a half-life of about 10 days has been recorded. In field studies in Delaware, Illinois, and Florida, a half-life of about 1 week was found for the parent compound (Han 1979a). Fosamine ammonium shows only limited mobility in column leaching studies. After 56 cm of leaching water had been applied, 60 to 80 percent of the herbicide was contained in the top 10-cm zone of the soil column. After 1 year and 165 cm of rain in the field, 93 percent of the chemical present was in the top 10 cm of soil. Studies of leaching in soil columns showed that fosamine ammonium is readily bound by soil particles and, despite its high water solubility, has little tendency to leach.

The probability of ground-water contamination or movement of fosamine ammonium to streams by leaching is negligible.

Field data on stream contamination with fosamine ammonium are lacking, but because direct application and drift are probably the principal routes of entry, the data base for 2,4-D is probably applicable. Fosamine ammonium undergoes decomposition in water. In laboratory tests, fosamine ammonium (pH 5) was completely degraded in 2 weeks. At more neutral pH's, however, the compound was quite stable in water (Han 1979a). A strong interaction of fosamine ammonium with soil suggests it is likely to be adsorbed on suspended sediments or stream-bottom sediments where it enters forest streams. Stream-bottom sediments lose fosamine in 3 months or less.[11/]

Specific information on bioaccumulation of fosamine ammonium is limited; as with other pesticides of high water solubility, however, the probability of bioaccumulation is limited. Laboratory tests have demonstrated that fosamine ammonium is not bioaccumulated. Residues in fish tissues were comparable to the concentration of the herbicide in water (Newton and Norgren 1977). Residues in channel catfish exposed to 1.1 mg/liter $^{14}$C-carbonyl-labeled fosamine ammonium in water for 4 weeks were found to plateau in 2 to 3 weeks, with an accumulation factor (ratio of residue in fish to residue in water) of less than 1. In a separate experiment, channel catfish were placed for 4 weeks in a tank containing ($^{14}$C) fosamine-ammonium-treated soil (15 mg/liter) that had been aged for 30 days before flooding and initiation of fish exposure. The residue levels in this group of catfish also reached

---

11/ Unpublished data of J. Harrod, Biochemicals Dep., 1007 Market St., Wilmington, Del., 1979.

a plateau in 2 to 3 weeks with an accumulation factor of less than 1. In both experiments, after the 4-week exposures, the fish were transferred to fresh water for 2-week depuration periods, during which residue levels dropped 50 to 90 percent. No effects on the fish were observed during these experiments (Han 1979b). In rats, fosamine ammonium was rapidly excreted (0.05-percent retention in body .beyond 72 hours) (Chrzanowski et al. 1979).

## Toxicity

There is little published data on the toxicity of fosamine ammonium to fish. Unpublished findings of E. I. duPont de Nemours and Company, Inc., [12] indicate that rainbow trout and fathead minnows have a 96-hour $LC_{50}$ of 1000 mg/liter (product); bluegills exhibit a 96-hour $LC_{50}$ of 670 mg/liter (product).

In a recent series of laboratory bioassays, McLeay and Gordon (1980) added extensively to the toxicity data for Krenite with their partial life-cycle studies of coho salmon (egg through smolt) and rainbow trout (egg through fingerling). Highlights from their study are as follows:

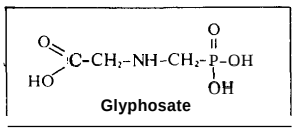
- For both fish species, the'alevin (yolk-sac fry) was the stage most sensitive to fosamine ammonium; 4-day median lethal concentrations ($LC_{50}$), including postexposure mortality, were 618 (coho) and 367 (rainbow) mg/liter.
- Egg stages were generally highly tolerant of fosamine ammonium.
- Swim-up fry and young fingerlings of both species were somewhat more tolerant to fosamine ammonium than was the alevin stage--yearling coho presmolts were slightly more tolerant than coho fingerlings (4-day $LC_{50}$ values of 7014 and 5361 mg/liter, respectively); coho smolts showed a minor increase in sensitivity compared to the presmolt stage.
- Postexposure mortalities occurred after a 4-day exposure of all life stages of each salmonid species to fosamine ammonium.
- No groups surviving previous exposure to fosamine ammonium showed any latent effects throughout the observation period in fresh water.
- Four-day $LC_{50}$ values for swim-up fry exposed to fosamine ammonium varied 12-fold when the diluent waters varied in pH, hardness, and alkalinity (increases in toxicity were related to increases in pH, alkalinity, and hardness).
- Comparison of the acute toxicity of fosamine ammonium with other brush-control herbicides (2,4-D, 2,4,5-T, silvex, picloram, amitrole, and glyphosate) to salmonid fish indicated that fosamine ammonium was 2 to 4 orders of magnitude less toxic.

In another study, no mortality was observed in yearling coho salmon exposed to concentrations up to 200 mg/liter of fosamine ammonium (Lorz et al. 1979) nor was subsequent survival in seawater affected.

---

[12] Unpublished data of E. I. DuPont de Nemours and Co., Inc., Biochemicals Dep., 1007 Market St., Wilmington, Del., 1979.

## Glyphosate



Glyphosate

Glyphosate is a new herbicide that is expected to be used increasingly in forestry. It is proving useful for both site preparation and release treatments at rates of application up to 4.48 kg/ha. National Forest Products Association (see footnote 3) and Newton (1981) discuss the use of glyphosate in more detail; Chykaliuk et al. (1981) have published an extensive bibliography on this chemical.

### Behavior in the Environment

Glyphosate is highly water soluble (12 000 mg/liter at 25 °C) with much lower solubility in organic solvents. It has negligible vapor pressure.

In general, glyphosate is very immobile in soil, rapidly adsorbed by soil particles, and subject to some degree of microbial degradation. Sprankle et al. (1975a, b) showed glyphosate was rapidly inactivated in soil, apparently by physical adsorption processes, because autoclaving the soil did not stop the inactivation. Addition of phosphate to the soil altered the availability of the glyphosate (Hance 1976). The initial binding of glyphosate to the soil was reversible, with phosphate ions competing for binding sites. Thus, the initial rapid inactivation of glyphosate in soil probably results from rapid adsorption rather than degradation. Some microbial degradation of the herbicide does occur in soil--45-percent degradation occurs in 28 days, according to Sprankle et al. (1975a, b). They also showed by thin-layer chromatography that glyphosate was immobile in soil.

Moshier and Penner (1978) reported decomposition of glyphosate was substantially different in different soils. After 32 days, the rate of decomposition in three soils was 40, 9.5, and 3 percent of the amount originally added. Rueppel et al. (1977), reporting on extensive work in both soil and water, found the degree of glyphosate decomposition in soils ranged from 5 to 50 percent in 28 days, depending on the soil. In two of three soils examined, 90 percent of the chemical was dissipated in less than 12 weeks. Aminomethylphosphonic acid was the only significant soil metabolite of glyphosate, and it degraded 16 to 35 percent in 60 days in various soils. The authors classified the chemical as immobile in soil, based on leaching experiments. These findings on the behavior of glyphosate in soil are consistent with the research reported by Torstensson and Aamesepp (1977) and Hance (1976).

Glyphosate was applied to an agricultural watershed at 1.10, 3.36, and 8.96 kg/ha (Edwards et al. 1980), and runoff from natural rainfall after treatments in early spring was measured and analyzed to define concentration and transport. The highest concentration (5.2 mg/liter) was found in runoff occurring 1 day after treatment at the highest rate. Glyphosate (0.004 mg/liter) was detected in runoff from this watershed up to 4 months after treatment. For the lower rates of application, maximum concentration of the herbicide in runoff was 0.094 mg/liter for events occurring 9 to 10 days after application, and decreased to 0.002 mg/liter within 2 months of treatment. The maximum amount transported by runoff was 1.85 percent of the amount applied, most of which occurred during a single storm on the day after application of the highest rate of glyphosate (8.96 kg/ha). In each of the three study years, herbicide transported in the first runoff event after treatment accounted for 99 percent of the total herbicide

runoff on one watershed. Glyphosate residues in the upper 2.5 cm of treated soil decreased logarithmically with time; they persisted several weeks longer than in the runoff water.

Most of the data on the fate of glyphosate in water come from canal studies where glyphosate was used for weed control on banks. Comes et al. (1976) looked for both glyphosate and its principal metabolite in the first flow of water through two canals, after applications of 5.6 kg/ha to ditch banks when the canals were dry. Some of the herbicide was applied to surfaces of the canal that would be below the normal waterline. No glyphosate or metabolite was detected in the first flow of water through the canals. Soil samples collected the day before the canals were filled (about 23 weeks after treatment) contained 0.35 mg/liter glyphosate and 0.78 mg/liter metabolite in the 0- to 10-cm layer. When glyphosate was added to flowing canal water (sufficient to achieve 150 µg/liter), about 30 percent of the herbicide was lost in 1.6 km of travel. Thereafter, the rate of disappearance diminished; about 58 percent was present 8 and 14 km downstream from the introduction sites on two study canals, which implies interaction between the concentration and the mechanism of loss. Rueppel et al. (1977) reported less than 0.02 percent of applied glyphosate was removed in runoff processes from soil after artificial rain was applied at the rate of 1.9 cm/hour 1, 3, and 7 days after application of chemical.

Sacher (1978) summarized the behavior and impact of glyphosate in the aquatic environment. He reported glyphosate dissipated rapidly in static water, following first-order kinetics; the half-life of glyphosate in the pond was 12 days.

Relatively little has been done on the bioaccumulation of glyphosate, primarily because its physical-chemical properties are such that bioaccumulation is not expected to be substantial. Studies of fish metabolism demonstrated that glyphosate has an extremely low bioaccumulation factor. In three fish species--channel catfish, largemouth bass, and rainbow trout--the maximum levels of glyphosate, after 14 days exposure to 10 mg/liter, did not exceed 0.55, 0.12, and 0.11 mg/liter, respectively. These represent bioaccumulation factors of less than 0.18 (Sacher 1978).

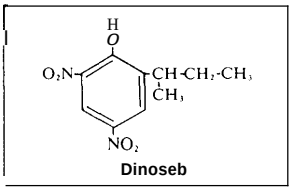
No residues of glyphosate or its primary metabolite (aminomethyl-phosphonic acid) were detected in the fillets or eggs of rainbow trout exposed to the isopropylamine salt (Folmar et al. 1979). In rainbow trout exposed to 2.0 mg/liter of Roundup, however, the fillets contained 80 mg/kg of glyphosate and the eggs contained 60 mg/kg, representing accumulation factors of 40 and 30. Midge larvae collected in drift and substrate samplers for 7 days after exposure to 2.0 mg/liter did not contain detectable glyphosate residues (Folmar et al. 1979).

## Toxicity

Folmar et al. (1979) conducted studies to determine the acute toxicity to four aquatic invertebrates and four species of fish of glyphosate, the isopropylamine salt of glyphosate, the formulated herbicide Roundup, and the Roundup surfactant. The 96-hour $LC_{50}$ values for Roundup varied from 2.3 mg/liter for the fathead minnow to 13 mg/liter for the channel catfish to 43 mg/liter for mature scuds (Gammarus pseudolimnaeus). Rainbow trout were intermediate in sensitivity. Technical-grade glyphosate, the active ingredient in Roundup, was less toxic than Roundup or the surfactant; the 96-hour $LC_{50}$ for rainbow trout was 140 mg/liter (glyphosate) and 2 mg/liter (surfactant). Roundup was more toxic to rainbow trout and bluegills at higher test temperatures, and was more toxic at pH 7.5 than at pH 6.5. Eyed eggs of rainbow trout were the most resistant life stage, and sensitivity increased as the fish entered the sac-fry and swim-up stages. In avoidance studies, rainbow trout did not avoid concentrations of the isopropylamine salt up to 10.0 mg/liter; mayfly nymphs avoided Roundup at concentrations of 10 mg/liter, but not at 1.0 mg/liter.

In a simulated aerial application of Roundup to a forested area, Hildebrand et al. (1980) found no detectable effects on Daphnia magna in a forest pond after application of 2.2, 22, and 220 kg/ha.

## Dinoseb



Dinoseb

Dinoseb is a contact-action herbicide available in two forms--free phenol and amine or ammonium salt of the phenol. Its only registered use in forestry is as a desiccant before burning for forest-site preparation (State of Oregon only). Although its scope of use is limited, it is included in this paper because of its high toxicity to aquatic species and its use in areas of important anadromous fish habitat.

## Behavior in the Environment

The phenol form of dinoseb has substantial vapor pressure (0.01 mmHg at 78 °C) and is soluble to 52 mg/liter in water, 23.4 percent in ethyl alcohol, and 8.7 percent in diesel fuel at 25 °C (Melnikov 1971). The salt forms are highly soluble in water and have substantially less vapor pressure. Interconversion between the salt and free phenol forms is expected, depending on the pH of the medium and the presence of other ions.

Dinoseb is subject to volatilization from soil. Hollingsworth and Ennis (1953) showed that this process depended on ambient temperature, moisture content of the soil, and the formulation applied. It was attributed to water-vapor distillation by Barrons et al. (1953) and did not occur at soil pH above 8.

The residual life of dinoseb is 3 to 5 weeks in warm, moist soils. Carryover from one season to the next is not expected (Klingman and Ashton 1975). Dinoseb is not tightly adsorbed on most agricultural soils, and it can leach in many sandy soils; Davis and Selman (1954) reported that the phenol form moved less than 2 cm with 5 cm of rain in any soil they tested. The amine salt, however,

leached 3.8 cm in sandy loam, 6.3 cm in clay loam, and 8.9 cm in loam after the same amount of rain. Upchurch and Mason (1962) reported dinoseb interacted strongly with soil organic matter. Dinoseb was almost completely adsorbed at pH 2.3. In zones of moderate temperature and rainfall, and at normal rates of application, dinoseb should not be leached from the top foot of the acid forest soils of the Pacific Northwest in the 1st year after application. Substantial decomposition by microbial action takes place within the 1st year of application. Phytotoxic levels may remain in soil from 2 weeks to 6 months, depending on the environment in which it is used.

We found no published information on the levels or persistence of dinoseb in stream water. We assume 2,4-D is a reasonable model for dinoseb because direct application and drift are probably the main routes of entry into streams.

Data on dinoseb bioaccumulation are lacking. In the phenol form, bioaccumulation during periods of exposure should be expected. In the salt form, this behavior will be less pronounced. Lorz et al. (1979) found measurable residues of dinoseb in a few coho salmon exposed to 0.02 mg/liter of dinoseb for 384 hours. Most samples did not contain detectable residues (levels ranged from less than 0.01 to 0.03 mg/kg in gill, 0.37 mg/kg in kidney, 0.4 mg/kg in liver, and 1.4 mg/kg in spleen).

## Toxicity

Dinoseb is more toxic to humans, animals, and fish than most herbicides. The acute and chronic effects of dinoseb on cutthroat and lake trout were investigated by Woodward (1976), who found that toxicity of a given exposure was greatly influenced by water quality. Decreasing the pH of the water resulted in a corresponding increase in dinoseb toxicity to fish. Similar findings were reported by Lipschuetz and Cooper (1961) for technical-grade dinoseb. Decreasing the pH from 8.0 to 6.9 increased the toxicity of dinoseb to rainbow trout by a factor of 5. High temperature and hardness of the water also tend to maximize the toxicity of dinoseb to fish, but to a lesser extent than pH (Webb as cited by Lipschuetz and Cooper 1961, Woodward 1976).

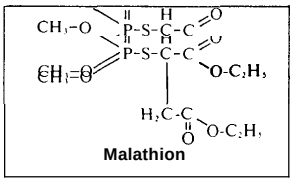
The 96-hour $LC_{50}$'s, under varying conditions of pH, temperature, and hardness, ranged from 0.41 to 1.35 mg/liter dinoseb for cutthroat trout and from 0.032 to 1.40 mg/liter for lake trout (Woodward 1976). Lipschuetz and Cooper (1961) observed similar dinoseb values for rainbow trout. Their 24-hour $LC_{50}$ at pH 8.0 was 0.30 mg/liter (technical) and at pH 6.9 was 0.073 mg/liter (18 °C). Western blacknose dace in water of pH 8.0 and 21 °C had a 24-hour $LC_{50}$ of 0.24 mg/liter dinoseb (technical). Using a dinoseb formulation composed of secondary butyl dinitrophenol and secondary amylbutyl dinitrophenol, Lipschuetz and Cooper (1961, citing the 1955 work of Webb) reported a 24-hour $LC_{50}$ for redside shiners of 0.16 mg/liter in soft water (18 mg/liter hardness, pH 7.6) and of 0.24 mg/liter in hard water (105 mg/liter hardness, pH 8.2).

Woodward (1976) observed no cumulative mortality of lake and cutthroat trout with chronic exposures (8 to 12 days) to dinoseb. Prolonged exposures of 0.005 to 0.010 mg/liter dinoseb, however, affected yolk absorption time and fry growth. Yolk absorption time increased by 6 to 9 days over that of the controls, and fry growth was reduced at all concentrations of dinoseb tested.

Lorz et al. (1979) calculated the 24-hour $LC_{50}$ of dinoseb to yearling coho salmon to be 0.190 mg/liter, and the 144-hour $LC_{50}$ was estimated to be 0.088 mg/liter (under static conditions) at 10 °C and pH 7.0. When survivors of this bioassay were challenged with seawater, no mortalities occurred. In a flowing-water system, the toxicity of dinoseb appeared greater--93 percent of coho salmon yearlings exposed for 6 days to 0.060 mg/liter died, and all were dead in 16 days; in the group exposed to 0.040 mg/liter, 94 percent died during the 16-day exposure. Releasing dinoseb-exposed coho salmon and monitoring their downstream movement showed that groups exposed to 0.040 and 0.060 mg/liter for 48 hours were less migratory than the controls. Yearling coho salmon exposed to 0.100 mg/liter for 114 hours showed extensive necrosis of the liver, kidney, and gill lamellae; however, examination of tissues of fish exposed to 0.040 and 0.060 mg/liter showed only minor degenerative changes.

## INSECTICIDES

### Malathion



Malathion

Malathion is an organophosphate insecticide that receives extensive use in both agriculture and forestry. It has been available for use since 1959. Information on the use and impact of malathion in the forest are in two environmental impact statements (U.S. Department of Agriculture, Forest Service' 1976; U. S. Department of Agriculture, Animal and Plant Health Inspection Service, 1980). The most recent uses of malathion on lands managed by the Forest Service have been for control of western spruce budworm and grasshoppers on western forests and ranges (aerially applied at 0.8 kg/ha).

#### Behavior in the Environment

Various aspects of the behavior of malathion in the environment are cited in several chapters of Haque and Freed (1975). Malathion has a vapor pressure of $4 \times 10^{-5}$ mmHg (30 °C) and a water solubility of 145 mg/liter. It is soluble in most organic solvents, but is of limited solubility in petroleum oils.

Malathion disappears rapidly from soil, probably by both chemical and biological means. Konrad et al. (1969) reported 50- to 90-percent disappearance in 24 hours, in both sterile and nonsterile soil systems, depending on the type of soil. Malathion disappears rapidly from soil even at high rates of application. Roberts (1962) reported malathion applied to the soil from a tractor-mounted sprayer at 85.8 kg/ha 'completely disappeared after 6 months; only 3.1 kg/ha remained after 1 month. Both the persistence and mobility of malathion were determined at land waste-water disposal sites where the chemical was applied by sprinkler irrigation for long periods. In these tests, the chemicals were applied (0.1 mg/liter) in the secondary effluent from a two-stage trickling filter for 15 weeks. Malathion was never present in excess of 0.002 mg/kg in the soil and 0.001 mg/liter in the soil water. These results indicate malathion will neither accumulate in soil nor translocate in soil waters under the types of conditions tested (Jenkins et al. 1978).

Concentrations of malathion in stream water were monitored in connection with the spruce budworm control projects conducted in the State of Washington in 1976 (table 8). Tracy et al. (1977) reported the peak concentrations of malathion in streams without buffer strips were 0.037 to 0.042 mg/liter; in streams with buffer strips, peak concentrations ranged from nondetectable to 0.017 mg/liter. Residues were not detected in most of the 48-hour postspray samples. No residues were found in fish or benthic organisms from these streams.

Table 8--Peak concentrations of malathion and Sevin-4-Oil in stream water as affected by stream size and buffering, Okanogan and Wenatchee National Forests[1/]

| Chemical and stream | Streamflow | Protection | Peak concentration |
|---|---|---|---|
| | Cubic meters per second | | Micrograms per liter |
| Malathion:[2/] | | | |
| Hansel Creek | 0.34 | Nonbuffered | 37 |
| Gold Creek | .11 | Nonbuffered | 42 |
| Nason Creek | 25 | Buffered | 3 |
| Little Bridge Creek | .57 | Buffered | 0 |
| Taneum Creek-- | | | |
| Spray day 1 | 3.3 | Buffered | 17 |
| Spray day 3 | 3.3 | Buffered | 4 |
| Teanaway River-- | | | |
| Spray day 1 | 5.1 | Buffered | 1 |
| Spray day 2 | 5.1 | Buffered | 21 |
| Sevin-4-Oil:[3/] | | | |
| Gate Creek | .14 | Nonbuffered | 11 |
| Yellow Jacket Creek | .01 | Nonbuffered | 5 |

1/Tracy et al. (1977).
2/Malathion applied as ultralow-volume spray, 13 oz/acre (0.91 kg/ha).
3/Sevin-4-Oil (carbaryl) applied at about 1 lb/acre (1.12 kg/ha).

Eichelberger and Lichtenberg (1971) determined the persistence of malathion in river water. When the malathion was added at 0.010 mg/liter, 0.002 mg/liter was observed after 1 week, 0.001 mg/liter was measured after 2 weeks, and no detectable residue was found after 4 weeks. In a soil-free, aqueous system that had been inoculated with a soil extract, malathion disappeared in two phases; a relatively slow phase accounted for about 30 percent disappearance in 180 hours, and a more rapid phase accounted for more than 50 percent disappearance in the next 60 hours. Degradation in the aquatic system would have represented both chemical degradation (the slow phase) and microbial degradation (the rapid phase). Freed et al. (1979) reported half lives of malathion of 1.3 days at 37 °C and 11 days at 20 °C, when the pH was 7.4 (in a laboratory system that would be expected to have low biological activity). At pH 6.1, the half-life of malathion was 120 days. Konrad et al. (1969) reported a similar effect of pH on malathion persistence in aqueous systems. In a more natural aqueous system, however, they reported more than 90-percent decomposition of malathion in the water in 230 hours. Walker (1978) reported malathion was the shortest lived of the insecticides tested in both fresh and salt water. Half lives in his system varied from 11 days in fresh water to less than 2 days in saline water.

Malathion is expected to show little bioaccumulation. Kenaga (1980a, b) predicted a bioconcentration factor of 37. Paris et al. (1975) found no measurable absorption of malathion by dense populations of microorganisms. The high water solubility and the low fat solubility of malathion will result in its rapid excretion or elimination from organisms that have accumulated it. Residues of malathion have been found in milk collected from cattle 5 hours after they were sprayed at rates several times the normal rate used in aerial applications in forestry. Only trace amounts were found 3 days after treatment. The rapid disappearance of the insecticide was attributed to its rapid excretion by the animal. The short persistence of malathion in the aquatic environment will also be an important factor in limiting its bioaccumulation.

## Toxicity

Hoffman (1957), Stavinoha et al. (1966), and Livingston (1977) reviewed the literature and noted that the organophosphate insecticides were generally short lived in the environment, did not significantly bioaccumulate or biomagnify, and had a relatively uniform and well-understood effect on a variety of organisms. The "safe" concentrations of organophosphate insecticides have been estimated through acute toxicity studies and determination of environmental persistence (Benson 1969). Although the acute toxicity of these compounds to aquatic organisms is generally lower than the organochlorines, it varies widely (Tarzwell 1958, Pickering et al. 1962, Macek and McAllister 1970, Johnson and Finley 1980).

Toxic action to various species has been associated with specific reactions to synergistic or antagonistic effects between the parent compounds and hydrolysis products. Numerous studies have shown that cholinesterase is the primary site of action of the organophosphate compounds. Symptoms of acute toxicity vary from species to species, however, and diverse formulations have different acute effects.

Eaton (1970) conducted a study of chronic malathion toxicity to bluegills similar to the study by Mount and Stephan (1967) on the fathead minnow. In Eaton's study, concentrations of 66 and 28 µg/liter were lethal to all fish within 16 and 54 days, respectively, and some were killed at 15 µg/liter. Reproduction and early fry survival were unaffected by the 7.4 µg/liter concentration that crippled adult fish after exposure for several months.

Mulla and Mian (1981) synthesized and interpreted much of the available information on the impact of malathion and parathion on nontarget flora and fauna in aquatic ecosystems, as well as the persistence and distribution of these chemicals in aquatic habitats. Malathion was shown to have low toxicity to several mollusks, but was considerably more toxic to crustaceans (water fleas, amphipods, shrimp, and juvenile crabs). Immature nontarget insects, such as caddisflies, stoneflies, and mayflies, were highly sensitive. Malathion exhibited differential toxicity to various fish species; some species showed a substantial degree of tolerance.

Although malathion is a widely used organophosphate insecticide entering surface waters in various ways, residue interpretation is difficult because of the toxicity of a "persistent" metabolite (malaoxon), which is not easily identified in tissues. Cook et al. (1976) suggested alternate methods of analysis, including analysis for malathion monoacid in the gut and measurement of brain acetylcholinesterase activity, because the parent compound is rapidly absorbed and altered by fish. Bender (1969) found that a basic hydrolysis byproduct of malathion, with a pronounced synergistic effect demonstrated between malathion and its two hydrolysis products, was more toxic to fathead minnows than the parent compound. Bender and Westman (1976) found that malathion could damage eastern mudminnows through either acute or chronic toxicity at concentrations of 0.09 to 0.24 mg/liter (the $LC_{50}$'s of malathion and its principal hydrolysis products). Desi et al. (1976) found that although malathion was only slightly toxic to guppies, it was highly toxic to invertebrates such as Daphnia magna ($LC_{50}$ 0.003 mg/liter) and to juvenile forms of various species. They found that malathion affects aquatic organisms differently and, by exerting stress on "sophisticated functions" and exhausting the adaptability of such organisms, it is "not an entirely harmless agent for the environment." Table 9 summarizes some of the data on acute toxicity available for malathion to important invertebrate and fish species.

Table 9--Acute toxicity of malathion to various invertebrate and fish species

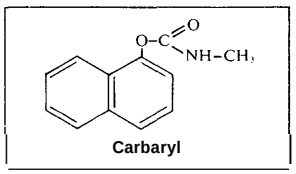
| Test organism | Stage or weight | Temperature | 96-h $LC_{50}$ | Reference |
|---|---|---|---|---|
| | Grams | Degree C | mg/liter | |
| Invertebrates: | | | | |
| *Daphnia magna* | I[1] | 15 | [2]0.0010 | Johnson and Finley 1980 |
| *D. pulex* | I[1] | 15 | [2].0018 | Johnson and Finley 1980 |
| *Asellus* sp. | M[3] | 21 | 3.0 | Johnson and Finley 1980 |
| *Gammarus fasciatus* | M[3] | 21 | .00076 | Johnson and Finley 1980 |
| *Isoperla* sp. | YC[4] | 15 | .00069 | Johnson and Finley 1980 |
| *Limnephilus* sp. | J[5] | 15 | .0013 | Johnson and Finley 1980 |
| fish: | | | | |
| Chinook salmon | 1.4-5 | 20 | .023 | Katz 1961 |
| Coho salmon | .9 | 12 | .17 | Johnson and Finley 1980 |
| Coho salmon | .6-1.7 | 13 | .101 | Macek and McAllister 1970 |
| Cutthroat trout | 1.0 | 12 | .28 | Johnson and Finley 1980 |
| Rainbow trout | 1.4 | 12 | .20 | Johnson and Finley 1980 |
| Rainbow trout | .6-1.7 | 13 | .20 | Macek and McAllister 1970 |
| lake trout | .3 | 12 | .076 | Johnson and Finley 1980 |
| Brown trout | 1.1 | 12 | ,101 | Johnson and Finley 1980 |
| Brown trout | .6-1.7 | 13 | .20 | Macek and McAllister 1970 |
| Fathead minnow | .9 | 18 | 8.650 | Johnson and Finley 1980 |
| Fathead minnow | 1-1.5 | 25 | 12.5 | Henderson and Pickering 1958 |
| Fathead minnow | 1-2 | 25 | 23 | Pickering et al. 1962 |
| Black bullhead | 1.2 | 18 | 12.9 | Johnson and Finley 1980 |
| Bluegill | 1-2 | 25 | .09 | Pickering et al. 1962 |
| Bluegill | 1.5 | 18 | .103 | Johnson and Finley 1980 |
| Bluegill | 1.5 | 25 | .095 | Henderson et al. 1959 |
| Walleye | 1.3 | 18 | .064 | Johnson and Finley 1980 |
| Yellow perch | 1.4 | 18 | .263 | Johnson and Finley 1980 |

[1] First instar.
[2] 48-hour $EC_{50}$.
[3] Mature.
[4] First-year class.
[5] Juvenile.

Johnson and Finley (1980) noted that 0.3-g lake trout fry were twice as sensitive to malathion as 45-g fingerlings. An increase in temperature from 7 °C to 29 °C caused a 4-fold increase in toxicity to bluegills. Variations in water hardness did not appreciably alter the toxicity to fish or invertebrates. Salmonids exposed to 0.120 to 0.300 mg/liter malathion showed 70- to 80-percent inhibition of acetylcholinesterase (AChE), and activity indexes were reduced by 50 to 70 percent of that of unexposed fish. Goldfish exposed to sublethal levels showed a significantly reduced avoidance response at levels below that causing a reduced AChE activity.

Exposures of rainbow trout to sublethal levels of malathion for 1 hour caused severe tissue damage to the gills and minor nonspecific liver lesions. Ponds given four semimonthly treatments up to 0.02 mg/liter during May through July produced no discernible effects on resident bluegills or channel catfish. Populations of aquatic insects, however, were significantly depressed by high but not low treatment rates. These data indicate that use of malathion needs careful planning because some species and habitat-specific reactions to this pesticide can cause adverse effects.

## Carbaryl



Carbaryl

Carbaryl is a broad-spectrum, relatively nonpersistent carbamate insecticide that has been used for nearly 30 years to suppress various types of insect infestations. Registered for use against many insects, its principal use in agriculture is at rates of 0.5 to 2.24 kg/ha active ingredient, often in repeated spray treatments. In forestry, it is used to control defoliating insects (U.S. Department of Agriculture, Forest Service 1976). Forest-application rates of more than 1.12 kg/ha are uncommon. In fiscal year 1980, most of the carbaryl used in Forest Service programs was aerially applied for grasshopper control on western forest and range lands. Mount and Oehme (1981) published an extensive literature review on carbaryl--its chemistry, toxicity, metabolism, environmental degradation, and so on.

### Behavior in the Environment

Carbaryl is soluble in most polar organic solvents and to about 0.01 percent in water. In the soil, carbaryl is attacked by soil microbes and is not expected to leach significantly from the upper soil surfaces. Bollag and Liu (1971) isolated several microorganisms capable of metabolizing carbaryl in soil. A half-life of about 12 days was noted in several of these systems. In soil, carbaryl has shown a half-life of about 8 days for application rates ranging from 3.36 to 30.2 kg/ha (Union Carbide 1968). Carbaryl was detected in soil and the forest floor for 64 and 128 days, respectively, after application (Willcox 1972). Goring et al. (1975) estimated the persistence of several pesticides in soil, based on a search of many references. They indicate the estimated time required for 50-percent disappearance of carbaryl from soil ranged from 1.5 to 6 months.

LaFleur (1976) studied carbaryl movement and loss in the soil profile and its accumulation in underground water over a 16-month period. Rainfall during the study was 182 cm. The upper 1 m of soil contained about 6 percent of the applied carbaryl 16 months after application. None was found in the 10- to 20-cm layer after the 4th month. Loss of carbaryl with time in the upper 1 m of soil depended on concentration, and the half-life was less than 1 month. In underlying ground water, carbaryl appeared within 2 months after application and persisted through the 8th month. The maximum ground-water concentration was 0.3 µmole/liter at the end of the 2d month.

No carbaryl was detected in the field plot or soil water at a land wastewater disposal site that received carbaryl (0.1 mg/liter in water) over a 15-week period (Jenkins et al. 1978). The authors concluded that carbaryl does not accumulate or translocate under the field conditions of this test. Haque and Freed (1974) predicted carbaryl will leach less than 20 cm in the soil profile that receives an annual rainfall over 150 cm.

Caro et al. (1974) reported 95 percent of the carbaryl in an agricultural soil had disappeared within 135 days. Of the 4 kg of carbaryl applied, 5.8 g were recovered during the 1st year in runoff water and sediment. Over 90 percent of this loss occurred in a single rainfall 19 days after application. About 75 percent of the seasonal loss was contained in water and 25 percent in sediment.

Paris et al. (1975) indicated carbaryl was degraded both chemically and biologically; the rate of biological degradation was proportional to the density of microorganisms. Chemical degradation predominated in their study. The persistence of carbaryl in water appears to be brief. If carbaryl is applied over open water, such as small brooks or ponds, initial deposits of 1 mg/liter or less in water depths of about 10 cm may be expected to degrade completely or disappear in 1 or 2 days (Lichtenstein et al. 1966).13, 14/ Eichelberger and Lichtenberg (1971), in determining the persistence of several insecticides in river water, found only 5 percent of the original 0.01 mg/liter of carbaryl remained 1 week after application. No residues were detected after 2 weeks. Romine and Bussian (see footnote 13) found no detectable carbaryl in the water of a farm pond 2 days after it had been sprayed at the rate of 6.7 kg/ha. Residues immediately after spraying were 4.8 mg/liter. Residues in the sediment disappeared after 4 days.

---

13/ Unpublished report, "The degradation of carbaryl after surface application to a farm pond," Proj. Rep. 111A13, by R. R. Romine and R. A. Bussian, Union Carbide Corp., Salinas, Calif., 1971.

14/ Unpublished report, "An investigation into the effect on fish of Sevin (carbaryl) used in rice culture," Pittman-Robertson Proj. W-52-R, prepared by Resour. Agency, Wildl. Invest. Lab., Calif. Dep. Fish and Game, Sacramento, 1963.

Karinen et al. (1967) reported the concentration of carbaryl in estuarine water decreased 50 percent in 38 days. When mud was present, more than 90-percent loss occurred in 10 days. The carbaryl was adsorbed where decomposition continued at a slower rate. The principal metabolite of carbaryl, 1-napthal, was less persistent. Carbaryl applied (11.2 kg/ha) to a tidal mud flat disappeared rapidly. The initial residue level of 10.7 mg/kg decreased rapidly the 1st day when the tide removed carbaryl, and the 1-napthal metabolite was not adsorbed on mud. The level in the top 2.5 cm of mud decreased from 3.8 mg/kg 1 day after treatment to 0.1 mg/kg by the 42d day.

Carbaryl was monitored in stream and pond water in the Eastern United States. Values ranged from nondetectable to 0.03 mg/liter; most values were around 0.001 mg/liter (Tracy et al. 1977). In the Pacific Northwest, Tracy et al. (1977) reported 0.005 to 0.011 mg/liter as peak concentrations of carbaryl in streams in areas sprayed for control of western spruce budworm (table 8). These values were for streams without buffer strips. Residues were at or near detection limits in samples collected 48 hours after application. Tracy et al. (1977) did not find measurable carbaryl in aquatic insects from streams flowing through treated forest areas.

Where carbaryl was applied at 0.84 kg/ha for control of spruce budworm, Hulbert (1978) measured peak concentrations of 0.026 and 0.042 mg/liter in water, and Stanley and Trial (1980) detected peak concentrations of 0.0009 to 0.008 mg/liter in brooks (3 to 8 m wide) and 0.0004 to 0.002 mg/liter in rivers (15 to 100 m wide), shortly after application. Buffer strips of 150 m were used. Along one stream where no buffer strip was used, a peak concentration of 0.016 mg/liter was detected. The concentrations decreased exponentially with time after application. The half-life for carbaryl in these stream systems was

23 hours in brooks and 28 hours in rivers. The rate constant ($0.028\ hour^{-1}$) reported by Stanley and Trial (1980) for carbaryl disappearance in streams was similiar to the decay constants determined in the laboratory for carbaryl in river water ($0.017\ hour^{-1}$) and pond water ($0.028\ hour^{-1}$) (Eichelberger and Lichtenberg 1971, Kanazawa 1975). Marancik[15] noted that Atlantic salmon, brook trout, and slimy sculpins did not contain detectable residues of carbaryl 24, 48, or 168 hours after aerial application of 1.12 kg/ha to forests in the Eastern United States.

Bernhardt et al. (1978) conducted a much more intensive study of carbaryl in six streams in Washington State. Peak concentrations of 0.005, 0.013, 0.014, 0.020, 0.029, and 0.121 mg/liter were observed within the treated area in these six streams. Residues typically declined from peak levels within a few hours after application. Residue levels were much lower at downstream locations. In Squilchuck Creek, the stream that received the greatest exposure, residues of 100 to 120 mg/kg were measured in benthic organisms, 131 to 152 mg/kg in cutthroat trout, and 32 to 335 mg/kg in sediment. Residues were not found in these substrates at most other locations and, with the exception of the sediment, were not found 30 days after application in Squilchuck Creek.

Kenaga (1980b) predicted a bioaccumulation factor of 77 for carbaryl. In a model aquatic ecosystem, Kanazawa et al. (1975) reported higher values. They found bioaccumulation ratios of 2,000:1 to 4,000:1 for algae and duckweed, but values of only 100:1 to 500:1 for snails, catfish, and crayfish. The sediment in the system was the major repository for the chemical. The data suggest that carbaryl was tightly bound to soil particles and humic substances. Daphnia, which are extremely sensitive to carbaryl, were unaffected when placed in clean water that had been in contact with the sediments from this test for 3 days.

In a similar system, Sanborn (1974) did not detect any unmetabolized carbaryl in several components (including algae) of the ecosystem, although several metabolic products were prominent. Paris et al. (1975) found no measurable adsorption of carbaryl by microorganisms. Exposures of channel catfish for 28 days to $^{14}C$-carbaryl in the diet (2.8 mg/kg) or by bath (0.25 mg/liter) produced whole-body residues of 9 and 11 µg/kg, respectively. Within 28 days, 78 percent of these residues were eliminated by the diet-exposed fish but only 11 percent by the bath-exposed fish (Johnson and Finley 1980). Korn (1973) found that channel catfish did not accumulate carbaryl because they metabolize or excrete the compound. Marancik, citing Tompkins (1975, see footnote 15), reported that pumpkinseed (sunfish) exposed to 5 mg/liter Sevin for 2 hours accumulated 12.6 mg/kg in the tissue by the end of the exposure period, but eliminated 99.8 percent within 24 hours after exposure ended. These data suggest carbaryl bioaccumulation will be limited and its persistence brief.

---

15/ Unpublished report, "Effect of insecticides used for spruce budworm control in 1975 on fish," p. 11-34 in "1975 Cooperative Pilot Control Project of Dylox, Matacil, and Sumithion: forest spruce budworm control in Maine," by J. Marancik, U.S. Dep. Agric., For. Serv., Northeast. Area, Upper Darby, Penn., 1976.

Table 10--Acute toxicity of carbaryl to various invertebrate and fish species

| Test organism | Stage or weight | Temperature | 96-h $LC_{50}$ | Reference |
|---|---|---|---|---|
| | Grams | Degree C | mg/liter | |
| Invertebrates: | | | | |
| Daphnia pulex | I[1/] | 16 | 0.0064 | Johnson and Finley 1980 |
| Asellus sp. | M[2/] | 18 | .28 | Johnson and Finley 1980 |
| Gammarus fasciatus | M[2/] | 21 | .026 | Johnson and Finley 1980 |
| Pteronarcys sp. | YC[3/] | 16 | .0048 | Sanders and Cope 1968 |
| Fish: | | | | |
| Coho salmon | 1.0 | 13 | 4.34 | Johnson and Finley 1980 |
| Coho salmon | .6-1.7 | 13 | .764 | Macek and McAllister 1970 |
| Coho salmon | 2.7-4.1 | 20 | .997 | Katz 1961 |
| Cutthroat trout | .5 | 12 | 7.1 | Johnson and Finley 1980 |
| Cutthroat trout | -- | 9.5 | 6.7 | Woodward and Mauck 1980 |
| Rainbow trout | 1.5 | 12 | 1.95 | Macek and McAllister 1970 |
| Rainbow trout | 3.2 | 20 | 1.35 | Katz 1961 |
| Fathead minnow | .6-1.7 | 18 | 14.6 | Macek and McAllister 1970 |
| Fathead minnow | -- | -- | 6.7 | Henderson et al. 1960 |
| Fathead minnow | .8 | 18 | 14.6 | Johnson and Finley 1980 |
| Yellow perch | .6 | 12 | 5.1 | Johnson and Finley 1980 |
| Bluegill | 1.2 | 18 | 6.76 | Johnson and Finley 1980 |
| Bluegill | .7 | 17 | 4/39 | Johnson and Finley 1980 |
| Bluegill | .6-1.7 | 18 | 6.76 | Macek and McAllister 1970 |
| Bluegill | -- | -- | 5.3 | Henderson et al. 1960 |

-- = data not available.

1/ First instar.
2/ Mature.
3/ Second-year class.
4/ Contained in oil dispersion, 49 percent active ingredient.

## Toxci

Table 10 summarizes the toxicity of carbaryl for several invertebrate and fish species.

Courtemanch and Gibbs (1980) noted short- and long-term effects on stream invertebrates after spraying with carbaryl. The initial postspray response was an increase in drift, and the benthos showed significant declines among Plecoptera, Ephemeroptera, and Trichoptera. Plecoptera did not repopulate any treated stream by 60 days posttreatment. These findings are similar to those of Burdick et al. (1960), who reported a reduction in standing crop of total stream invertebrates after forest spraying with Sevin. The long-term effect of the chemical was most apparent on Plecoptera, especially in streams treated for two consecutive years.

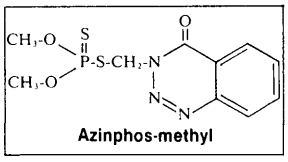
Stewart et al. (1967) studied the acute effects of carbaryl and its hydrolytic product l-naphtha1 on various marine species. They found that carbaryl was more toxic to larval and adult crustaceans than to larval and adult mollusks and juvenile fishes. Carbaryl was more toxic than l-naphthal. Carlson (1972) found that long-term exposure of fathead minnows to carbaryl at a concentration of 0.68 mg/liter caused adverse effects on survival and spawning.

The teratogenic effects of carbaryl and malathion on developing medaka embryos exposed in static tests were investigated by Solomon (1978) and Solomon and Weis (1979). The primary site of action of these insecticides was the circulatory system. Significant effects on circulatory anomalies were produced at concentrations of 5 mg/liter carbaryl and 20 mg/liter malathion.

Woodward and Mauck (1980) found stonefly naiads and amphipods were considerably more sensitive than cutthroat trout to carbaryl and thus would show the greatest impact after forest spraying that caused stream contamination. Johnson and Finley (1980) provided the following notes on carbaryl tests conducted at the Columbia Fisheries Research Laboratory:

> Little or no alteration in toxicity resulted when temperatures were increased from 10 to 21C for daphnids or from 7 to 17C for cutthroat trout and Atlantic salmon. Conversely, toxicity to brook trout and yellow perch was significantly increased (4- to 11-fold) by similar temperature increases. Increases in the pH of test solutions from 6.5 to 8.5 decreased toxicity to stoneflies by one-half. However, alkaline test solutions (pH 8.5 to 9.0) were 1.4 to 11.4 times more toxic to trout, salmon, and yellow perch than were test solutions with lower pH (6.5 to 7.5). Variations in hardness (12 to 300 mg/l) did not appreciably alter toxicity to scuds, trout, or yellow perch. Test solutions aged for 3 weeks were less toxic to stonefly naiads, yet more toxic to cutthroat trout.

## Azinphos-methyl



Azinphos-methyl

Azinphos-methyl is an organophosphate insecticide registered for use on a wide variety of plants to control many insect pests. It has been available since it was first registered for use on cotton in 1954; its most extensive use in forestry is in ground applications for control of seed and cone insects in seed production areas.

## Behavior in the Environment

A comprehensive review of the use and behavior of azinphos-methyl in American agriculture was made by Anderson et al. (1974). Inferences can be made to forestry uses. Azinphos-methyl is soluble to 29 mg/liter in water (25 °C) and is readily soluble in most organic solvents (except aliphatics).

The Chemagro Division of BayChem Corporation has conducted soil persistence studies of azinphos-methyl (Anderson et al. 1974). They report the average half-life of the compound was about 3 months, although it varied substantially in different soil types and in different geographic locations. In muck-sand soils of Florida, the reduction averaged more than 50 percent in 1 month. In clay soils of Louisiana, a SO-percent reduction required 3 months, but in Kansas clay soils, the time was between 2 and 3 months. Rainfall on the Louisiana plots was 24.4 cm; on the Kansas plots, it was 36.6 cm (Anderson et al. 1974). Haque and Freed (1974) estimate azinphos-methyl would leach less than 20 cm in soils receiving 150 cm of rainfall. Results of these tests suggest the persistence of azinphos-methyl and its mobility are not sufficent to result in either buildup of the compound in the soil or its transfer into ground water.

We did not find any published reports of azinphos-methyl in forest waters. There are unconfirmed reports of azinphos-methyl in surface and subsurface water draining from seed orchards on sandy soils in the southeastern United States. Its predominant pattern of use minimizes the likelihood that forestry uses of this insecticide will result in its residues entering forest surface waters in the West.

Meyer (1965) reported the half-life of azinphos-methyl would be about 2 days in the aquatic environment. In Meyer's study, the pH of the various ponds ranged from 7.2 to 8.0 and,

Table 11--Acute toxicity of azinphos-methyl to various invertebrate and fish species

| Test organism | Stage or weight | Temperature | 96-h $LC_{50}$ | Reference |
|---|---|---|---|---|
| | Grams | Degree C | mg/liter | |
| Invertebrates: | | | | |
| Asellus sp. | M[1] | 15 | 0.021 | Johnson and Finley 1980 |
| Gammarus fasciatus | M[1] | 21 | .00015 | Johnson and Finley 1980 |
| Pteronarcys sp. | YC[2] | 15 | .0019 | Johnson and Finley 1980 |
| Fish: | | | | |
| Coho salmon | 0.7 | 12 | .0061 | Johnson and Finley 1980 |
| Coho salmon | -- | 20 | .0042 | Katz 1961 |
| Coho salmon | .6-1.7 | 13 | .017 | Macek and McAllister 1970 |
| Rainbow trout | -- | 20 | .0032 | Katz 1961 |
| Rainbow trout | .6-1.7 | 13 | .0014 | Macek and McAllister 1970 |
| Rainbow trout | 1.0 | 12 | .0043 | Johnson and Finley 1980 |
| Fathead minnow | 1-2 | 25 | .0093 | Henderson et al. 1960 |
| Fathead minnow | .6-1.7 | 18 | .235 | Macek and McAllister 1970 |
| Fathead minnow | 1.2 | 18 | .235 | Johnson and Finley 1980 |
| Bluegill | 1.5 | 18 | .022 | Johnson and Finley 1980 |
| Bluegill | .6-1.7 | 18 | .022 | Macek and McAllister 1970 |
| Bluegill | 1-2 | 25 | .0052 | Henderson et al. 1960 |
| Largemouth bass | .6-1.7 | 18 | .005 | Macek and McAllister 1970 |
| Largemouth bass | .9 | 18 | .0048 | Johnson and Finley 1980 |

-- = data not available.

1/Mature.
2/Second-year class.

after application of 1 mg/liter azinphos-methyl, the residues dropped from 0.8 mg/liter at 4 hours to 0.4 mg/liter at 48 hours after application. At 4 clays, the residue level was 0.1 mg/liter and was nondetectable at 14 days. Flint et al.[16] reported a half-life of 1.2 days in an outdoor pond. The degradation was more rapid where both sunlight and microorganisms were active than in indoor tests. Liang and Lichtenstein (1972) showed azinphos-methyl was subject to photodecomposition in aquatic systems. These tests suggest that the decomposition of azinphos-methyl in an aquatic environment is relatively rapid, and accumulation is not expected. Iwata et al. (1977) reported that disappearance of azinphos-methyl followed first-order kinetics in a silty clay loam soil with a 100-fold decrease in concentration in 105 days.

Azinphos-methyl should show little potential for bioaccumulation. In dairy cattle, it appears to be rapidly excreted (Everett et al. 1966, Loeffler et al. 1966). No residues are found in milk 1 to 2 days after treated feed was withdrawn. This information, combined with what is known of the physical properties of azinphos-methyl, suggests that its bioaccumulation is not likely to be extensive, and it should clear rapidly from organisms once exposure ceases.

## Toxci

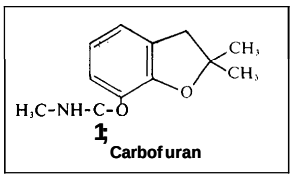
Several researchers have studied the toxicity of azinphos-methyl to invertebrates and fishes (Henderson et al. 1960, Katz 1961, Macek and McAllister 1970, Johnson and Finley 1980). It is 2 to 10 times more toxic than malathion. Table 11 provides a summary of its acute toxicity to a variety of invertebrates and fishes.

16/Unpublished report, "Soil runoff, leaching, and adsorption and water stability studies with Guthion," Rep. 28936, by D. R. Flint, D. D. Church, H. R. Shaw, and J. Armour, Chemagro Corp., Kansas City, Mo., 1970.

Johnson and Finley (1980) noted that variations in test temperatures from 2 °C to 18 °C for rainbow trout and 12 °C to 22 °C for bluegills produced no change in toxicity of azinphos-methyl at the lower temperatures and a 2-fold increase at the higher temperatures. Yellow perch became substantially more susceptible with an increase in temperature; 96-hour $LC_{50}$'s decreased from 0.04 to 0.0024 mg/liter when temperature was increased from 7 °C to 22 °C. Variations in water hardness from 12 to 300 mg/liter produced no change in toxicity to scuds or fish. Alkaline solutions (pH 8.5 to 9.0) were slightly less toxic to fish than more acidic solutions (pH 6.5 to 7.5). Aqueous degradation from 1 to 3 weeks produced a 1.3- to 2-fold increase in 96-hour $LC_{50}$'s for Atlantic salmon and yellow perch. Atlantic salmon eggs were highly tolerant to poisoning (11-day $LC_{50}$ greater than 50 mg/liter). The susceptibility of yolk-sac fry equaled that of fingerlings. Time-independent $LC_{50}$'s ($TILC_{50}$) were 0.000 23, 0.000 29, and 0.000 32 mg/liter for Atlantic salmon, bluegills, and yellow perch, respectively. The $TILC_{50}$ is a statistical estimate of the toxicant concentration at which 50 percent of the test population would be expected to survive a long-term exposure. Cumulative toxicity indexes varied from 10.9 to 20.5, indicating a moderate to high degree of cumulative action (for an organophosphate).

The cumulative toxicity index is the numerical ratio of the 96-hour $LC_{50}$ to the $TILC_{50}$ for a chemical. This ratio can serve as an estimate of the cumulative action of a toxicant. For example, a ratio of 2:1 suggests little cumulative action. Adelman et al. (1976) found that 0.000 51 mg/liter but not 0.000 33 mg/liter drastically reduced egg production of the fathead minnow, but caused no other apparent adverse effects.

## Carbofuran



1; Carbofuran

Carbofuran is a broad-spectrum carbamate insecticide. It has major registrations for a wide variety of soil and foliar insect pests in numerous agricultural crops. Forestry uses include control of seed and cone insects in nurseries and seed orchards and as a root dip at time of planting (see footnote 3).

### Behavior in the Environment

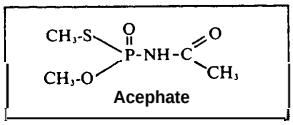
Carbofuran is soluble to 700 mg/liter in water (25 °C) and has a vapor pressure of $2x10^{-5}$ mmHg (33 °C). The moderately low vapor pressure suggests low volatility from soil. Tu and Miles (1976) classed carbofuran as slightly volatile (the least volatile group) in soil at 20 °C. Carbofuran, like the other carbamate insecticides, disappears rapidly from soil. Fuhremann and Lichtenstein (1980) measured about 60- and 90-percent losses of carbofuran in 14 days from loam and sandy soils in which oats were germinated and grown. Caro et al. (1973) reported it disappeared from soil by apparent first-order kinetics with half lives ranging from 46 to 117 days. The reaction is likely mediated by both chemical and biological processes. Getzin (1973) reported half lives ranging from 3 to 50 weeks in different sterile and nonsterile soils. The loss was 7 to 10 times faster in alkaline soils (pH 7.9) than in acid or neutral soils (pH 4.3 to 6.5). Goring et al. (1975) included carbofuran in the "moderately persistent in soil" group of pesticides (half lives of 1.5 to 6 months). Sanborn (1974) did not detect a bioaccumulation of carbofuran in a multicomponent model ecosystem, although evidence of substantial degradation on tight binding appeared in all components. Additional specific data on carbofuran are lacking, but it is expected to behave similarly to other carbamate insecticides.

We did not find any published reports of carbofuran in forest waters. There are unconfirmed reports of carbofuran in surface and subsurface water draining from seed orchards on sandy soils in the southeastern United States. Its predominant pattern of use minimizes the likelihood that forestry uses of this insecticide will result in its residues entering forest surface waters in the West.

### Toxicity

Data on carbofuran toxicity are limited. It is considerably more toxic than the other carbamate insecticides (such as carbaryl). Johnson and Finley (1980) summarized the work carried out at the National Fish Research Laboratory, Columbia, Missouri. Salmonids had 96-hour $LC_{50}$'s of 0.164 to 0.560 mg/liter, cyprinids (fathead minnow) 0.872 mg/liter, and percids (yellow perch) 0.147 mg/liter. Parrish et al. (1977) calculated the 96-hour $LC_{50}$ of carbofuran to the sheepshead minnow to be 0.386 mg/liter. Adults exposed to concentrations equal to or greater than 0.049 mg/liter showed significantly greater mortality than control fish during the 131-day study. Hatching success from eggs spawned by fish exposed to 0.049 mg/liter was significantly less than for eggs of nonexposed fish. Mortality of fry hatched from eggs spawned by fish exposed to 0.23 and 0.049 mg/liter was significantly greater than control fry mortality. Davey et al. (1976) noted that carbofuran was the least toxic of five rice-field pesticides to the mosquitofish and green sunfish. Klaassen and Kadoum (1979) found that carbofuran was present in the water and mud of a farm pond only immediately after application of 0.025 mg/liter. No adverse effects were observed.

## Acephate



Acephate

Acephate is a moderately persistent, organophosphate insecticide. It is used to control defoliating insects on several agricultural crops. In forestry, it is used to control seed and cone insects in seed orchards and the western spruce budworm in forest stands (1.5 kg/ha) (see footnote 3).

### Behavior in the Environment

Willcox and Coffey[17] summarized the behavior and the toxicity of acephate. It is degraded in soil by microbial action. Chevron[18] reported that in soil from nine locations across the United States, acephate had a half-life ranging from 0.5 to 13 days when the soil was fortified to 1 or 10 mg/kg. The longest persistence was in a highly organic muck soil. In eight of the nine soils, the half-life ranged from 0.5 to 4 days. In the field, acephate residues declined from 5.5 mg/kg in 1 day to 0.03 mg/kg in 20 days and to less than 0.02 mg/kg 70 days after aerial application of 0.56 kg/ha in Pennsylvania. Soil residues were not detected after 3 days (Devine 1975). Szeto et al. (1978) reported a 90-percent decrease in acephate in 10 days in open forest floor, but only about a 70-percent decrease in semiopen or densely covered areas in a Pacific Northwest forest. No residues were detected 30 days after application.

---

[17] Unpublished report, "Environmental impact of acephate insecticide (Orthene)," by H. Willcox III and T. Coffey, Jr., U.S. Dep. Agric., For. Serv., State and Priv. For., For. Insect and Dis. Manage., Northeast. Area, Upper Darby, Penn., 1977.

[18] Unpublished report, "The impact of Orthene on the environment," prepared by Chevron Chem. Co., Richmond, Calif., 1973.

In laboratory studies, acephate (freshly added) was readily leached in soil. Aged soil residues are much less mobile. The short persistence of acephate in biologically active soils is believed to minimize the likelihood of significant movement to ground water. According to Chevron (see footnote 18) acephate is hydrolyzed slowly in water (half-life at 21 °C: 46 days at pH 7.0, 55 days at pH 5.0, and 16 days at pH 9.0). In tests conducted to determine if acephate would be moved by runoff water, residues were found in both runoff water and associated soil particles. Sediments and submerged vegetation will also absorb acephate, but the residue levels decline rapidly.

Flavell et al. (1977) summarized the results of aquatic monitoring of pilot-scale applications of acephate to control the western spruce budworm in three, 405-ha blocks in Montana in 1976. Stream water collected the day of application from five streams in the treated areas contained acephate residues that ranged from 0.003 to 0.961 mg/liter. The average peak concentration for all five streams was 0.466 mg/liter (only one sample of the 69 collected in this study contained a concentration greater than 0.5 mg/liter). Rapid reduction in concentration occurred, typically 10-fold in 2 to 6 hours. Residues averaged 0.065 mg/kg (range 0.026 to 0.139 mg/kg) in fish and 0.036 mg/kg (range 0.0 to 0.107 mg/kg) in insects.

Rabeni and Gibbs[19] found 0.135 and 0.113 mg/liter acephate in two streams 1 hour after application of 0.56 kg/ha. After 1 day, these levels were reduced to 0.013 and 0.065 mg/liter. No residues were found in four species of fish or in bottom sediments from a pond 2 weeks after treatment with acephate at 0.56 kg/ha (Bocsor and O'Connor 1975).

Sanborn (1974) reported acephate did not accumulate in algae, clams, crabs, Daphnia, Elodea, mosquitofish, or snails in a model ecosystem that had both terrestrial and aquatic components. The acephate was applied at 1.12 kg/ha to the terrestrial portion of the system. The data also indicate more than 95-percent decomposition of acephate in the system in 33 days. Chevron (see footnote 18) reported acephate applied to achieve 0.1 mg/liter in water was not detected 6 weeks later.

Bluegills were continuously exposed to 1.0 or 0.01 mg/liter acephate ($^{14}$C-labeled) for 35 days, and tissue samples analyzed periodically to determine the rate and extent of $^{14}$C-residue accumulation. After the exposure period, the fish were transferred to untreated water for 14 days.[20] The maximum tissue concentration of radio-labeled residues in the edible portion was about 10 times the concentration in water. Upon transfer to uncontaminated water, fish exposed at both levels eliminated more than 50 percent of the residues in the edible portion within 3 days. These data indicate a low potential for bioaccumulation.

## Toxicity

The effects on stream fishes and invertebrates of an operational spraying of acephate to suppress spruce budworm were investigated by Rabeni and Stanley (1979). Acephate reached its maximum concentration of 0.14 mg/liter in North Brook and

---

[19] Unpublished report, "The impact of Orthene, a spruce budworm insecticide, on aquatic macroinvertebrates in Maine, 1977," Rep. NA-FR-7, by C. F. Rabeni and K. E. Gibbs, U.S. Dep. Agric., For. Serv., Northeast. Area, Broomall, Penn., 1979.

[20] Unpublished report, "Exposure of fish to $^{14}$C-labelled Orthene: accumulation, distribution and elimination of residues," by B. O. Sleight, Bionomics Inc., Wareham, Mass., 1972.

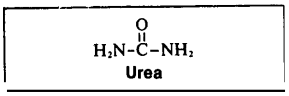
0.113 mg/liter in South Brook within an hour of spraying, and residues remained in streamwater for at least 2 days. The authors concluded that acephate caused relatively minor, short-term perturbations to the stream ecosystem (drift of macroinvertebrates increased; the standing crop of most invertebrates remained unchanged; brain AChE activity was depressed in suckers, but not in trout or salmon; and brook trout altered their diet, but growth was not affected). The authors drew these conclusions because the effects observed were either transitory or were not adverse. If the streams were adversely affected by spray drift, it was not detected by the methods used.

Willcox and Coffey (see footnote 17) summarized the pertinent literature on environmental impacts of acephate insecticide. Acephate has an extremely low toxicity to fish (rainbow trout, 96-hour $LC_{50}$ of 1000 mg/liter); though more toxic for invertebrates, no affects to Plecoptera or Ephemeroptera were recognized after treatment at 0.56 kg a.i./ha in a Pennsylvania stream and pond. Chevron[21/] reported 96-hour $LC_{50}$'s of acephate to various fish species from 1000 mg/liter for rainbow trout to 9550 mg/liter for goldfish.

Johnson and Finley (1980) reported 96-nour $LC_{50}$'s of 1100 mg/liter for rainbow trout exposed to the technical material (94 percent), but 730 mg/liter when the soluble powder (75 percent) was used. The 96-hour $LC_{50}$ for various invertebrates ranged from 9.5 mg/liter (Plecoptera) to 1000 mg/liter (Diptera larvae),

Woodward and Mauck (1980) suggested that acephate would be the most acceptable of five forest insecticides tested from the standpoint of impact on nontarget aquatic organisms, It was nontoxic to cutthroat trout, and the lowest concentration toxic to aquatic invertebrates was much higher than the concentrations that could be expected in postspray water residue.

## FERTILIZERS




Nitrogen (as urea) is the element most commonly applied as a fertilizer in Pacific Northwest forests. Application rates vary, but are usually 168 to 224 kg/ha urea-nitrogen (Moore and Norris 1974). Bengtson (1979) reviewed the use of fertilizers in forestry.

### Behavior in the Environment

Urea is highly water soluble and is readily moved from surface deposits into the forest floor and soil. Hydrolysis to ammonium ion is usually complete in 2 weeks. Ammonium nitrogen may be adsorbed by humic substances, held as an exchangeable cation, incorporated by soil microorganisms, or taken up by forest vegetation. Usually, it is quickly distributed through the biomass and is cycled within the forest ecosystem (Moore and Norris 1974).

Fertilizer nitrogen may enter the aquatic environment by the same routes described for pesticides. The highest concentrations of urea result from direct application to stream surfaces. Mobilization in ephemeral stream channels and subsurface drainage are important routes of entry for urea transformation products, primarily from the riparian zone.

---

21/ Unpublished report, "Orthene insecticide - technical information," prepared by Chevron Chem. Co., Richmond, Calif., 1976.

Although forest soils are excellent filters for plant nutrients, increased levels of various nitrogen species have been measured in several stream systems in Pacific Northwest forests. In one of the more intensive efforts, Moore (1970) measured the amounts and forms of nitrogen entering streams during and after aerial application of 224 kg/ha urea-nitrogen in a southwestern Oregon forest. Urea concentrations increased slowly and reached a maximum of 0.39 mg/liter urea-N 48 hours after application started. Ammonium-N increased slightly above background but did not reach 0.10 mg/liter. Nitrate-N reached a peak concentration of 0.168 mg/liter in 72 hours and was 0.140 mg/liter at the end of 2 weeks. Nitrite-N was not detected. Only 0.01 percent of the nitrogen applied to the watershed was found in the stream up to 15 weeks after application.

Low streamflow because of limited precipitation in summer and fall resulted in essentially no loss of applied nitrogen during the next 24 weeks. Early storm activity in November brought the soil moisture back to maximum storage capacity, and in December the nitrate-N concentration in samples for the fertilized watershed reached a second peak of 0.177 mg/liter. Both streamflow and nitrate-N levels remained high through December and January, resulting in the loss of an additional 23.8 kg applied nitrogen. This is 92 percent of the total amount of fertilizer nitrogen lost during the 1st year.

Total loss of applied nitrogen from the 68-ha, fertilized watershed during the 1st year amounted to 25.85 kg (table 12). Over the same period, the total amount of soluble inorganic nitrogen lost from the 49-ha control watershed was 2.15 kg. Data for soluble organic nitrogen, total phosphorus, silica, and exchangeable cation content of the stream samples, including sodium, potassium, calcium, magnesium, iron, manganese, and aluminum, indicate that nitrogen fertilization had no apparent effect on loss of native soil nitrogen or other plant nutrients. Movement may have occurred in the soil profile, but no measurable change occurred in stream-water quality.

Table 12--Nitrogen lost from treated Watershed 2 and untreated Watershed 4, South Umpqua Experimental Forest, Juring the 1st year after application of 224 kilograms urea-N per hectare[1/]

| | Urea-N | $NH_3$-N | $NO_3$-N | Total |
|---|---|---|---|---|
| | | Kilograms N | | |
| Watershed 2 | 0.65 | 0.28 | 27.09 | 28.03 |
| Watershed 4 | .02 | .06 | 2.07 | 2.15 |
| Net loss | .63 | .22 | 25.02 | 25.88 |
| Percent of total loss | 2.44 | .86 | 96.70 | 100.00 |

1/From Moore (1971).

Similar data were reported by Moore (1975b) for several other monitoring studies conducted throughout the Douglas-fir region. Peak concentrations of urea-, ammonium-, and nitrate-nitrogen measured at each site monitored after fertilization are given in table 13. Monitoring varied from a few weeks after treatment to 6 or 7 months and, in a few studies, it continued at least a full year. Most sampling continued until the forms of nitrogen being measured had decreased to near pretreatment levels.

Increases in the concentration of urea-N ranged from very low to a high of 44.4 mg/liter. These increases were almost entirely because of direct application to surface water, and the peak concentration reached is directly proportional to the amount of open surface water in the treated unit. The high peak concentrations of urea-N measured in Dollar Creek and

Table 13--Peak concentrations of nitrogen in stream samples after forest fertilization with urea in Alaska, Idaho, Oregon, and Washington[1/]

| Study site | Rate of application | Date of application | Treated area | Concentration of nitrogen | | |
|---|---|---|---|---|---|---|
| | | | | Urea-N | $NH_3$-N[2/] | $NO_3$-N |
| | kg N/ha | | Hectares | mg/liter | | |
| Burns Creek | 3/56 | Oct. 1970 | 562 | 0 | 0 | 0.068 |
| Canyon Creek | 224 | Oct. 1969 | 1346 | 15.20 | -- | .80 |
| Coyote Creek | 224 | Mar. 1970 | 68 | 1.39 | .048 | .177 |
| Crabtree Creek | 224 | May 1969 | 230 | 24.00 | .080 | .25 |
| Dollar Creek | 224 | Apr. 1971 | 34 | 44.40 | .490 | .13 |
| Elochoman River | 224 | Nov. 1969 | 297 | 19.10 | -- | 4.00 |
| Fairchilds Creek | 224 | Apr. 1972 | 192 | 23.40 | .280 | .828 |
| Falls Creek | 213 | May 1970 | 263 | -- | 1.28 | 1.67 |
| Jackson Creek | 168 | May 1969 | 95 | .09 | .044 | .116 |
| Jimmycomelately Creek | 224 | Apr. 1970 | 49 | .71 | .040 | .042 |
| McCree Creek | 56 | Oct. 1970 | 513 | .62 | 0 | .210 |
| Mica Creek | 224 | Sept. 1972 | 47 | .30 | 0 | .28 |
| Mill Creek | 224 | Dec. 1969 | 228 | .68 | .12 | 1.32 |
| Nelson Creek | 224 | Apr. 1970 | 38 | 8.60 | .32 | 2.10 |
| Newaukum River | 168 | Sept. 1971 | 2463 | .26 | .008 | .438 |
| Pat Creek | 224 | Apr. 1972 | 243 | 3.26 | .079 | .388 |
| Quartz Creek | 224 | May 1972 | 51 | 1.75 | trace | .70 |
| Roaring Creek | 224 | Mar. 1972 | 267 | .76 | .040 | .210 |
| Row River | 168 | Oct. 1972 | 2630 | .13 | .022 | .044 |
| Skookumchuck River | 168 | Sept. 1969 | 191 | 2.63 | .026 | .085 |
| Spencer Creek | 224 | Nov. 1972 | 3108 | .37 | .123 | 4/.005 |
| Tahuya River | 224 | Oct. 1972 | 1602 | 27.20 | 1.40 | 1.83 |
| Thrash Creek | 5/224 | May 1974 | 121 | -- | .06 | 1.88 |
| Three Lakes | 213 | May 1970 | 69 | -- | .13 | 2.36 |
| Trapper Creek | 224 | Apr. 1970 | 64 | .70 | .010 | .121 |
| Trout Creek | 224 | Mar. 1968 | 648 | 14.00 | .700 | .160 |
| Turner Creek | 224 | Mar. 1972 | 352 | 4.36 | .046 | .243 |
| Waddell Creek | 224 | Dec. 1969 | 600 | 2.48 | .340 | .99 |
| Wishbone Creek | 224 | May 1972 | 46 | .30 | 0 | .28 |

-- = data not available.

1/ From Moore (1975b).
2/ Includes both ionized ($NH_4^+$) and un-ionized ($NH_3$) ammonia-nitrogen.
3/ Applied as ammonium sulfate.
4/ In this study, no increase occurred in $NO_3$-N. Concentration given is background level.
5/ Applied as amnonium nitrate.

Fairchilds Creek were because of greatly expanded stream-drainage systems caused by spring runoff of snowmelt. A buffer strip was left along Fairchilds Creek, but the expanded system of small feeder streams still resulted in direct application of fertilizer to a much larger area of surface water than would normally occur.

The peak concentrations of urea-N did not persist for more than a few hours. Concentrations characteristically reached a peak the day of application and then decreased rapidly. Within 3 to 5 days after application, urea-N in the stream returned to pretreatment concentrations.

Ammonium-nitrogen levels also increased as a result of direct application of urea fertilizer to open water. Urea is readily hydrolyzed to ammonium-N in the stream system. Urea applied to forest floor and soil surfaces will not reach the stream, because it hydrolyzes rapidly to ammonium carbonate and is then held on cation-exchange sites in the soil and forest floor like any other ammonium salt.

Peak concentrations of nitrate–N in streams after forest fertilization ranged from no increase in Spencer Creek to a maximum of 4.00 mg/liter in a tributary stream of the Elochoman River. The concentration of nitrate–N in stream samples usually reaches a peak 2 to 4 days after spring application of fertilizer. Concentrations then decrease, but may remain above background for 6 to 8 weeks. Losses of applied nitrogen are very small because the maximum concentrations reached are generally below 1 mg/liter nitrate–N, and streamflow rapidly decreases with the onset of the dry summer season. About half of the applied nitrogen entering the stream during the first 30 days is from direct application and is measured as urea– and ammonium–N. The other half enters as nitrate. In early fertilization projects where buffer strips were either inadequate or not used, estimated total loss was between 2 and 3 percent of the applied nitrogen. In later projects, however, where direct application to open surface water was minimized by buffer strips along the main streams and tributaries, measured losses were less than 0.5 percent.

In monitoring studies where sampling has continued through the first winter after fertilization, additional peaks in the concentration of nitrate–N have been measured. These peaks usually coincide with intense winter storms, and the concentration drops sharply between them. Maximum concentrations measured were low and tended to decrease with each successive storm (Moore 1971).

Patterns of nitrate–N loss to streams after early fall (September, October) application of fertilizer are similar to those after spring application. Peak concentrations measured during winter storms may not be as high, however, because of less time during warm weather for conversion of nitrogen to nitrate. The initial peak in nitrate–N concentration also occurs after fertilizer application in November and December. Subsequent peaks during winter storms are similar to those in streams draining untreated areas. Additional losses as nitrate–N may occur the next winter, however.

## Toxicity

Ammonia is one of the toxic breakdown products of fertilizers. U. S. Environmental Protection Agency (1976) stated:

> Ammonia is a pungent, colorless, gaseous, alkaline compound of nitrogen and hydrogen that is highly soluble in water. It is a biologically active compound present in most waters as a normal biological degradation product of nitrogenous organic matter. It may also reach ground and surface waters through discharge of industrial wastes containing ammonia as a byproduct, or wastes from industrial processes using "ammonia water."
>
> When ammonia dissolves in water, some of the ammonia reacts with the water to form ammonium ions. A chemical equilibrium is established which contains un–ionized ammonia ($NH_3$), ionized ammonia ($NH_4^+$), and hydroxide ions ($OH^-$). . . . The toxicity of ammonia is very much dependent upon pH as well as the concentration of total ammonia. Other factors also affect the concentration of $NH_3$ in water solutions, the most important of which are temperature and ionic strength. . . .
>
> It has been known since early in this century that ammonia is toxic to fishes and that the toxicity varies with the pH of the water. . . .

In most natural waters, the pH range is such that the $NH_4^+$ fraction of ammonia predominates; however, in highly alkaline waters, the $NH_3$ fraction can reach toxic levels. Many laboratory experiments of relatively short duration have demonstrated that the lethal concentrations for a variety of fish species are in the range of 0.2 to 2.0 mg/l $NH_3$, with trout being the most sensitive and carp the most resistant. Although coarse fish such as carp survive longer in toxic solutions than do salmonids, the difference in sensitivity among fish species to prolonged exposure is probably small. . . . The lowest lethal concentration reported for salmonids is 0.2 mg/l NH3 for rainbow trout (Salmo gairdneri Liebmann, 1960). The toxic concentration for Atlantic salmon smolts, (Salmo salar) (Herbert and Shurben, 1965), and for rainbow trout (Ball, 1967) was found to be only slightly higher. Although a concentration of NHB below 0.2 mg/l may not kill a significant proportion of a fish population, such concentration may still exert an adverse physiological or histopathological effect (Lloyd and Orr, 1969, Smith and Piper, 1975). . . . Burrows (1964) found progressive gill hyperplasia in fingerling chinook salmon, (Oncorhynchus tshawytscha), during a 6-week exposure to a total ammonia concentration (expressed as $NH_4$) of 0.3 mg/l (0.002 mg/l $NH_3$), which was the lowest concentration applied.

Another breakdown product of fertilizers is nitrate. The U.S. Environmental Protection Agency (1976) has only a recommended standard rather than a mandatory standard because nitrate has long been considered almost nontoxic to fish. Westin (1974) reported a 96-hour medium tolerance limit ($TL_m$) of 5800 mg/liter nitrate for chinook salmon fingerlings and 6000 mg/liter for rainbow trout fingerlings. Few data are available on other life stages, but Kincheloe et al. (1979) reported on nitrate exposure to egg and early fry stages of chinook and coho salmon, steelhead, rainbow, and Lahontan cutthroat trout. They found that nitrate (sodium) was mildly toxic to the early life stages of several salmonids. Coho salmon eggs and fry were resistant to nitrate toxicity. Chinook salmon, rainbow, Lahontan cutthroat, and steelhead trout eggs and fry exhibited mortalities during exposure to nitrate concentrations as low as 5 mg/liter. A complication was Saprolegnia (fungus) infestations of the eggs. The authors believe that nitrate levels of 10 mg/liter (2 mg/liter nitrate-N) in surface waters of low total hardness would limit survival of some salmonid fish populations because of impaired reproductive success.

Ammonium fertilizers have also been used to enrich fish ponds to increase productivity (Swingle 1947, Boyd and Sowles 1978). The application of ammonium fertilizers can lead to losses of alkalinity (Hunt and Boyd 1981) that subsequently require addition of agricultural limestone or liming to neutralize the acidity.

Stay et al. (1979) studied the effects of fertilizing a second-growth Douglas-fir forest with 224 kg urea-N/ha. Although they found sharp increases of urea in the stream during fertilization because of direct application, all nitrogen forms returned to near background levels shortly afterward. A 2-month rainbow trout bioassay showed no mortalities that could be attributed to byproducts or contaminants of urea. Changes in benthic and drifting invertebrates could not be related to the fertilization project.

## FIRE RETARDANTS

Modern chemical fire retardants are a complex mixture. The most abundant constituent (responsible for the fire-retardant action) is diammonium phosphate (Phos-Chek products), ammonium sulfate (Fire-Trol 100), or ammonium polyphosphate (other Fire-Trol products). Numerous other constituents are in the formulations applied in the field, however (tables 14 and 15). The behavior and impact of chemical fire retardants have not been extensively studied. Douglas (1974) reviewed this topic, Van Meter and Hardy (1975) conducted an initial simulation study of retardant distribution in streams, and C. W. George reviewed the literature on retardant toxicity to aquatics (see footnote 2).

The principal toxic ingredient of the chemical fire retardants currently in use is believed to be an ammonium salt (in the form of un-ionized ammonia, $NH_3$) (see footnote 2). A recent analysis, however, suggested that photolysis of the ferrocyanide in several Fire-Trol retardant formulations may lead to production of sufficient cyanide to be the primary toxicant in these products.[22/]

[22/] Unpublished draft environmental assessment report, "Toxicity and environmental effects of fire retardant chemicals," prepared by U.S. Dep. Agric., For. Serv., Pac. Northwest Reg., Portland, Oreg., 1979.

### Behavior in the Environment

The behavior of ammonium and ammonia in the environment is similar to that of the nitrogen derived from the hydrolysis of urea described in the previous section on urea fertilizer. Fire-Trol 931L and 934L are also ammonium-based fire retardants, but they contain ferrocyanide as a corrosion inhibitor. According to Burdick and Lipschuetz (1948, quoting Baudish and Bass 1922), ferrocyanide solutions "are decomposable to some extent under the influence of light" (sunlight). The product of photolysis is cyanide:

$$Fe(CN)_6^{-4} \text{ yields } Fe(CN)_5^{-3} + CN^- .$$

The $CN^-$ then reacts with water: $CN^- + H_2O$ yields $HCN + OH^-$, in an equilibrium reaction that is strongly pH dependent. $CN^-$ is relatively low in toxicity to aquatic species but HCN is quite toxic (analogous to the difference between $NH_4^+$ and $NH_3$). At pH 9.3, about half the cyanide will be HCN and half $CN^-$ (D'amore and Bellorno 1958). The environmental significance of this reaction was brought to light by a fish kill in New York in 1948. The fish kill extended over 12 miles of river and was associated with industrial discharge of ferrocyanides and ferricyanides (Burdick and Lipschuetz 1948). Investigators showed that the ferrocyanide and ferricyanide concentrations were below those generally accepted as lethal.

Table 14--Typical composition of some chemical fire retardants[1/]

| Chemical name | Empirical formula | Percent by weight in dry powder or liquid concentration | Parts per million in mixed retardant |
|---|---|---|---|
| Phos-Chek XAR,[2/] Monsanto | | | |
| Diammonium phosphate | $(NH_4)_2HPO_4$ | 85-90 | 1.02-1.08x10$^5$ |
| Modified polysaccharide | -- | 5-10 | 6,000-12,000 |
| Iron oxide | $Fe_2O_3$ | 0-1 | 0-1,200 |
| Corrosion inhibitors: | | | |
| Soluble salt of-- | | | |
| Silicofluoride | $SiF_6^{-2}$ | .25-.58 | 300-700 |
| Thiosulfate | $S_2O_3$ | .01-5 | 1,200-6,000 |
| 2-Mercapto-benzothiazole | $C_6H_4SCSH:N$ | .0005-2 | 600-2,400 |
| Flow conditioner (insoluble) | -- | 2-4 | 2,400-4,800 |
| Phos-Chek 259R-(1.14 lb/gal), Monsanto | | | |
| Diammonium phosphate | $(NH_4)_2HPO_4$ | 92 | 111,000 |
| Modified polysaccharide | -- | 2.5 | 3,000 |
| Iron oxide | $Fe_2O_3$ | .75 | 902 |
| Corrosion inhibitors: | | | |
| Soluble salt of-- | | | |
| Silicofluoride | $SiF_6^{-2}$ | 1.47 | 1,768 |
| Thiosulfate | $S_2O_3$ | .71 | 854 |
| 2-Mercapto-benzothiazole | $C_6H_4SCSH:N$ | .20 | 241 |
| Flow conditioner (insoluble) | -- | 2.0 | 2,405 |
| Phos-Chek 259R-(1.6 lb/gal), Monsanto | | | |
| Diammonium phosphate | $(NH_4)_2HPO_4$ | 92 | 148,000 |
| Modified polysaccharide | -- | 2.5 | 4,024 |
| Iron oxide | $Fe_2O_3$ | .75 | 1,207 |
| Corrosion inhibitors: | | | |
| Soluble salt of-- | | | |
| Silicofluoride | $SiF_6^{-2}$ | 1.47 | 2,366 |
| Thiosulfate | $S_2O_3$ | .71 | 1,143 |
| 2-Mercapto-benzothiazole | $C_6H_4SCSH:N$ | .20 | 322 |
| Flow conditioner (insoluble) | -- | 2.0 | 3,219 |

Table 14--Typical composition of some chemical fire retardants[1/], continued

| Chemical name | Empirical fonnula | Percent by weight in dry powder or liquid concentration | Parts per million in mixed retardant |
|---|---|---|---|
| Fire-Trol 100,[3/] Chemonics | | | |
| Ammonium sulfate | $(NH_4)_2SO_4$ | 62 | 169,000 |
| Attapulgite clay | -- | 36 | 90,000 |
| Iron oxide | $Fe_2O_3$ | 1 | 2,500 |
| Corrosion inhibitors: | | | |
| Soluble salt of-- | | | |
| Dichromate | $CrO_7^{-2}$ | 1 | 2,500 |
| Fire-Trol 931L,[4/] Chemonics | | | |
| Ammonium polyphosphate (10-34-0) | -- | 93 | 249,000 |
| Attapulgite clay | -- | 4 | 10,700 |
| Iron oxide | $Fe_2O_3$ | 1-2 | 2,600-5,400 |
| Corrosion inhibitors: | | | |
| Sodium ferrocyanide | $Na_4Fe(CN)_6$ | 1-2 | 2,600-5,400 |
| A dye[5/] | -- | -- | -- |
| Fire-Trol 934L, Chemonics | | | |
| Ammonium polyphosphate (10-34-0) | -- | 97.5-98 | 258,000 |
| Sodium ferrocyanide | $Na_4Fe(CN)_6$ | 1.5 | 3,900 |
| Surfactant and water[5/] | -- | -- | -- |

-- = information not available.

1/ From Chemical Economics Handbook, January 1978, Menlo Park, California, Phosphorus Products, Page L.
2/ U.S. Patent 3,024,100 (March 6, 19621, Corrosion-Inhibited Liquid Fertilizer Compositions, granted to Langguth and Seifter and assigned to Monsanto Chemical Company. U.S. Patent 3,342,749 (September 19, 1967), Corrosion-Inhibited Phosphate Solutions, granted to Handleman, Groves, and Langguth and assigned to Monsanto Company.
3/ U.S. Patent 3,196,108 (July 20, 1965), Fire Supressing Composition for Aerial Application, granted to Nelson and assigned to Arizona Agrochemical Corp. (now Chemical Industries).
4/ U.S. Patent 3,960,735 (June 1, 1976), Corrosion-Inhibited Polyphosphate Compositions, granted to Lacey and assigned to Early California Industries, Inc.
5/ The formulation and concentration of these compounds were furnished by Chemonics and are not included because of their proprietary natures.

Table 15--Concentration of specific ions in some chemical fire retardants (estimated from data in table 14)[1/]

| Specific ion | Formula | Parts per million in mixed retardant |
|---|---|---|
| Phos-Chek XA, Monsanto: | | |
| Ammonium + ammonia[2/] | $NH_3$ | 26,300-27,900 |
| Phosphate | $PO_4^{-3}$ | 73,000-77,700 |
| Silicofluoride | $SiF_6^{-2}$ | 300-700 |
| Thiosulfate | $S_2O_3^{-2}$ | 1,200-6,000 |
| Mercaptobenzothiazole (MBT) | $C_7H_5NS_2$ | 600-2,400 |
| Phos-Chek 259R (1.14 lb/gal), Monsanto: | | |
| Ammonium + ammonia[2/] | $NH_3$ | 28,600 |
| Phosphate | $PO_4^{-3}$ | 79,800 |
| Silicofluoride | $SiF_6^{-2}$ | 1,768 |
| Thiosulfate | $S_2O_3^{-2}$ | 854 |
| Mercaptobenzothiazole (MBT) | $C_7H_5NS_2$ | 241 |
| Phos-Chek 259R (1.6 lb/gal), Monsanto: | | |
| Ammonium + ammonia[2/] | $NH_3$ | 40,140 |
| Phosphate | $PO_4^{-3}$ | 112,000 |
| Silicofluoride | $SiF_6^{-2}$ | 2,366 |
| Thiosulfate | $S_2O_3^{-2}$ | 1,143 |
| Mercaptobenzothiazole (MBT) | $C_7H_5NS_2$ | 322 |
| Fire-Trol 100, Chemonics: | | |
| Ammonium + ammonia[2/] | $NH_3$ | 43,600 |
| Sulfate | $SO_4^{-2}$ | 122,900 |
| Dichromate | $Cr_2O_7^{-2}$ | 2,500 |
| Fire-Trol 931L, Chemonics: | | |
| Ammonium + ammonia[2/] | $NH_3$ | 30,300 |
| Phosphate | $PO_4^{-3}$ | 113,300 |
| Ferrocyanide | $Fe(CN)_6^{-4}$ | 1,800-3,800 |
| Fire-Trol 934L, Chemonics: | | |
| Amnonium + ammonia[2/] | $NH_3$ | 31,370 |
| Phosphate | $PO_4^{-3}$ | 117,000 |
| Ferrocyanide | $Fe(CN)_6^{-4}$ | 2,720 |

1/ From table 2, Draft Fire Retardant Environmental Assessment, USDA Forest Service, Pacific Northwest Region, Portland, Oregon, undated, 157 p.

2/ The distribution of N between the ammonium and the ammonia forms is both temperature and pH dependent. See unpublished report, The Behavior and Impact of Chemical Fire Retardants in Forest Streams, by Norris, Hawkes. Webb, Moore, Bollen. and Holcombe, U.S. Department of Agriculture, Forest Service, Pacific Northwest Forest and Range Experiment Station, Forestry Sciences Laboratory, Corvallis, Oregon.

Studies of ferrocyanide conversion to cyanide were carried out by Burdick and Lipschuetz (1948) in open vessels exposed to sunlight during May and October. Initial potassium ferrocyanide concentrations ranged from 1 to 100 mg/liter, and exposure time was 1 to 5 hours. The inconsistent results were attributed to varying light intensity and temperature. The highest percent-conversions were at the low concentrations (1 to 5 mg/liter of potassium ferrocyanide at time zero). The term percent-conversion means transformation of mg/liter potassium ferrocyanide to mg/liter cyanide. Some of the runs with the lower concentrations showed a 25-percent conversion of potassium ferricyanide to cyanide.

In a more closely controlled experiment comparing 1, 2, and 3 mg/liter of potassium (K) ferrocyanide, the percent-conversions ranged from 10 to 15 percent in 1 hour, followed by a decrease in cyanide values. The decrease is attributed to loss of HCN and recombination of reaction products. In any event, although the percent-conversions of K ferrocyanide to cyanide vary (mg/liter $K_4Fe(CN)_6$ to mg/liter $CN^-$), the maximum value is about 25 percent.

The amount of sodium ferrocyanide that could reach surface water can be calculated, given the following assumptions:

- Fire-retardant mixtures contain up to 5400 mg/liter $Na_4Fe(CN)_6$ (equivalent to 3800 mg/liter $Fe(CN)_6^{-4}$).
- Air drop covers an area 75 m by 20 m with the rate of deposition of 2 liters/$m^2$.
- A stream 3 m wide and 0.2 m deep runs through the middle and along the long axis of the drop zone.
- Retardant mixing with the stream is instantaneous.

Based on these assumptions, the concentration of $Fe(CN)_6^{-4}$ would be 38 mg/liter [or 65 mg/liter as $K_4Fe(CN)_6$]. In a small pond (1233 $m^3$ = 1 acre foot), the concentration would be 9 mg/liter $Fe(CN)_6^{-4}$ or 15.6 mg/liter $K_4Fe(CN)_6$.

At a 25-percent conversion factor for the photolysis of the ferrocyanide to cyanide with 90 percent as HCN, the corresponding values are more than 8 mg/liter HCN in the stream and 2 mg/liter in the pond.

HCN disappears from water with time, as indicated by the reports of Rurdick and Lipschuetz (1948) and Doudoroff (1956). Although their studies were in the laboratory, we expect the same phenomenon in natural streams, especially where continual mixing allows HCN to be released at the interface of air with water.

The cyanide ion readily forms complexes with many metals, particularly the "d" block of the periodic table. This would probably be more of a factor in lowland streams than in upland forest waters, however, because of the incidence of these heavier metals. Reports of cyanide degradation in water are lacking; it has been shown, however, that degradation occurs in activated sludges and in nonsterile soil. In nonsterile soil, the carbon of $CN^-$ is oxidized to carbonate, and the N goes to $NH_3$.

In summary, if sodium ferrocyanide is deposited in streams through the use of fire retardants, some cyanide will be produced through photolysis. The concentration will obviously depend on the amount of ferrocyanide deposited in the stream, the light intensity after deposition, and the volume of the stream. The $CN^-$ will not pose a long-term hazard because of volatility losses, dilution, and perhaps complexing with metals.

Norris et al. (see footnote 2) conducted an extensive study of the entry, behavior, and impact of an ammonium-based fire retardant in forest streams in Oregon, Idaho, and California; the results of this study are summarized below.

The tests (where retardant was applied across streams at four western locations) showed that direct application of retardant to the surface of the stream produced detectable changes in stream-water chemistry for distances up to 1000 m downstream. The changes were of short duration and not important, either toxicologically or with respect to eutrophication downstream. The rate of application was low, however, and only a single application was made on each stream. The effects of rate of application, vegetation density in the streamside zone, and other factors on retardant levels in streams were examined in simulation studies.

The stream-chemistry studies showed that direct application to the stream surface was the primary source of retardant components in streams. Once these initial residues left the stream reach, only minor amounts of retardant entered from the streamside zone. Relatively narrow, untreated strips in the streamside zone were sufficient to virtually eliminate movement of retardant from the land to the stream. In the Norris et al. (see

footnote 2) study where the retardant was applied parallel to the stream, the edge of the treated area was only 3 m from the stream at several points.

The principal chemical species found in the stream within the first 24 hours after application were ammonium nitrogen and total phosphorus, Ammonium is of primary importance because of its potential effects on aquatic species. Phosphorus may be important in downstream eutrophication. After 24 hours, nitrate and soluble organic nitrogen were the primary retardant components in the stream. These are transformation products of the diammonium phosphate in the retardant mixture. Both chemicals are low in toxicity and are natural components of aquatic ecosystems.

Leaching studies showed that use of fire retardant on sites adjacent to streams can result in the entry of nitrogen into the stream in measurable quantities and in a form toxic to fish. The probability of occurrence of toxic levels is low, however, and can be further minimized by avoiding use of ammonium-based fire retardants on shallow, rocky, poorly developed soils occurring on steep slopes that drain directly into stream channels.

The simulation studies used computer-aid mathematical techniques with a combination of real and generated data to: develop methods for predicting the amount (concentration) of retardant in streams at the time they receive direct application to the stream surface; develop methods for describing the dispersal of retardant in streams, both with time after application and distance downstream from the zone of application; and integrate these two techniques with data on retardant toxicity to fish to evaluate the effects of various types of retardant application in various types of streams on fish mortality.

These simulations suggest:

- Direct application of retardant to streams is likely to cause fish mortality.
- The magnitude of the mortality and the distance over which it occurs vary with characteristics of the application, the site, and streamflow.

  Characteristics of the Application. The characteristics of application (assuming a constant pattern of distribution) include orientation of the line of flight to the stream, size of load dropped, number of loads dropped, and the timing and placement of subsequent loads relative to the first load. For instance, applying retardant perpendicular to a stream produces a much smaller zone of mortality than when its long axis is centered on the stream. If the rate of application is doubled over the same area, the zone of mortality increases by a factor of 10 or more. We did not simulate the effects of multiple loads or the timing and placement of subsequent loads on the mortality zone, but believe the effects of additional loads will be at least additive to the effects of the first load. Where the rate of application increases, substantial increases in the length of the mortality zone occur. The characteristics of the application can be controlled by the fire-control officer and the applicator to minimize impacts on the stream.

Characteristics of the Site. For a given retardant application, several characteristics of the zone of application are important in determining the length of the fish-mortality zone. These include the width and depth of the stream and the density of overstream vegetation (leaf-area index). Results of the simulation suggest that narrow, deep streams have a much shorter mortality zone than shallow, wide streams (assumes equivalent flow properties). Furthermore, the more dense the vegetation canopy over the stream, the less chemical will fall into the stream and the shorter will be the mortality zone. The characteristics of the site can be recognized and allowed for by the manager and the applicator, thus minimizing chemical entry into the stream.

Characteristics of Streamflow. Streamflow characteristics influence the length of the mortality zone by determining the degree and speed of mixing and dilution of retardant with downstream travel. Simulation results for streams of roughly equal gradient (steepness) show that a stream with a smooth, straight channel is likely to have a longer mortality zone than one with many pools and riffles. Pools and riffles cause the peak of retardant concentration to spread out, thus reducing the magnitude of exposure. The other streamflow characteristic of importance is the increase in stream discharge with distance downstream (because of the inflow of ground water and contribution from side streams). Increased stream discharge results in dilution of the retardant. Characteris'tics of streamflow can be recognized by the manager and taken into consideration when planning fire-control strategies to minimize stream impacts.

## Toxicity

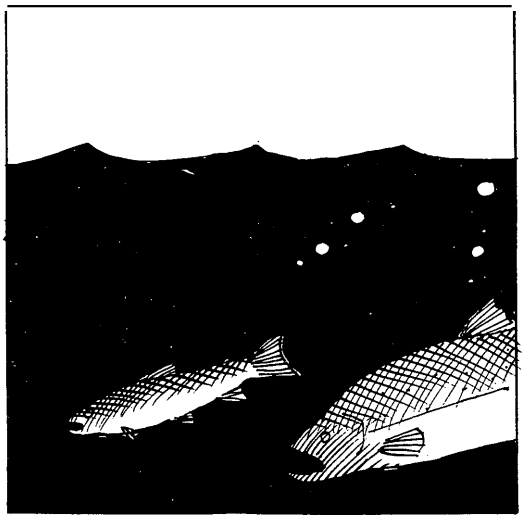
Douglas (1974) stated that retardants appear to have their greatest ecological impact on aquatic ecosystems. Numerous fish kills have been reported but few have been documented (see footnote 2). The few studies on the effects of fire retardants on fish populations show varying results, mainly because of the multitude of conditions that may be encountered. Blahm (1979) demonstrated that commercial fire retardants were toxic to juvenile coho salmon and rainbow trout and attributed the mortality to ammonia in the retardants. He noted that increasing the pH of diluent waters from 7 to 8 increased the toxicity. McKee and Wolf (1971) noted that ammonia concentrations as low as 0.3 mg/liter were lethal to trout fry and 75 mg/liter was extremely lethal to mature trout. Un-ionized ammonia ($NH_3$) has been reported to be the component of retardants likely to be toxic to fish and other organisms. The concentration of free $NH_3$ in any of the retardant-water mixtures depends on the amount of $NH_4^+$ contained in the retardant and the pH of the retardant-water solution. Blahm et al.[23/] found that two species of juvenile salmonids exposed to four commercial fire retardants had 96-hour $TL_m$'s of 120 to 940 mg/liter.

---

23/ Unpublished report, "Effect of chemical fire retardants on the survival of juvenile salmonids," by T. H. Blahm, W. C. Marshall, and G. R. Snyder, Bur. Land Manage. Contract 53500-CT2-85(N), Natl. Mar. Fish. Serv., Environ. Field Stn., Prescott, Oreg., 1972.

Johnson and Finley (1980), investigating the toxicity of a Phos–Chek fire–retardant formulation, found that the 96–hour $LC_{50}$ for coho salmon and rainbow trout ranged from 160 to 320 mg/liter, but noted that yolk–sac fry of both species were more sensitive than fingerlings. Warm–water species were less sensitive than salmonids to two Phos–Chek formulations. The 96–hour $LC_{50}$ values for Phos–Chek 202 were 650 and 840 mg/liter for salmonids and fathead minnows, and for Phos–Chek 259 they were 300 and 350 mg/liter for salmonids and bluegills. A gammarid amphipod was the most sensitive, having a 96–hour $LC_{50}$ of 40 to 52 mg/liter, depending on the formulation tested.

The toxicological effects of sodium ferrocyanide, a corrosion inhibitor used in some retardant mixtures, may not have been adequately assessed (sodium ferrocyanide is presently used in Fire–Trol 931–L and 934-L). Doudoroff (1976) stresses that the suitability of cyanide–polluted waters for aquatic life has to be expressed as a concentration of free cyanide or molecular HCN, not of total cyanide. Free cyanide concentrations from 0.05 to 0.01 mg/liter as CN have proved fatal to many sensitive fishes (Jones 1964), and levels above 0.2 mg/liter are rapidly fatal for most species of fish. A level as low as 0.01 mg/liter is known to have a pronounced, rapid, and lasting effect on the swimming ability of salmonid fishes (National Academy of Sciences–National Academy of Engineering 1973). Blahm (1979) performed comparative evaluations of toxicity for different retardants and concluded that Phos–Chek formulations were more toxic to salmonid fishes than were Fire–Trol compounds. The higher toxicity was believed to be a function of pH and ammonia toxicity; Phos–Chek formulations are more basic than Fire–Trol compounds. Blahm's relative toxicity values for the two compounds is valid only if Fire–Trol 931 is mixed at a 4:1 ratio for field application. When applied as 3:1 or 2:1 mixtures, Fire–Trol 931 may have a higher ammonium toxicity than Phos–Chek compounds. Additional tests are warranted, because Fire–Trol 931 and 934 could apparently be more toxic to aquatic life than other approved retardants, especially on sunny days.



# HAZARD ASSESSMENT

## HAZARDASSESSMENTATTHE ORGANISM LEVEL

The toxicological risk (or hazard) of forest chemicals to anadromous fish may be manifested through direct action on the fish themselves or indirectly through action on fish–food organisms. One means of expressing toxic risks is the margin of safety, the ratio of the no–effect level to the actual exposure level. When the exposure is equal to the no–effect level, the margin of safety is one. Margins of safety less than one indicate the exposure level is greater than the no–effect level and suggest that a direct toxic effect is likely. The larger the margin of safety, the less likely toxic effects will occur.

What constitutes an adequate margin of safety is a matter of judgment for many pharmaceuticals, caffeine, alcohol, and other materials many humans encounter daily. The margins of safety are as low as 1.5 to 15, and margins of safety of less than 100 are common. Margins of safety of about 100 are commonly used in setting pesticide tolerances in food and feed. Where the species are extremely valuable or rare, a much larger margin of safety may be appropriate. These margins of safety usually assume long-term chronic exposure will occur. Some margin of safety is necessary because the toxicity testing done thus far may not have identified the "lowest" no-effect level, toxicity-testing conditions usually differ from field conditions, and individuals in the population differ in susceptibility.

Forest chemicals have been investigated mostly for lethal effects; sublethal effects, however, may occur at lower exposures than those that are lethal. Potential sublethal effects of forest chemicals on salmonids include effects on growth, behavior, reproduction, resistance to stress, migration, biochemistry, and physiology. Picloram, 2,4-D, and DDT have been observed to cause reductions in fish growth in the field and laboratory (Warner and Fenderson 1962, Cope et al. 1970, Woodward 1976). Several types of behavior (for example, learning, swimming, temperature preference, predator avoidance) have been shown to be altered by exposure to pesticides (Ogilvie and Anderson 1965; Warner et al. 1966; Anderson 1968, 1971; Anderson and Peterson 1969; Hatfield and Anderson 1972; Hatfield and Johansen 1972; Symons 1973, 1977). Both DDT and 2,4-D have been demonstrated to cause decreased reproductive success in fish (Macek 1968, Wilbur and Whitney 1973). Diquat and picloram caused migratory inhibition in coho salmon smolts in coastal streams of Oregon (Lorz et al. 1979). Many studies have demonstrated biochemical or physiological change in fish after exposure 'to pesticides (Weiss and Gakstatter 1964; Grant and Mehrle 1970; Wildish et al. 1971; Hiltibran 1972a, b). One of the best documented biochemical effects of a forest chemical is the inhibition of AChE activity by organophosphates (Williams and Sova 1966). Scientists are aware of many potential sublethal effects of forest chemicals; however, data on sublethal effects are scarce and a large portion of the available information pertains to organochlorines, particularly DDT. The no-effect levels for sublethal effects of forest chemicals are most likely to be much lower than for acute or chronic toxicities. Our lack of knowledge prevents hazard assessment of forest chemicals for sublethal effects and forces us to use margins of safety; increased research on sublethal effects may allow us to better evaluate the potential effects of forest chemicals on anadromous salmonids.

Organisms can exhibit numerous kinds of responses when exposed to toxic chemicals. Survival, growth, reproductive success, and behavior characteristics are probably the most important, but the bulk of the aquatic toxicology literature reports only survival for short-term acute exposure in the water. Although this deficiency in the data base is obvious, fortunately in forest aquatic systems short-term acute exposures predominate, if exposure occurs at all. Thus, we can use toxicity data on survival of fish (or other more sensitive organisms) to approximate a no-effect level for short-term exposure.

We have selected a concentration of 0.1(96-hour $LC_{50}$) as the no-effect level for survival after brief acute exposures to peak concentrations of forest chemicals. This value is a little more conservative than the 0.1 of the 48-hour $LC_{50}$ tentatively suggested by the Aquatic Life Advisory Committee (1955). Some have treated this application factor almost as an

immutable constant, but others have attacked it as an oversimplification. Tarzwell (1966) pointed out that 0.1 toxic units is a concentration that has been used successfully for the safe disposal of some wastes, when firm information was lacking. Sprague (1971) argued that no single value could be expected to fit all types of pollution. In his review of sublethal and "safe" concentrations, Sprague (1971) noted that a number of application factors were recommended but, in general, the accepted maximum levels are 0.1 or 0.05 toxic units for nonpersistent pollutants and 0.1 or 0.01 toxic units for persistent chemicals and pesticides. The National Academy of Sciences–National Academy of Engineering (1973) Water Quality Criteria also recommended the use of application factors not exceeding 0.1 of the 96-hour $LC_{50}$, where concentration of materials is nonpersistent or has noncumulative effects, to estimate "safe" concentrations of toxic wastes into receiving streams, unless specific application factors have been determined for a given material.

Relatively few data are available on the no-effect level for other types of responses, particularly for prolonged exposure to the chemicals we have discussed in this paper. The National Academy of Sciences–National Academy of Engineering (1973) Water Quality Criteria recommends that the concentration should not exceed 0.05(96-hour $LC_{50}$) at any time or place, nor should the 24-hour average concentration exceed 0.01 (96-hour $LC_{50}$) for persistent or cumulative toxicants.

Allison (1977) and Larsen et al. (1978) have investigated the relation of exposure duration, concentration, and periodicity of toxicants to toxicity, reproduction, and growth. These studies, using exposure units in conjunction with established toxicity data, may allow us to identify a "safe" level and thereby assess the environmental impact of variable-level, short-term pesticide exposure on the aquatic environment.

## Assessment of Acute-Toxicity Hazard

Numerous exposure values can be used in calculating margins of safety (table 16). We used the single highest instantaneous concentration we found in surveying the literature and a peak concentration of 0.02 mg/liter, which we believe is the maximum likely to occur if minimum buffer strips are used and some direct application to surface water occurs. We used these values with 0.1(96-hour $LC_{50}$) to calculate the margin of safety for survival to acute exposure (table 17). These calculations yield conservative estimates of the margin of safety because the instantaneous peak concentration in the field does not persist for the 96-hour period in the toxicity test and current "best management practices" in the use of forest chemicals will not produce exposure levels that approach the peak concentrations listed in tables 16 and 17.

Table 16--Integral of concentration-time curves for 48 hours for several pesticides and 192 hours for urea in forest streams after aerial application[1/]

| Pesticide | Actual peak concentration | Integral for actual peak concentration | Integral for assumed peak concentration of 0.02 mg/liter for pesticides and 7.02 mg/liter[2/] for urea |
|---|---|---|---|
| | mg/liter | (mg/liter)hours | (mg/liter)hours |
| 2,4-D | 0.014 | 0.116 | 0.167 |
| Amitrole | .110 | .498 | .091 |
| Dicamba | .037 | .310 | .167 |
| Malathion | .040 | .074 | .037 |
| Carbaryl | .121 | .343 | .057 |
| Acephate | .471 | 1.708 | .072 |
| Urea[3/] | 1.389 | 38.2 | 193 |
| Urea[4/] | .700 | 19.4 | 195 |

[1/] Based on figure 4.
[2/] Mean peak concentration of 28 fertilizer-monitoring projects summarized by Moore 1975b.
[3/] Based on figure 4G.
[4/] Based on figure 4H.

Table 17--Estimated margins of safety for survival of salmon, trout, or other more sensitive aquatic species to "short-term" exposure to selected forest chemicals

| Forest chemical | Organism | 96-h $LC_{50}$ | No-effect level, acute exposure[1] | Highest observed exposure and margin of safety[2] | Margin of safety for 0.02 mg/liter peak concentration[3] | No-effect level, chronic exposure (48 h)[4] | Short-term chronic exposure (48 h)[5] | Margin of safety |
|---|---|---|---|---|---|---|---|---|
| | | mg/liter | | | | (mg/liter)hours | | |
| **Herbicides:** | | | | | | | | |
| 2,4-D-- | | | | 0.84 mg/liter | | | [6]/0.334 | |
| Oimethylamine | Rainbow trout | 100 | 10 | 11.9 | 500 | 48 | | 144 |
| | Daphnia sp. | 4 | .4 | .5 | 20 | 1.92 | | 5.7 |
| | Glass shrimp | .15 | .015 | <.1 | .7 | .072 | | .2 |
| Butyl ester | Cutthroat trout | .9 | .09 | .1 | 4.5 | .432 | | 1.3 |
| | Pteronarcella sp | 1.5 | .15 | .2 | 7.5 | .72 | | 2.2 |
| PGBE ester | Cutthroat trout | 1.0 | .1 | .1 | 5 | .480 | | 1.4 |
| | Daphnia sp. | 1.2 (48-h) | .12 | .1 | 6 | .576 | | 1.7 |
| | Glass shrimp | .4 | .04 | <.1 | 2 | .192 | | .6 |
| Picloram-- | | | | 2.0 mg/liter | | | [7]/.083 | |
| Technical | Cutthroat trout | 3.5 | .35 | .2 | 17 | 1.68 | | 20 |
| | Pteronarcys sp. | .048 | .0048 | <.1 | .2 | .023 | | .3 |
| Potassium salt | Cutthroat trout | 1.5 | .15 | <.1 | 7.5 | .72 | | 8.7 |
| Tordon 101[8] | Rainbow trout | 8.6 | .86 | .4 | 43 | 4.13 | | 50 |
| Atrazine | | | | .42 mg/liter | | | [9]/.668 | |
| | Chironomus tenans | .72 (48-h) | .072 | .2 | 3.6 | 3.46 | | 5.2 |
| | [redacted] sp. | 6.9 (48-h) | .69 | 1.6 | 34 | 3.31 | | 5.0 |
| | [redacted] | 6.7 (48-h) | .67 | 1.6 | 34 | 3.22 | | 4.8 |
| | Brook trout | 4.9 (48-h) | .49 | 1.2 | 24 | 2.35 | | 3.5 |
| MSMA-- | | | | .01 mg/liter | | | [10]/-- | |
| Liquid formulation | Cutthroat trout | 100 | 10 | 1 000 | 500 | 48 | | [11]/-- |
| | Gamnarus fasciatus | 100 | 10 | 1 000 | 500 | 48 | | [11]/-- |
| | Bluegi11 | 12 | 1.2 | 120 | 60 | 5.76 | | [11]/-- |
| Plus surfactant | Bluegill | 49 | 4.9 | 490 | 245 | 23 | | [11]/-- |
| Fosamine ammonium | | | | [12]/-- | | | [9]/.668 | |
| | Coho salmon, fingerling | 5 361 | 536 | [11]/-- | >10 000 | 2 573 | | 3 852 |
| | Rainbow trout, yolk sac | 528 | 52.8 | [11]/-- | 2 640 | 253 | | 379 |
| Glyphosate-- | | | | 2.6 mg/liter | | | [9]/.668 | |
| Technical | Rainbow trout | 130 | 13.0 | 5 | 650 | 62.4 | | 93 |
| Liquid formulation | Rainbow trout, fingerling | 8.3 | .83 | .3 | 41 | 3.98 | | 6.0 |
| | Rainbow trout, swim-up fry | 2.4 | .24 | [redacted] | 12 | 1.15 | | 1.7 |
| | Daphnia sp. | 3 | .3 | .1 | 15 | 1.44 | | 2.2 |
| Oinoseb | | | | [12]/-- | | | [6]/.334 | |
| | Cutthroat trout | .041 | .004 | [11]/-- | .2 | .019 | | <.1 |
| **Insecticides:** | | | | | | | | |
| Malathion | | | | .042 mg/liter | | | [13]/.037 | |
| | Daphnia sp. | .001 (48-h) | .000 1 | <.1 | [redacted] | .000 5 | | <.1 |
| | Pteronarcys sp. | .01 | .001 | [redacted] | [redacted] | .004 a | | .1 |
| | Coho salmon | .17 | .017 | .4 | .8 | .082 | | 2.7 |
| Carbary1 | | | | .121 mg/liter | | | [13]/.057 | |
| | Daphnia sp. | .006 (48-h) | .000 6 | [redacted] | <.1 | .002 3 | | [redacted] |
| | Pteronarcys sp. | .001 7 | .000 17 | [redacted] | [redacted] | .000 8 | | [redacted] |
| | Coho salmon | 4.34 | .434 | 3.6 | 22 | 2.08 | | 36 |
| Azinphos-methyl | | | | [12]/-- | | | [10]/-- | |
| | G. fasciatus | .15 | .015 | [11]/-- | .7 | .072 | | [11]/-- |
| | Pteronarcys sp. | .001 9 | .000 19 | [11]/-- | [redacted] | .000 9 | | [11]/-- |
| | Coho salmon | .006 | .000 6 | [11]/-- | [redacted] | .003 | | [11]/-- |
| Carbofuran | | | | [12]/-- | | | [10]/-- | |
| | Coho salmon | .530 | .053 | [11]/-- | 2.6 | .25 | | [11]/-- |
| | Rainbow trout | .380 | .038 | [11]/-- | 1.9 | .18 | | [11]/-- |
| Acephate | | | | .961 mg/liter | | | [13]/.072 | |
| | Pteronarcella sp | 9.5 | .95 | 1.0 | 47 | 4.56 | | 63.3 |
| | Cutthroat trout | 100 | 10 | 10 | 500 | 48 | | 667 |
| | Rainbow trout | 1 100 | 110 | 114 | 5 500 | 528 | | 7 333 |
| **Fertilizer:** | | | | | | | | |
| Urea | | | | 44.4 mg/liter | | | [13]/193.0 | |
| Amnonia | | | | [14]/.014 mg/liter | | | [15]/.83 | |
| | Coho salmon | .2 | .02 | 1.4 | 1 | .096 | | .1 |
| **Fire retardant:** | | | | | | | | |
| Amnonia | | | | [16]/1.30 mg/liter | | | [17]/-- | |
| | Coho salmon | .2 | .02 | <.1 | 1 | .096 | | [11]/-- |

[1]/ 0.1(96-hour $LC_{50}$) is assumed to be the no-effect level for survival for short-term acute exposure.
[2]/ Single highest instantaneous concentration reported in text or in cited reference (some values are adjusted for registered rates of application in forestry) and corresponding margin of safety (no-effect level/exposure level).
[3]/ Margin of safety (no-effect level/exposure level) for assumed peak concentration of 0.02 mg/liter for pesticides, 7.02 mg/liter for fertilizer (urea), and 130 mg/liter for fire retardant.
[4]/ Integral of 0.01(96-hour $LC_{50}$) concentration-time curve for 48 hours.
[5]/ Integral of concentration-time curves in figure 4, assuming a peak concentration of 0.02 mg/liter (see also table 16). A peak concentration of 0.02 mg/liter is assumed to be the maximum instantaneous concentration likely to result, based on operational monitoring. It assumes applications are made with minimum buffer strips and some direct application to the stream surface.
[6]/ From table 16, adjusted to 2.24 kg/ha application rate (based on data for 2.4-D, figure 4A), and peak concentration of 0.02 mg/liter.
[7]/ From table 16, adjusted to 0.56 kg/ha application rate (based on data for 2,4-D, figure 4A), and peak concentration of 0.02 mg/liter.
[8]/ Tordon 101 is a 4:1 mixture of 2,4-D and picloram. The risk-assessment calculations are done using exposure data for picloram only. See 2,4-D for relevant data on 2,4-D.
[9]/ From table 16, adjusted to 4.48 kg/ha application rate (based on data for 2,4-D, figure 4A), and peak concentration of 0.02 mg/liter.
[10]/ Data not available. Normal use is not expected to result in stream contamination.
[11]/ Margin of safety not calculated because no value for exposure is available.
[12]/ Data not available.
[13]/ From table 16, assumed peak concentration 0.02 mg/liter, except for urea which is based on assumed concentration of 7.02 mg/liter.
[14]/ From table 13, assumes 1-percent un-ionized ammonia (25 °C, pH 7.5).
[15]/ Based on proportion of ($NH_3$ + $NH_4^+$) to urea, table 12, and 1-percent un-ionized ammonia (25 °C, pH 7.5).
[16]/ Assumes 1-percent ammonia in 130 mg/liter retardant in stream water, from Norris et al. (1978).
[17]/ No estimate because of high variability in patterns of retardant use. Applications directly into streams will produce levels of ammonia greater than 1 mg/liter.

## Assessment of Chronic–Toxicity Hazard

We calculated the margin of safety for survival from chronic exposure using the integral of the concentration–time curve for chemicals in forest streams with the integral of the concentration–time curve for exposure that is equal to 0.01(96-hour $LC_{50}$). This concept is based on the use of exposure units--that is, the integral of duration and level of exposure, as developed by Allison (1977) and Larsen et al. (1978). The derivation of each of these values follows.

Numerous data sets report the concentration of herbicides in forest streams. In an effort to find one that would be representative, we normalized the Concentration data for three herbicides and shifted the time scale slightly to show the peak concentration (100 percent) occurring 3 hours after application (fig. 4a, b, and c). The data for both amitrole–T in Wildcat Creek and 2,4-D in Preacher Creek show an increase in concentration as a result of rain (approximately 0.7 cm at Wildcat Creek and 0.6 cm at Preacher Creek on the 1st day after application). The areas under the curves were measured to give a time–concentration expression [(mg/liter)hours] of contamination for the first 48 hours after application, using the actual peak concentration observed, and assuming an instantaneous peak concentration of 0.02 mg/liter (table 16). The latter value is our estimate of the maximum contamination level likely to result if minimum buffer strips are used and some direct application to surface water occurs (see related discussion in the chemicals in water section and U.S. Department of Agriculture, Animal and Plant Health Inspection Service 1980).

The results show reasonably good agreement among chemicals with 0.167 (mg/liter)hours for 2,4-D, 0.168 (mg/liter)hours for amitrole, and 0.91 (mg/liter)hours for dicamba. The higher value for dicamba reflects its longer than normal persistence. Based on this analysis, we decided to use the 48–hour time–concentration expression of exposure of 0.167 (mg/liter)hours derived from the 2,4-D data from Preacher Creek (fig. 4a) (adjusted *for* rate of application) for all the aerially applied herbicides in table 17. MSMA was excluded because it is not usually applied aerially, and the limited monitoring *for* MSMA has not shown measurable residues in forest streams. Use *of* the 2,4-D data for the other herbicides is reasonable because we believe the predominant processes of entry are drift, direct application to the stream surface, and mobilization in ephemeral stream channels shortly after application. These processes are largely mechanical and should not vary greatly among the aerially applied herbicides discussed in this paper.

Data for the concentrations of malathion, carbaryl, acephate, and urea (from fertilizer) in streams at various times after application were plotted, and the area under the curves was integrated in the same way as for the herbicides (fig. 4d-h, table 16). The normal patterns of use of azinphos–methyl and carbofuran (used for control of seed and cone insects) will not result in contamination of forest streams.

The no–effect level for survival from chronic exposure to each chemical is expressed as the integral (over 48 hours) of the time–concentration curve equivalent to 0.01(96-hour $LC_{50}$) for that chemical. These values are expressed as (mg/liter)hours for 48 hours, just as the exposure data from field studies are expressed. For example, the 96–hour $LC_{50}$ for carbaryl and coho salmon is 4.34 mg/liter and 0.01(96-hour $LC_{50}$) is 0.0434 mg/liter. Because the

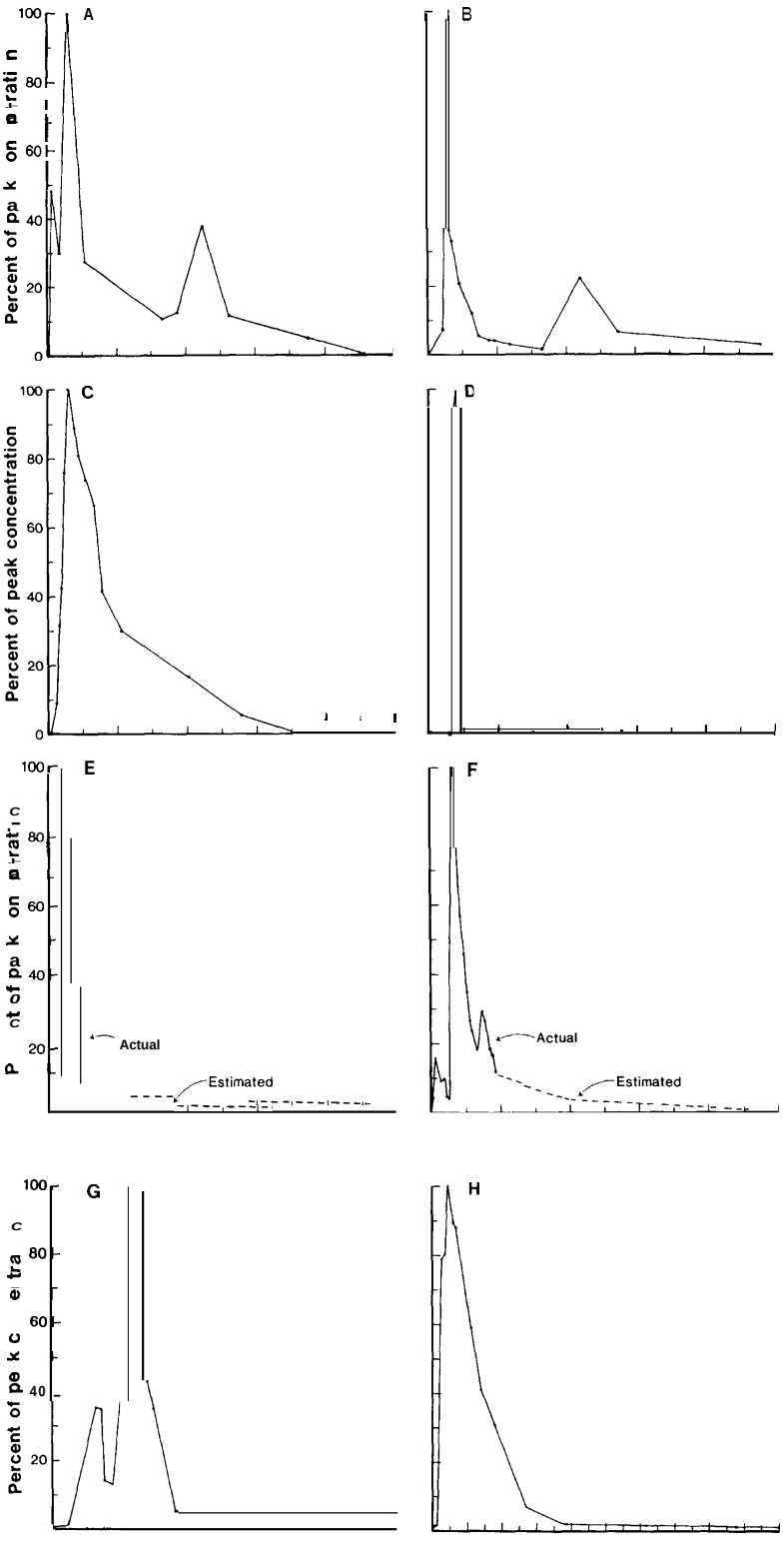



Figure 4.--Concentration of chemicals in forest streams at different times after aerial application. Concentration is expressed as a percentage of the peak concentration.

A. 2,4-D in Preacher Creek, with a partial stream buffer. Actual peak concentration vas 0.0139 mg/liter after aerial application of 2,4-D table 10, Fredriksen et al. 1975).

C. Dicamba in Farmer Creek, with no stream buffer. Actual peak concentration vas 0.037 mg/liter after aerial application of dicamba at 1.12 kg/ha (from fig. 2, Norris and Montgomery 1975).

D. Malathion in Hansel Creek, vith no stream buffer. Actual peak concentration vas 0.040 mg/liter after aerial application (from fig. 2, Tracy et al. 1977).

E. Carbaryl in Squilchuck Creek, with concentration vas 0.121 mg/liter after aerial application at 1.12 kg/ha (from fig. 7, Bernhardt et al. 1978). Note projection of estimated concentration curve beyond 11 hours.

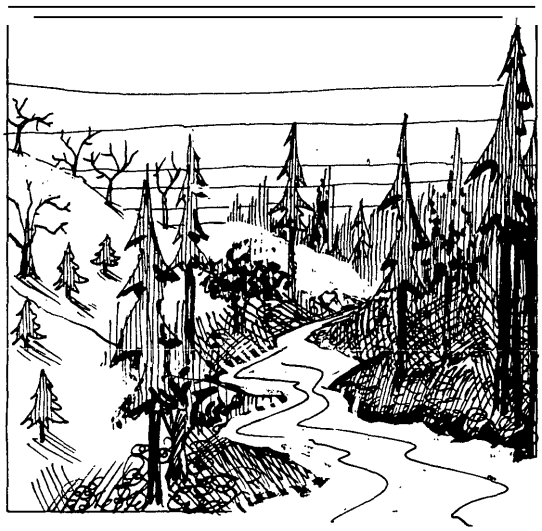
F. Acephate in Cabin Creek, with no stream buffer. Actual peak concentration vas 0.471 mg/liter after aerial application at 1.12 kg/ha (from table 12, Flavell et al. 1977). Note projection of estimated Concentration curve beyond 9.25 hours.

after aerial application at 224 kg N/ha (as urea) (from table 1, Moore 1975b; and personal communication from D. G. Moore, For. Sci. Lab., Corvallis, Oreg., 1980). Note time scale is not the same as in figures 4A-F.

E. Urea in Trapper Creek, with 60-m stream buffer. Actual peak

exposure level is constant over the 48-hour period we are interested in, the integral of the time-concentration curve is 0.0434 mg/liter x 48 hours = 2.08 (mg/liter)hours. The ratio of the no-effect exposure integral to the field exposure integral is the margin of safety (table 17).

We believe the margins of safety calculated for chronic exposure are conservative because the toxicity data are based on continuous exposure at the specified level, although we know from field data that the exposure is quite transitory. For instance, if we were to extend the period of evaluation from 48 hours to 30 days, the no-effect exposure integral would increase 15 times, but the field-exposure integral would not change because no further exposure occurs. Thus, the margin of safety would increase 15 times.



## HAZARD ASSESSMENT AT THE ECOSYSTEM LEVEL

Assessing the hazard to organisms rests on a reasonably adequate data base. But a disadvantage is that it focuses on individual organisms and does not take into account either time or space or the basic resiliency of ecosystems. For instance, our assessment for carbaryl indicates coho salmon will not be directly affected, but some individual invertebrates may be killed in a segment of a stream shortly after aerial application. It fails to recognize that some other individuals will survive (by avoidance or greater individual tolerance to the chemical) and that repopulation of the affected portion of the stream will occur (by migration from unaffected areas or hatching). In addition, it fails to recognize that the affected area is likely to be small because of efforts to avoid direct application to streams and because most treatments do not cover large, contiguous areas (some large insect-control projects may be an exception). The same area is not likely to be affected repeatedly because, over the course of any one timber rotation, more than three applications to the same area are rare and the time between repeat applications will usually be more than 1 year. As a consequence, we believe that the risk assessments in table 17 are conservative; the true margins of safety for anadromous fish from exposure to forest chemicals on the large watershed or ecosystem scale are larger than we have calculated.

# INDIRECT EFFECTS OF FOREST **CHEMICALS**

Effects of forest chemicals on aquatic organisms have been investigated for several decades, but these studies have generally been restricted to direct toxic effects. The use of herbicides, insecticides, fertilizers, and fire retardants may change the composition and production of terrestrial plants and invertebrates. Alteration of terrestrial vegetation and invertebrate communities may result in changes in allochthonous inputs into streams and in environmental factors such as light, temperature, water quality, sediment composition, and geomorphology. All of these factors are components of anadromous fish habitat as discussed by Reiser and Bjornn (1975). Land managers must be aware of potential indirect effects of forest chemicals on patterns and processes of stream ecosystems in the Pacific Northwest and Alaska.

## HERBICIDES

The following discussion of indirect effects of forest herbicides focuses on alteration of riparian vegetation adjacent to streams, rivers, and lakes. General aquatic processes that may be affected by terrestrial use of herbicides are described; documented studies of indirect effects of forest chemicals on aquatic systems are rare, however. Indirect effects of herbicides on aquatic communities have been observed as a result of mortality of particular components--for example, aquatic plants--and subsequent shifts in other components of stream ecosystems (Haven 1963, Smith and Isom 1967). They usually involve direct application of herbicide for aquatic weed control. In reviews of secondary effects of pesticides in aquatic systems, Hurlbert (1975) and Newbold (1975) viewed only the results of mortality to aquatic plants as the indirect effects of herbicides. Concentrations of herbicide in surface waters after forest application are much lower (less than 0.1 mg/liter) than those needed to control aquatic weeds (more than 2 mg/liter) (Norris and Moore 1971, 1976; National Research Council of Canada 1978; Norris 1978). Therefore, the effects that result from death of portions of biotic communities in streams are not pertinent to the subject of forest herbicides, except in unusual circumstances.

Herbicides may alter natural patterns of plant succession along streams. Herbicide application is intended to control nonconiferous trees and shrubs so that growth and development of commercial conifer species will be accelerated during the first few decades after timber harvest. In the following discussion, note that forest herbicides are applied to watersheds that have recently been logged. Therefore, we are comparing natural patterns of succession with patterns that are selectively constrained by human actions.

Plant succession after logging or burning generally goes through three stages: the weed stage, generally lasting less than 5 years; the shrub stage, roughly lasting from the 5th year through the 15th year; and the tree-dominated stage, which begins after about 10 to 15 years (Dyrness 1973; Franklin and Dyrness 1973). Tree communities are often dominated by deciduous trees, such as alder (Alnus spp.), bigleaf maple (Acer macrophyllum Pursh), or vine maple (A. circinatum Pursh) in the early stages of succession. Large shrubs, such as rhododendron (Rhododendron macrophyllum D. Don), Ceanothus spp., and salmonberry (Rubus spectabilis Pursh) are also major components of plant communities during this time. Between 20 and 60 years after cutting, coniferous species begin to dominate the tree communities.

In timber management, herbicides are often applied during the first decade after logging to control nonconiferous trees and shrubs. In essence, natural patterns of succession are altered because development and duration of early successional stages of trees and shrubs are reduced. Dominance of terrestrial vegetation is changed from herbs, shrubs, or hardwoods to conifers. This change in plant communities has many implications for stream communities in logged watersheds because deciduous vegetation differs greatly from coniferous vegetation in form, growth habits, timing of litterfall, and quality of organic matter produced. In addition, control of pioneer plant communities in the Pacific Northwest may have other long-term ecological implications because several pioneer species such as red alder (Alnus rubra Bong.) and Ceanothus are nitrogen fixers, and this region is generally nitrogen limited. Reduction of pioneer communities of nitrogen fixers may, therefore, alter nitrogen dynamics in watersheds treated with herbicides; few data from herbicide-treated areas are available, however (Tarrant and Trappe 1971).

Control of terrestrial vegetation may alter physical characteristics of the stream environment, such as streamflow, temperature, and light intensity. Immediately after reduction of nonconiferous plant biomass by herbicides, streamflow may increase because of reduced evapotranspiration (Hibbert 1967). Cutting of forests has been demonstrated to cause increases in both base flow and peak discharge (Hewlett and Hibbert 1961, Hornbeck et al. 1970, Harr 1977). Similar responses have been observed in watersheds treated with herbicides on rangelands (Ingebo 1971) and in northeastern forests (Mrazik et al. 1980). Herbicides are generally applied to watersheds that have recently been logged; therefore, herbicide use is not the primary cause of increased streamflow, but rather a factor that may extend this period of increased streamflow after deforestation. Increased base flow may be beneficial to many stream organisms, but increased peak flows may be detrimental. Reduction of streamside vegetation increases solar radiation reaching the stream channel and eliminates cover. Increases in solar radiation can increase summer stream temperature, depending on flow, gradient, and geomorphology (Brown 1969).

Alteration of the vegetative structure of watersheds may result in decreased streambank stability and increased erosion from hillslopes. The degree to which the vegetation and the rooting systems are altered determines the extent of sedimentation and channel modification, but the sedimentation rates of deforested watersheds are frequently more than double those of forested watersheds (Swanston and Swanson 1976). As previously mentioned, herbicides are applied to logged watersheds, which are much more erodible than forested watersheds. Therefore, herbicides must not be viewed as the primary cause of sedimentation but as a factor that retards the vegetative recovery of the watershed and may extend the period of increased sedimentation. Unfortunately, no studies have been published on the effects of herbicides on erosional patterns in logged watersheds. The detrimental effects of sedimentation and channel degradation on the structure and function of stream ecosystems have been documented extensively.[24/]

---

[24/] Unpublished report, "Sediment and water'quality: a review of the literature including a suggested approach for water quality criteria," by R. N. Iwamoto, E. O. Salo, M. A. Madej, and R. L. McComas, U.S. Environ. Prot. Agency, Reg. X, Seattle, Wash., 1978.

In sufficient concentrations, herbicides may directly affect aquatic primary producers (plants); indirect effects of herbicide applications may also alter primary producers in streams. As indicated earlier, increases in solar radiation may result from alteration of streamside vegetation and these increases may, in turn, cause greater rates of aquatic primary production (McIntire and Phinney 1965, Hansmann 1969, Gregory 1980). Such responses by aquatic primary producers have been observed after control of riparian vegetation by herbicides in rangelands of the Southwest (Smith et al. 1975). Temperature increases may also cause elevated rates of gross primary production in streams (McIntire 1966). Sedimentation from terrestrial systems that are influenced by herbicide use may cover benthic algal communities or scour algal cells from substrate surfaces, causing a reduction in standing crops of primary producers (Cordone and Pennoyer 1960, Chapman 1963, Nuttal 1972).

Primary production in streams may be stimulated by increased nutrient concentrations. Herbicide application has been observed to result in increased nitrogen inputs to streams (Sollins et al. 1981). A large amount of nitrogen was put into a stream after herbicide treatment of a watershed in New Hampshire; however, the rate of herbicide applied in this study was much greater than commonly used in forestry (Likens et al. 1970). Primary production in most Pacific Northwest streams has been demonstrated to be nitrogen limited (Thut and Haydu 1971, Gregory 1980); therefore, increases in nitrogen content of stream water as a result of herbicide application may stimulate rates of primary production.

Aquatic invertebrates may be affected by physical changes that result from herbicide application. Any increased sedimentation that may result from use of herbicides could have detrimental effects on communities of aquatic invertebrates through scour and alteration of habitat (Cumins and Lauff 1969, Burns 1972, Brusven and Prather 1974, Cederholm and Lestelle 1974). Temperature increases may result in increased growth and production of aquatic insects if the increases are slight; if stream temperatures exceed the optimum temperature for a particular species, however, the effects are negative.

Alteration of terrestrial vegetation by herbicides may influence communities of aquatic invertebrates. The initial increase in deciduous leaf fall into streams after herbicide application temporarily increases the food supply of aquatic invertebrates. In addition, the nitrogen content of this leaf material is greater than that of leaves that go through normal abscission (Jenson 1929, Sollins et al. 1981). Aquatic invertebrates attain faster growth and higher production on leaf material with high nitrogen content (Russell–Hunter 1970, Sedell et al. 1975). The duration of enhancement is short, however, because the conversion of the terrestrial stand to conifers results in a reduction of the more palatable deciduous material and an increase in less palatable coniferous material. Therefore, the quality of the food supply of detrital–feeding inverte–brates decreases as the riparian zone returns to dominance by conifers.

The potential stimulation of aquatic primary production as a result of herbicide application could result in increased production of grazing insects. Grazers in streams are often food limited (McIntire and Colby 1978); therefore, increases in their food supply enhances their production. This enhancement of grazing inverte–brates is gradually diminished as the developing coniferous stands shade the streams.

Aquatic predators, both invertebrate and vertebrate, could benefit from the enhancement of lower trophic levels. If production of grazing, collecting, and shredding invertebrates is increased as previously described, production of aquatic predators would also increase. Production of predators in streams in logged watersheds has sometimes been found to be greater than production in forested sections (Aho 1976, Erman et al. 1977, Hall et al. 1978, Murphy 1979). If herbicide treatment prolongs the stage of opened canopy after logging, this period of increased production could be extended. Release of the conifers may shorten the deciduous successional phase, however, and this phase may well be more productive for stream biota. Enhanced production of aquatic biota must therefore be viewed in the context of the normal patterns of ecosystem development.

Fish populations, especially salmonids, could also be detrimentally affected by herbicides. Salmonids prefer cold, clear streams; therefore, increased temperature and sedimentation from herbicide use may adversely affect salmonid populations. Sedimentation may reduce egg and fry survival (Neave 1947, Phillips 1964, Koski 1966)[25/] and the quality of rearing habitat (Everest and Chapman 1972, Bjornn et al. 1974). Saimonids also require cover; streamside vegetation provides a major portion of this type of habitat (Lewis 1969, Hunt 1978). Reduction of streamside vegetation by forest herbicides would, therefore, adversely affect salmonid populations.

---

25/ Unpublished annual completion report, "Embryo survival and emergence studies," by T. C. Bjornn, Proj. F-49-R-6, Job 6, Salmon-Steelhead Investigations, Embryo Survival and Emergence Studies, Idaho Fish and Game Deq., Boise, 1969.

Herbicides may indirectly affect stream ecosystems either positively or negatively. The degree of impact is a function of the extent, level, patterns, and timing of applications. Evaluation of potential impacts of herbicides on stream ecosystems must incorporate all of these factors to achieve an accurate assessment.

## INSECTICIDES

Application of forest insecticides can indirectly influence stream ecosystems, primarily through the mortality of terrestrial or aquatic insects. These insects have relatively short life cycles (often 1 year or less); therefore, these communities can be expected to recover in less than 5 years. For this reason, indirect effects of forest insecticides on stream ecosystems may be more dramatic than those of herbicides, but they are of shorter duration.

Benthic algal commmunities in streams are frequently controlled by grazing invertebrates. The mortality of these aquatic invertebrates from insecticides may release the primary producers and result in higher standing crops. In streams in which insecticides were released directly, either intentionally or accidentally, standing crops of primary producers have been observed to increase from 2- to 20-fold (Barnley and Prentice 1958, Hynes 1961, Binns 1967, Chutter 1970). Similar responses have been observed in streams in watersheds that were treated with DDT for control of forest insects (Adams et al. 1949, Morgan and Kremer 1952, Webb and MacDonald 1958, Filteau 1959). Benthic algal communities are reduced as soon as invertebrate communities recover (Chutter 1970).

Insecticides usually cause direct mortality of stream invertebrates as a result of toxicity. Those invertebrates that are resistant or have short generations may actually increase in number or size because of decreased competition, decreased predation, or increased algal food supply. In a Canadian stream that was inadvertently contaminated with gamma-BHC, populations of oligochaetes and midges increased (Hynes 1961). This increase was attributed to mortality of predators. An increase in small chironomids was observed after aerial application of DDT to forests in New Brunswick (Ide 1967). A decrease in predatory insects was also observed in this stream. A similar pattern of changes in invertebrate community structure was observed in streams within watersheds treated with carbaryl for control of spruce budworm (Courtemanch and Gibbs 1980).

Recolonization of streams affected by insecticides is dominated initially by invertebrates with short life cycles. Aquatic insects with life cycles of 1 year or more require several years to return to pretreatment populations. In addition to re-entry, survival, and reproduction, relative success of longer lived species is influenced by competition with established short-lived species. Predators tend to have long life cycles relative to other types of invertebrates; therefore, invertebrate communities do not return to previous levels of predation for several years. Full recovery of invertebrate communities therefore involves several complex interactions and may require 5 to 10 years.

Invertebrate predators have also been observed to increase after application of forest insecticides. Nigronia (dobsonfly) larvae populations increased in streams flowing through watersheds in Connecticut that were treated for spruce budworm (Hitchcock 1965). Other populations of predator insects, such as plecopterans, were observed to decrease during this period.

Contamination of streams with insecticides can cause increased drift of aquatic insects (Crouter and Vernon 1959) and increased entry of terrestrial insects (Warner and Fenderson 1962). These insects are ingested by drift-feeding fish such as trout and salmon. This may result in an indirect toxic effect on the fish. If the toxic effect is slight or nonexistent, this sudden increase in food may cause a brief acceleration of growth. Reduced competition resulted in greater growth of surviving brook trout after forest spraying of DDT in Maine (Warner and Fenderson 1962). Over the long term, however, the decrease in populations of benthic invertebrates will most likely result in decreased growth and production of fish populations. Recovery of fish populations is therefore determined by recovery of invertebrate communities.

## FERTILIZERS

Forests are often fertilized in the Pacific Northwest during several decades after logging. Urea is the most common fertilizer. It is quickly converted to ammonium or nitrate in the soil; therefore, nitrogen can enter streams in the form of urea, ammonium, or nitrate. Most of the urea that enters streams does so within the first 48 hours after application (Moore 1975a, b). After that, nitrogen enters streams primarily as nitrate. Fertilization generally results in nitrogen increases in stream water of 50 mg/liter or less, and these pulses of increased nitrogen last for about 1 year (Fredriksen et al. 1975; Moore 1975a, b).

As previously described, streams in the Pacific Northwest are commonly nitrogen limited. Primary production in such streams may be enhanced by increased nitrogen (Thut and Haydu 1971, Stockner and Shortreed 1976, Gregory 1980). The response to nutrient addition depends on light intensities because heavily shaded streams are light limited. Nutrient stimulation of primary production occurs only with sufficient light

intensity (Gregory 1980). Because most fertilized watersheds are less than 40 years old, fertilization probably results in enhanced primary production in the streams.

Increased primary production can result in greater production of consumers. Greater insect and trout production in open streams has been observed in many studies (Albrecht and Tesche 1961, Albrecht 1968, LeCren 1969, Mills 1969, Hall et al. 1978). Greater primary production in open reaches has been proposed as the cause for these increases. Fertilization could therefore result in greater production of trout and salmon. This increase would be limited to less than 5 years at best, but would be extended by repeated application of fertilizer at 5- or 10-year intervals.

## FIRE RETARDANTS

Chemical fire retardants such as ammonium sulfate, ammonium polyphosphate, or diammonium phosphate are used extensively in the Pacific Northwest for the suppression and control of forest fires. Fires often start on ridgetops, away from streams. As fires develop, they often sweep across streams and rivers, so direct entry of fire retardants into streams is possible.

Application of fire retardants usually results in increased concentrations of ammonia in stream waters (see footnote 2). These concentrations may range from 0.01 to 100 mg/liter of nitrogen. As discussed in the sections on herbicides and fertilizers, such nitrogen increases in streams of the Pacific Northwest have a great potential for stimulation of primary production and subsequent enhancement of higher trophic levels. Increased production of aquatic biota could be precluded if toxic effects occurred. Potential indirect effects on mortality of invertebrates or fish described in the section on indirect effects of insecticides would apply to fire retardants, if concentrations in streams were sufficient to cause mortality. In an experimental release of a fire retardant containing diammonium phosphate, no significant effects on benthic invertebrates or fish were observed (see footnote 2). The pulsed nature of the introduction may have prevented the stimulatory effect that might result when a large area is treated, with a longer release of nitrogen to the stream. Although most fire-retardant drops will occur along streams, retardants could be dropped in or around basins with longer hydrologic retention times than streams, such as oligotrophic lakes, bogs, or swamps. In such aquatic systems, the effect of fire retardants may be prolonged and exaggerated.

Included in the concept of using fire retardants to control forest fires is the basic consideration of the ramifications of controlling or not controlling fires. Fire is a natural reset mechanism in forests of the Pacific Northwest and a fundamental component of terrestrial plant succession. Human activities have duplicated many of the characteristics of fire in clearcutting and burning and have converted a major portion of the forests of the Pacific Northwest to a pioneer stage of succession. If fire retardants are not used and the land is allowed to burn, certain changes will occur in the terrestrial system and these will affect aquatic ecosystems. These effects were reviewed by Norris et al. (see footnote 2) and Swanston (1980). Briefly, potential effects of fire on anadromous fish habitat may include the following: decreased input of leaves and needles; increased input of wood; increased sedimentation; increased streamflow; increased solar radiation at the water surface; increased stream temperature; and increased nutrient inputs. The previous discussions of the effects of herbicides and fertilizers have dealt with these factors, and the potential indirect effects described would apply to watersheds that have been burned. Indirect effects of fire retardants must be weighed against those that would result if fires were not controlled, for accurately assessing the hazards of fire retardants.

## GENERAL PERSPECTIVES ON INDIRECT EFFECTS OF FOREST CHEMICALS

Forest chemicals have great potential for indirectly altering aquatic communities and anadromous fish habitat. Such changes must be examined within the context of all land-use practices. Forest chemicals are seldom used on watersheds that have not been previously altered; therefore, impacts of forest chemicals must be considered in relation to previous or simultaneous effects of other practices on habitat of anadromous fish.

Herbicides modify the natural patterns of terrestrial plant succession on logged watersheds so that the duration of early deciduous-dominated stages is reduced and coniferous vegetation develops more rapidly. The following characteristics of aquatic systems are influenced by the alteration of terrestrial succession: allochthonous organic inputs; tree and shrub canopy over the stream; stream chemistry; and sedimentation rates. These factors are major determinants of the structure and function of stream ecosystems and are affected by logging, with or without the use of herbicides. The basic effect of herbicides is to extend the early stages of watershed recovery, minimize intermediate stages, and accelerate development of coniferous stages. Potential indirect effects of herbicides on aquatic ecosystems must be therefore viewed within this successional framework.

Fertilizers, like herbicides, are applied to logged watersheds to stimulate production of vegetation. Nutrient inputs to streams from application of fertilizer may influence aquatic communities, particularly through stimulation of primary producers; however, these aquatic communities will have already been altered by the effects of logging. Fertilizers may indeed enhance many of the stimulatory effects of logging on aquatic primary producers. Fertilizers are usually applied after canopy closure to avoid stimulating growth of competing species and provide greater utilization by conifers. Fertilization at 5-year intervals could gradually increase nitrogen concentration in forest streams at base flow.

Fire retardants, unlike herbicides and insecticides, are generally applied while watersheds are being directly affected--in this case by fire. Fire has many impacts on anadromous fish habitat; these effects were reviewed by Swanston (1980). Indirect effects of fire retardant are generally limited to stimulation of primary production, and even that effect is greatly influenced by the fire itself through reduction of canopy over the stream.

Insecticides, more than any other forest chemical, may be applied to watersheds that are least influenced by human activities. Even with insecticides, the impacts of the chemical must be viewed in relation to effects on aquatic systems of insect damage to forests. These insect-related effects would be much less severe than the effects associated with logging or fire, but still must be incorporated into decisionmaking processes.

In assessing potential indirect effects of forest chemicals on anadromous fish habitat, land managers must consider the influence of protective measures (particularly buffer strips) on the patterns and processes of aquatic systems that have been described. Frequently, corridors along streams or around lakes are left unsprayed and the terrestrial communities and processes within these "spray buffer strips" may be practically identical to similar areas in untreated watersheds. Effects of chemical spraying would have to be transferred through such spray buffer zones and would therefore have much less impact on aquatic communities. In clearcut watersheds where buffer strips of uncut vegetation are left, the use of spray buffer strips would be even more effective in reducing potential indirect effects of forest chemicals. If buffer strips of uncut vegetation and no-spray zones are used in watershed management, many of the patterns of indirect effects of forest chemicals on stream ecosystems described in this chapter would not occur.

Forest chemicals are a major tool in managing our Nation's forests. Risks in the use of chemicals must be evaluated, however. Direct toxic effects of chemicals on aquatic organisms are major concerns; forest chemicals may have indirect effects on aquatic ecosystems at concentrations much lower than those observed to cause mortality. Potential effects of forest chemicals must be evaluated on the basis of four factors:

- Changes in aquatic communities caused by forest chemicals;
- Subsequent changes in other communities of aquatic organisms;
- Alteration of terrestrial systems that influence aquatic ecosystems; and
- Effects on patterns of recovery in watersheds that have already been altered by logging or fire.

Though few studies of indirect effects of forest chemicals on anadromous fish habitat are available to land managers, the perspectives presented in this paper will provide a basis for evaluating potential indirect effects and designing management systems to minimize them.

## RESEARCH NEEDS

The greater the amount and quality of information available on any subject, the more certain a decisionmaker can be of reaching correct conclusions about it. This fact and the desire to reach correct conclusions prompts scientists to prepare lengthy lists of research needs, many of which seem to be endless repetitions of earlier lists. All research needs are not equally important. We have attempted to focus attention on those gaps in knowledge that we believe have resulted in the greatest uncertainty in the information presented in the earlier part of this paper. We identify these gaps specifically. We believe they are discrete research areas that are small enough to be solved by a single scientist or a small group of scientists. No one area will require major long-term grants or funding programs, although in aggregate, the solution of these problems will require substantial effort. We present the list of research needs in the order the subjects appear in the paper.

## BEHAVIOR OF CHEMICALS IN THE ENVIRONMENT

- Quantify the role of buffer strips in influencing the concentration of forest chemicals in streams.

Research and practice have demonstrated that buffer strips reduce the entry of chemicals into forest streams, but the degree of protection provided by strips of different widths has not been quantified. Some relatively simple experiments are needed to show the degree of improvement that can be achieved with buffer strips of various widths.

- Determine the pattern of entry of atrazine, fosamine ammonium, glyphosate, and dinoseb herbicides, as well as fire retardants, into western forest streams under actual conditions of use.

Most of the research and monitoring of the entry of chemicals into streams, particularly in connection with operational application, were done when the phenoxy herbicides were the predominant forest chemicals. Consequently, few data are available on other forest chemicals. The lack of data is particularly acute for atrazine, fosamine ammonium, glyphosate, and dinoseb herbicides and fire retardants.

- Determine more precisely the fate of all forest chemicals in forest streams.

Almost no data are available on the distribution of chemicals among the various parts of western forest stream systems. The data used in our paper are mostly from laboratory studies or where the chemical in question was intentionally applied to ponds or slow-moving streams for aquatic weed control. Extensive work in this area is not needed, only enough to establish the degree to which concepts developed in other types of aquatic systems fit in aquatic systems used by anadromous fish.

## TOXICITY OF CHEMICALS TO AQUATIC SPECIES

- Determine the toxicity characteristics of the combinations of forest chemicals that are likely to be applied together.

Studies on the effects of combinations of chemicals (for example, picloram and 2,4-D) have generally been restricted to plants. Similar work with sensitive aquatic vertebrates and invertebrates is required to assess adequately the effects of combined chemicals.

- Characterize the interaction between concentration and duration of exposure among forest chemicals and the more sensitive aquatic species.

Most toxicity tests hold the concentration of chemical constant for a specified period (such as 24, 48, or 96 hours) and evaluate organism response soon afterward. Exposure of aquatic organisms in the field is typically to concentrations of chemical that increase to a peak within a few hours after aerial application and then decrease rapidly to much lower levels in a few hours. A relation needs to be established that will permit the use of the extensive existing toxicity data base (which was developed by using constant exposure methods) in evaluating the toxicological significance of field exposure data. In this paper, we used an integral of the time-concentration curve, but this approach has not been fully validated.

- Determine if the results of classical 96-hour exposure tests are adequate predictors of the long-term well-being of aquatic organisms.

Nearly all the toxicity testing on aquatic organisms has used short-term exposure and only short-term observation of effects. Research is needed to determine if long-term latent effects result from short-term exposure. Tests with a few chemicals and a few key species may be sufficient to establish this point. With a few notable exceptions, we do not believe that latent effects will develop from short-term exposure to most toxicants.

## INDIRECT EFFECTS OF FOREST CHEMICALS

- Quantify indirect effects of forest chemicals on aquatic organisms under field conditions.
- Determine sublethal effects of forest chemicals on aquatic organisms.

Research on these areas is critically needed before adequate conceptual frameworks can be developed for designing research to investigate indirect effects of forest chemicals under field conditions. Indirect effects on aquatic ecosystems will most probably involve several types of aquatic organisms; such effects, therefore, would be much more complex and subtle than direct toxic effects. Research must be tightly focused and properly timed to permit the observation of changes in aquatic communities. Because indirect effects may be caused by concentrations of chemicals much lower than lethal concentrations, further research on indirect effects of forest chemicals on aquatic ecosystems is required to assure safer use of chemicals in the forest environment.

# CONCLUSIONS

The use of forest chemicals can result in both direct and indirect effects on anadromous fish and their habitat. Direct toxic effects are those resulting from the exposure of fish to a chemical in water, food, or sediment. The potential for direct effects can be estimated, based on knowledge of the toxicity characteristics of the chemical and its movement, persistence, and fate in the environment.

The most important process by which chemicals enter streams is direct application, but drift from nearby treatment areas or units is also important. Mobilization of residues in ephemeral stream channels during the first storms after application is sometimes important. All three processes can be influenced by forest managers. Selection and orientation of spray units to avoid streams, and attention to the details of application to avoid drift, will minimize chemical entry into streams and thereby reduce the likelihood of direct toxic effects on stream organisms.

The margin of safety (no-effect level/exposure level) is a good index to the probability that the use of a specific forest chemical will result in direct effects on anadromous fish. The larger the margin of safety, the less likely direct effects will occur. Margins of safety of less than one indicate direct effects are likely to occur. We calculated the following margins of safety for fish, based on the maximum acute and short-term chronic exposures likely to occur in operational use of these chemicals:

| Chemical | Margin of safety: Acute | Margin of safety: Short-term chronic |
|---|---|---|
| 2,4-D (PGBE ester) | 5 | 1.4 |
| Picloram (Tordon 101) | 43 | 50 |
| Atrazine | 24 | 3.5 |
| MSMA | 500 | 26[1]/-- |
| Fosamine ammonium | 2640 | 379 |
| Glyphosate | 12 | 1.7 |
| Dinoseb | 0.2 | < 0.1 |
| Malathion | 0.8 | 2.2 |
| Carbaryl | 22 | 36 |
| Azinphos-methyl | 26[1]/-- | 26[1]/-- |
| Carbofuran | 26[1]/-- | 26[1]/-- |
| Acephate | 500 | 667 |
| Fertilizer | 1 | 0.1 |
| Fire retardant | 1 | 27[2]/-- |

These margins of safety will be 5 to 10 times greater when streams are not included in areas to be treated, buffer strips are used, and full attention is given to the details of application to prevent drift and direct application to surface water.

Indirect effects are manifested through chemically induced changes in the density and species composition of aquatic and terrestrial plants and insects. These effects may include alteration of nutrient, sediment, and temperature characteristics of the water, and changes in cover, food, or some other environmental characteristic that is important to the well-being of anadromous fish. These changes have not been as thoroughly studied as the direct effects, but may be the most likely to occur.

---

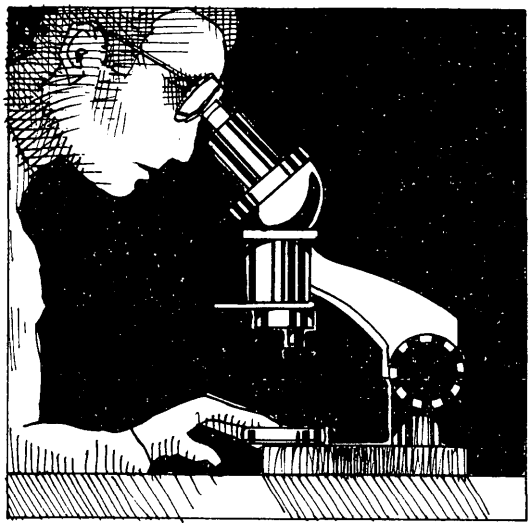
[1]/Current pattern of use does not result in measurable residues of chemical in water; therefore, exposure is very small and the margin of safety is very large.

---

[2]/Margin of safety not calculated because no value for exposure is available.

# ACKNOWLEDGMENTS

We acknowledge the excellent technical reviews of this lengthy paper provided by Dan Woodward, Duane Moore, and Marvin Montgomery, and the special efforts by Carroll Burkett and Westfornet to verify the accuracy of the citations.



# LITERATURE CITED

Adams, Lowell, Mitchell G. Hanavan, Neil W. Hosley, and David W. Johnston. 1949. The effects on fish, birds, and mammals of DDT used in the control of forest insects in Idaho and Wyoming. J. Wildl. Manage. 13(3):245-254.

Adelman, Ira R., Lloyd L. Smith, Jr., and Gary D. Siesennop. 1976. Chronic toxicity of Guthion to the fathead minnow (Pimephales promelas Rafinesque). Bull. Environ. Contam. Toxicol. 15(6):726-733.

Aho, Richard Stanley. 1976. A population study of the cutthroat trout in an unshaded and shaded section of stream. M.S. thesis. 87 p. Oreg. State Univ., Corvallis.

Albrecht, M. L., and F. W. Tesche. 1961. Das Wachstum der Bachforelle (Salmo trutta fario L.) in der Polenz in Abhangigkeit von Verschieden Umweltbedingungen. Z. Fisch. Nat. Forsch. 10:253-273.

Albrecht, Marie-Luise. 1968. Die Wirkung des Lichtes auf die quantitative Verteilung der Fauna in Fliessgewasser. Limnologica 6: 71-82.

Allison, Donald T. 1977. Use of exposure units for estimating aquatic toxicity of organophosphate pesticides. U.S. Environ. Prot. Agency Ecol. Res. Ser. EPA-600/3-77-077. 25 p. Duluth, Minn.

Altom, J. D., and J. F. Stritzke. 1972. Persistence of brush control herbicides in a blackjack and post oak soil. Proc. South. Weed Sci. Soc. 25:302.

Anderson, Charles A., J. C. Cavagnol, Charles J. Cohen, A. D. Cohick, R. Thomas Evans, Leroi J. Everett, J. Hensel, Richard P. Honeycutt, E. R. Levy, Walter W. Loeffler, Dallas L. Nelson, Thaddeus Parr, T. B. Waggoner, and James W. Young. 1974. Guthion® (azinphosmethyl): organophosphorus insecticide. Res. Rev. 51:123-180.

Anderson, J. M. 1968. Effect of sublethal DDT on the lateral line of brook trout, Salvelinus fontinalis. J. Fish. Res. Board Can. 25(12):2677-2682.

Anderson, J. M. 1971. Assessment of the effects of pollutants on physiology and behavior. Proc. R. Soc. Ser. B. Biol. Sci. 177:307-320.

Anderson, J. M., and M. R. Peterson. 1969. DDT: sublethal effects on brook trout nervous system. Science 164:440-441.

Aquatic Life Advisory Committee. 1955. Aquatic Life History Committee of the Ohio River Valley Water Sanitation Commission. Aquatic life water quality criteria: first progress report. Sewage Ind. Wastes 27:321-331.

Axe, Jim A., Aubra C. Mathers, and Allen F. Wise. 1969. Disappearance of atrazine, propazine and trifluraline from soil and water. (Abstr.) Proc. South. Weed Control Conf. 22:367.

Ball, I. R. 1967. The relative susceptibilities of some species of freshwater fish to poisons - 1. Ammonia. Water Res. 1:767.

Barnett, A. P., E. W. Hauser, A. W. White, and J. H. Holladay. 1967. Loss of 2,4-D in washoff from cultivated fallow land. Weeds 15:133-137.

Barnley, G. R., and M. A. Prentice. 1958. Simulium neavei in Uganda. East Afr. Med. J. 35:475.

Barrons, K. C., G. E. Lynn, and J. D. Eastman. 1953. Experiments on the reduction of high temperature injury to cotton from DNOSBP. Proc. South. Weed Control Conf. 6:33-37.

Baudish, Oscar, and Laurence W. Bass. 1922. Eisen als licht-chemischer Katalyzator. 1. Uber die Zersetzung von Ferrocyankalium im tageslicht. Chem. Berichte 55B:2698-2706.

Bender, M. E., and J. R. Westman. 1976. The toxicity of malathion and its hydrolysis products to the eastern mudminnow, Umbra pygmaea (DeKay). Chesapeake Sci. 17(2):126-128.

Bender, Michael E. 1969. The toxicity of the hydrolysis and breakdown products of malathion to the fathead minnow (Pimephales promelas Rafinesque). Water Res. 3:571-582.

Bengtson, G. W. 1979. Forest fertilization in the United States: progress and outlook. J. For. 77(4):222–229.

Benson, W. R. 1969. The chemistry of pesticides. Ann. N.Y. Acad. Sci. 160:7–20.

Bernhardt, John, Joseph Praveza, and Harry Tracy. 1978. Aquatic monitoring of the 1977 spruce budworm Sevin-4-oil aerial spray project in Washington State. State of Wash. Dep. Ecol., Tech. Rep. DOE 78–4. 22 p. Olympia, Wash.

Binns, Niles Allen. 1967. Effects of rotenone treatment on the fauna of the Green River, Wyoming. Wyo. Game and Fish Comm., Fish. Res. Bull. 1:114.

Birk, L. A., and F. E. B. Roadhouse. 1964. Penetration and persistence in soil of the herbicide atrazine. Can. J. Plant Sci. 44:21–27.

Bjornn, T. C., M. A. Brusven, M. Molnau, F. J. Watts, and R. L. Wallace. 1974. Sediment in streams and its effect on aquatic life. 47 p. Idaho Water Resour. Res. Inst., Moscow. (Available from NTIS).

Blahm, Theodore Henry. 1979. Toxicity of chemical fire retardants to juvenile coho salmon and rainbow trout. Ph. D. Diss. Abstr. Int. Sci. Eng. 39:4115-B.

Bocsor, J. George, and Thomas F. O'Connor. 1975. Impact on the aquatic ecosystem. In Environmental impact study of aerially applied Orthene on a forest and aquatic ecosystem, p. 29–47. LOTEL Rep. 174. SUNY Lake Ont. Environ. Lab., Oswego, N.Y.

Böhm, H. H., and H. Mueller. 1976. Model studies on the accumulation of herbicides by microalgae. Naturwissenschaften 63(6):296.

Bollag, Jean–Marc, and Shu–Wyn Liu. 1971. Degradation of Sevin by soil microorganisms. Soil Biol. and Biochem. 3:337-345.

Boyd, C. E., and J. W. Sowles. 1978. Nitrogen fertilization of ponds. Trans. Am. Fish. Soc. 107(5):737-741.

Brown, George W. 1969. Predicting temperatures of small streams. Water Resour. Res. 5(1):68-75.

Brusven, M. A., and K. V. Prather. 1974. Influence of stream sediments on distribution of macrobenthos. J. Entomol. Soc. B.C. 71:25-32.

Burdick, G. E., H. J. Dean, and E. J. Harris. 1960. The effect of Sevin upon the aquatic environment. N.Y. Fish and Game J. 7:14-25.

Burdick, George Edgar, and Morris *Lipschuetz*. 1948. Toxicity of ferro- and ferricyanide solutions to fish, and determination of the cause of mortality. Trans. Am. Fish. Soc. 78:192-202.

Burns, James W. 1972. Some effects of logging and associated road construction on northern California streams. Trans. Am. Fish. Soc. 101(1):1-17.

Burrows, R. E. 1964. Effects of accumulated excretory products on hatchery–reared salmonids. USDI Bur. Sport Fish. and Wildl. Res. Rep. 66. U.S. Gov. Print. Off., Washington, D.C.

Burt, Gordon W. 1974. Volatility of atrazine from plant, soil, and glass surfaces. J. Environ. Qual. 3(2):114-117.

Butler, Phillip A. 1965. Effects of herbicides on estuarine fauna. Proc. South. Weed Conf. 18:576-580.

Capizzi, Joseph, and Glenn Fisher. 1977. Oregon insect control handbook. 20th rev. 220 p. Oreg. State Univ., Corvallis.

Capizzi, Joseph, and J. M. Witt, Eds. 1971. Pesticides, pest control and safety on forest and rangelands. 259 p. Oreg. State Univ., Corvallis.

Carlson, A. R. 1972. Effects of long-term exposure to carbaryl (Sevin) on survival, growth, and reproduction of the fathead minnow (Pimephales promelas). J. Fish. Res. Board Can. 29(5):583-587.

Caro, Joseph H., Horatio P. Freeman, Dwight W. Glotfelty, Benjamin C. Turner, and William M. Edwards. 1973. Dissipation of soil-incorporated carbofuran in the field. J. Agric. Food Chem. 21:1010-1015.

Caro, Joseph H., Horatio P. Freeman, and Benjamin C. Turner. 1974. Persistence in soil and losses in runoff of soil-incorporated carbaryl in a small watershed. J. Agric. Food Chem. 22(5):860-863.

Cederholm, Carl J., and Lawrence C. Lestelle. 1974. Observations on the effect of landslide siltation on salmon and trout resources of the Clearwater River, Jefferson County, Washington, 1972-73. Univ. Wash. Fish. Res. Inst., FRI-UW-7404. 133 p. Seattle, Wash.

Chapman, D. W. 1963. Physical and biological effects of forest practices upon stream ecology. In Symposium - Forest Watershed Management, p. 321-330. Society of American Foresters and Oregon State University, School of Forestry, Corvallis, Oreg.

Chiou, Cary T., Virgil H. Freed, David W. Schmedding, and Roger L. Kohnert. 1977. Partition coefficient and bioacummulation of selected organic chemicals. Environ. Sci. Technol. 11(5):475-478.

Chrzanowski, Robert L., Jerry C-Y Han, and Colin L. McIntosh. 1979. Metabolism of [$^{14}$C] fosamine ammonium in the rat. J. Agric. and Food Chem. 27(3):550-554.

Chutter, F. M. 1970. Hydrobiological studies in the catchment of Vaal Dam, South Africa. Part 1. River zonation and the benthic fauna. Int. Rev. ges. Hydrobiol. 55:445-494.

Chykaliuk, P. B., J. R. Abernathy, and J. R. Gipson. 1981. Bibliography of glyphosate. MP-1443. 85 p. Texas Agric. Exp. Stn., Texas A&M Univ., College Station.

Comes, R. D., V. P. Bruns, and A. D. Kelley. 1976. Residues and persistence of glyphosate in irrigation water. Weed Sci. 24(1):47-50.

Cook, Gary H., James C. Moore, and David L. Coppage. 1976. The relationship of malathion and its metabolites to fish poisoning. Bull. Environ. Contam. Toxicol. 16:283-290.

Cope, Oliver B. 1966. Contamination of the freshwater ecosystem by pesticides. Pesticides in the environment and their effects on wildlife: Proceedings of an Advanced Study Institute, sponsored by the North Atlantic Treaty Organization, Monks Wood Experimental Station, England, July 1-14, 1965. N. W. Moore, ed. In J. Appl. Ecol., Suppl. to vol. 3, p. 33-44. Blackwell Scientific Publications, Oxford, England.

Cope, Oliver B., Edward M. Wood, and George H. Wallen. 1970. Some chronic effects of 2,4-D on the bluegill (Lepomis macrochirus). Trans. Am. Fish. Soc. 99(1):1-12.

Cordone, Almo J., and Steven Pennoyer. 1960. Notes on silt pollution in the Truckee River Drainage. Calif. Dep. Fish and Game, Inland Fish. Adm., Rep. 60-14. 25 p. Sacramento, Calif.

Courtemanch, D. L., and K. E. Gibbs. 1980. Short- and long-term effects of forest spraying of carbaryl (Sevin-4-oil) on stream invertebrates. Can. Entomol. 112(3):271-276.

Crouter, R. A., and E. H. Vernon. 1959. Effects of black-headed budworm control on salmon and trout in British Columbia. Can. Fish-Cult. 24:23-40.

Cummins, Kenneth W., and George H. Lauff. 1969. The influence of substrate particle size on the microdistribution of stream macrobenthos. Hydrobiologia 34(2):145-181.

D'amore, Geovanni, and Athos Bellorno. 1958. Photolysis of solutions of potassium ferrocyanide. Attiv. Soc. Peloritana Sci. Fis. Mat. e Nat. 5:449-457.

Davey, Ronald B., M. V. Meisch, and Frank L. Carter. 1976. Toxicity of five ricefield pesticides to the mosquitofish, Gambusia affinis, and green sunfish, Lepomis cyanellus, under laboratory and field conditions in Arkansas. Environ. Entomol. 5(6):1053-1056.

Davis, E. A., P. A. Ingebo, and C. P. Pase. 1968. Effect of a watershed treatment with picloram on water quality. USDA For. Serv. Res. Note RM-100. 4 p. Rocky Mt. For. and Range Exp. Stn., Ft. Collins, Colo.

Davis, Franklin E., and Frank L. Selman. 1954. Effects of water upon movements of dinitro weed killers in the soil. Weeds 3:11-20.

Davis, James T., and W. S. Hardcastle. 1959. Biological assay of herbicides for fish toxicity. Weeds 7:397-404.

Davis, James T., and Janice S. Hughes. 1963. Further observations on the toxicity of commercial herbicides to bluegill sunfish. Proc. South. Weed Conf. 16:337-340.

Dennis, D. S., W. H. Gillespie, R. A. Maxey, and R. Shaw. 1977. Accumulation and persistence of picloram (Tordon 10K) in surface water and bottom sediments in West Virginia. Arch. Environ. Contam. Toxicol. 6:421-433.

Desi, I., G. Dura, L. Gonczi, Z. Kneffel, A. Strohmayer, and Z. Szabo. 1976. Toxicity of malathion to mammals, aquatic organisms, and tissue culture cells. Arch. Environ. Contam. Toxicol. 3:410-425.

Devine, James M. 1975. Persistence of Orthene residues in the forest and aquatic environment. In Environmental impact study of aerially applied Orthene on a forest and aquatic ecosystem, p. 48-82. LOTEL Rep. 174. SUNY Lake Ont. Environ. Lab., Oswego, N.Y.

Dickens, Ray, and A. E. Hiltbold. 1967. Movement and persistence of methanearsonates in soil. Weeds 15(4):299-304.

Doudoroff, Peter. 1956. Some experiments on the toxicity of complex cyanides to fish. Sewage and Ind. Wastes (Water Pollut. Control Fed. J.) 28(8):1020-1040.

Doudoroff, Peter. 1976. Toxicity to fish of cyanides and related compounds: a review. U.S. Environ. Prot. Agency Ecol. Res. Ser. EPA 600/3-76-038. 155 p. Duluth, Minn.

Douglas, G. W. 1974. Ecological impact of chemical fire retardants: a review. Environ. Can., For. Serv., North For. Res. Cent. Rep. NOR-X-109. 33 p. Edmonton, Alberta.

Douglass, J. E., D. R. Cochrane, G. W. Bailey, J. I. Teasley, and D. W. Hill. 1969. Low herbicide concentration found in streamflow after a grass cover is killed. USDA For. Serv. Res. Note SE-108. 3 p. Southeast. For. Exp. Stn., Asheville, N.C.

Dyrness, C. T. 1973. Early stages of plant succession following logging and burning in the western Cascades of Oregon. Ecology 54(1):57-69.

Eaton, John G. 1970. Chronic malathion toxicity to the bluegill (Lepomis macrochirus Rafinesque). Water Res. 4:673-684.

Edwards, W. M., G. B. Triplett, Jr., and R. M. Kramer. 1980. A watershed study of glyphosate transport in runoff. J. Environ. Qual. 9(4):661-665.

Eichelberger, James W., and James J. Lichtenberg. 1971. Persistence of pesticides in river water. Environ. Sci. Technol. 5(6):541-544.

Ellgehausen, Holm, Johann A. Guth, and Herbert O. Esser. 1980. Factors determining the bioaccumulation potential of pesticides in the individual compartments of aquatic food chains. Ecotoxicol. and Environ. Saf. 4:134-157.

Erman, Don C., J. Dennis Newbolt, and Kenneth B. Roby. 1977. Evaluation of streamside buffer strips for protecting aquatic organisms. Contrib. 165. 48 p. Calif. Water Resour. Cent., Univ. Calif., Davis.

Erne, Kurt. 1975. Phenoxy herbicide residues in Swedish fish and wildlife. In Environmental quality and safety. Suppl. Vol. 3, pesticides. p. 192-195. Frederick Coulston and Friedhelm Korte, eds. 3d Int. Congr. Pestic. Chem., Georg Thieme, Stuttgart.

Everest, F. H., and D. W. Chapman. 1972. Habitat selection and spatial interaction by juvenile chinook salmon and steelhead trout in two Idaho streams. J. Fish. Res. Board Can. 29(1):91-100.

Everett, L. J., A. Anderson, and Daniel McDougall. 1966. Nature and extent of Guthion residues in milk and tissues resulting from treated foliage. J. Agric. Food Chem. 14:47-52.

Filteau, G. 1959. Effets des vaporisations aeriennes au DDT sur les insects aquatiques. Nat. Can. (P.Q.) 86(517):113-128.

Flavell, Thomas H., Scott Tunnock, and Hubert E. Meyer. 1977. A pilot project evaluating trichlorfon and acephate for managing western spruce budworm. Helena National Forest, Montana, 1976. USDA For. Serv., State and Priv. For., Rep. 77-16. 48 p. For. Insect and Dis. Manage., North. Reg., Missoula, Mont.

Folmar, L. C., H. O. Sanders, and A. M. Julin. 1979. Toxicity of the herbicide glyphosate and several of its formulations to fish and aquatic invertebrates. Arch. Environ. Contam. Toxicol. 8:269-278.

Folmar, Leroy C. 1976. Overt avoidance reaction of rainbow trout fry to nine herbicides. Bull. Environ. Contam. Toxicol. 15(5):509-514.

Frank, R., and G. J. Sirons. 1979. Atrazine: its use in corn production and its loss to streamwater in southern Ontario, 1975-1977. Sci. Total Environ. 12:223-239.

Franklin, J. F., and C. T. Dyrness. 1973. Natural vegetation of Oregon and Washington. USDA For. Serv. Gen. Tech. Rep. PNW-8. 417 p. Pac. Northwest For. and Range Exp. Stn., Portland, Oreg.

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. In Forest soils and forest land management. p. 283-313. B. Bernier and C. H. Winget, eds. Les Presses de l'Université, Laval, Quebec.

Freed, Virgil H., Cary T. Chiou, and David W. Schmedding. 1979. Degradation of selected organophosphate pesticides in water and soil. J. Agric. Food Chem. 27:706-708.

Fuhremann, Tom W., and E. Paul Lichtenstein. 1980. A comparative study of the persistence, movement, and metabolism of six carbon-14 insecticides in soils and plants. J. Agric. Food Chem. 28(2):446-452.

Getzin, L. W. 1973. Persistence and degradation of carbofuran in soil. Environ. Entomol. 2:461-467.

Geyer, H., A. G. Kraus, and W. Klein. 1980. Relationship between water solubility and bioaccumulation potential of organic chemicals in rats. Chemosphere 9:277-291.

Goring, C. A. I., and J. W. Hamaker. 1971. The degradation and movement of picloram in soil and water. Down to Earth 27(1):12-15.

Goring, C. A. I., D. A. Laskowski, J. W. Hamaker, and R. W. Meikle. 1975. Principles of pesticide degradation in soil. In Environmental dynamics of pesticides. Environ. Sci. Res. Vol. 6, p. 135-184. R. Haque and V. H. Freed, eds. Plenum Press, New York.

Grant, B. F., and P. M. Mehrle. 1970. Chronic endrin poisoning in goldfish, Carassius auratus. J. Fish. Res. Board Can. 27(12): 2225-2232.

Gregory, Stanley Vincent. 1980. Effects of light, nutrients, and grazing on periphyton communities in streams. Ph. D. thesis. 151 p. Oreg. State Univ., Corvallis.

Gunkel, G., and B. Streit. 1980. Mechanisms of bioaccumulation of a herbicide (atrazine, S-triazine) in a freshwater mollusc (Ancylus fluviatilis Mull.) and a fish (Coregonus fera Jurine). Water Res. 14:1573-1584.

Gunther, Francis A., and Jane Davies Gunther. 1970. The triazine herbicides. Residue Rev. 32:1-413.

Haas, R. C., C. J. Scifres, M. G. Merkle, R. R. Han, and G. W. Hoffman. 1971. Occurrence and persistence of picloram in grassland water sources. Weed Res. 11:54-62.

Hall, James D., Michael L. Murphy, and Richard S. Aho. 1978. An improved design for assessing impacts of watershed practices on small streams. Verh. Int. Ver. Limnol. 20:1359-1365.

Hall, J. K., M. Pawlus, and E. R. Higgins. 1972. Losses of atrazine in runoff water and soil sediment. J. Environ. Qual. 1(2):172-176.

Han, Jerry C-Y. 1979a. Stability of [14C] fosamine ammonium in water and soils. J. Agric. and Food Chem. 27(3):564-571.

Han, Jerry C-Y. 1979b. Residue studies with ($^{14}C$) fosamine ammonium in channel catfish. J. Toxicol. Environ. Health 5(5):957-963.

Hance, Raymond J. 1976. Adsorption of glyphosate by soils. Pestic. Sci. 7:363-366.

Hansmann, Eugene William. 1969. The effects of logging on periphyton communities of coastal streams. Ph. D. thesis. 120 p. Oreg. State Univ., Corvallis.

Haque, R., and V. H. Freed. 1974. Behavior of pesticides in the environment: "environmental chemodynamics." Residue Rev. 52:89-116.

Haque, R., and V. H. Freed. 1975. Environmental dynamics of pesticides. Environ. Sci. Res. Vol. 6. 387 p. Plenum Press, New York.

Hardy, J. L. 1966. Effects of Tordon herbicides on aquatic food chain organisms. Down to Earth 22(2):11-13.

Harr, R. Dennis 1977. Water flux in soil and subsoil on a steep forested slope. J. Hydrol. 33:37-58.

Hatfield, C. T., and J. M. Anderson. 1972. Effects of two insecticides on the vulnerabillty of Atlantic salmon (Salmo salar) parr to brook trout (Salvelinus fontinalis) predation. J. Fish. Res. Board Can. 29(1):27-29.

Hatfield, C. T., and P. H. Johansen. 1972. Effects of four in'secticides on the ability of Atlantic salmon parr (Salmo salar) to learn and retain a simple conditioned response. J. Fish. Res. Board Can. 29(3) :315–321.

Haven, Dexter, 1963. Mass treatment with 2,4-D of milfoil in tidal creeks in Virginia. Proc. South. Weed Conf. 16:345-350.

Henderson, C., Q. H. Pickering, and C. M. Tarzwell. 1959. Relative toxicity of ten chlorinated hydrocarbon insecticides to four species of fish. Trans. Am. Fish. Soc. 88(1):23-32.

Henderson, C., Q. H. Pickering, and C. M. Tarzwell. 1960. The toxicity of organic phosphorus and chlorinated hydrocarbon insecticides to fish. U.S. Public Health Serv. Tech. Rep. W60-3, p. 35–88. Washington, D.C.

Henderson, Croswell, and Quentin H. Pickering. 1958. Toxicity of organic phosphorus insecticides to fish. Trans. Am. Fish. Soc. 87(1):39–51.

Herbert, D. W. M., and D. S. Shurben. 1965. The susceptibility of salmonid fishes to poisons under estuarine conditions - 2. Ammonium chloride. Int. J. Air and Water Pollut. 9:89.

Hewlett, John D., and Alden R. Hibbert. 1961. Increases in water yield after several types of forest cutting. Int. Assoc. Sci. Hydro. Bull. 6:5-17.

Hewlett, John D., and Alden R. Hibbert. 1967. Factors affecting the response of small watersheds to precipitation in humid areas. In Forest hydrology, p. 275–290. W. E. Sopper and H. W. Lull, eds. Pergamon Press, New York.

Hibbert, Alden R. 1967. Forest treatment effects on water yield. In Forest hydrology, p. 527–543. W. E. Sopper and H. W. Lull, eds. Pergamon Press, New York.

Hildebrand, Lloyd D., Druscilla S. Sullivan, and Thomas P. Sullivan. 1980. Effects of Roundup herbicide on populations of Daphnia magna in a forest pond. Bull. Environ. Contam. Toxicol. 25(3):353-357.

Hiltibran, R. C. 1972a. The hydrolysis of adenosine triphosphate by bluegill mitochondria in.the presence of 2,4,5-T and silvex derivatives. Trans. Ill. State Acad. Sci. 65:51-57.

Hiltibran, R. C. 1972b. Effects of the chloro groups of phenoxy compounds on the oxidative phosphorylation. Trans. Ill. State Acad. Sci. 65:81-85.

Hiltibran, Robert C. 1967. Effects of some herbicides on fertilized fish eggs and fry. Trans. Am. Fish. Soc. 96(4):414-416.

Hitchcock, S. W. 1965. Field and laboratory studies of DDT and aquatic insects. Conn. Agric. Exp. Stn. Bull. 668. 25 p. Storrs, Conn.

Hoffman, Robert A. 1957. Toxicity of three phosphorous insecticides to cold water game fish. Mosq. News 17(3) :213.

Hollingsworth, E. B., and W. B. Ennis, Jr. 1953. Some studies on the vapor action of certain dinitro compounds upon young cotton plants. Proc. South. Weed Control Conf. 6 :23–31.

Hornbeck, J. W., R. S. Pierce, and C. A. Federer. 1970. Streamflow changes after forest clearing in New England. Water Resour. Res. 6(4):1124-1132.

Hughes, Janice S., and James T. Davis. 1963. Variations in toxicity to bluegill sunfish of phenoxy herbicides. Weeds 11(1):50-53.

Hunt, D., and C. E. Boyd. 1981. Alkalinity losses from ammonium fertilizers used in fish ponds. Trans. Am. Fish. Soc. 110(1):81-85.

Hunt, R. L. 1978. Instream enhancement of trout habitat. In A national symposium on wild trout management; Proc. San Jose, Calif., p. 19-28. K. Hasagen, ed. California Trout, Inc., San Francisco.

Hulbert, P. J. 1978. Mattawamkeag River studies: 2. Effects of Sevin insecticide on fish and invertebrates in 1976. 98 p. Migratory Fish Res. Inst., Univ. Maine, Orono.

Hurlbert, Stuart H. 1975. Secondary effects of pesticides on aquatic ecosystems. Residue Rev. 57:81-148.

Hynes, H. B. N. 1961. The effect of sheep-dip containing the insecticide BHC on the fauna of a small stream, including Simulium and its predators. Ann. Trop. Med. Parasit. 55(2): 192-196.

Ide, F. P. 1967. Effects of forest spraying with DDT on aquatic insects of salmon streams in New Brunswick. J. Fish. Res. Board Can. 24(4):769-805.

Ingebo, Paul A. 1971. Supression of channel-side chaparral cover increases streamflow. J. Soil Water Conserv. 26(2):79-81.

Iwata, Yutaka, William E. Westlake, James H. Barkley, Glenn E. Carman, and Francis A. Gunther. 1977. Behavior of phenthoate deposits and residues on and in grapefruit, lemons, lemon leaves, oranges, and orange leaves and in the soil beneath orange trees. J. Agric. Food Chem. 25:362-368.

Jenkins, David, Stephen A. Klien, M-S Yang, R. G. Wagenet, and James W. Biggar. 1978. The accumulation, translocation, and degradation of biocides at land wastewater disposal sites: the fate of malathion, carbaryl, diazinon, and 2,4-D butoxyethyl ester.. Water Res. 12(9):713-723.

Jenson, H. L. 1929. On the influence of the carbon:nitrogen ratios of organic material on the mineralization of nitrogen. J. Agric. Sci. 19:71-82.

Johnsen, Thomas N., Jr. 1980. Picloram in water and soil from a semiarid pinyon-juniper watershed. J. Environ. Qual. 9(4):601-605.

Johnsen, Thomas N., Jr., and William L. Warskow. 1980. Picloram dissipation in a small southwestern stream. Weed Sci. 28(5):612-615.

Johnson, Waynon W., and Mack T. Finley. 1980. Handbook of acute toxicity of chemicals to fish and aquatic invertebrates. USDI Fish and Wildl. Serv. Resour. Publ. 137. 98 p. USDI Fish and Wildl. Serv., Washington, D.C.

Jones, J. R. E. 1964. Fish and river pollution. 200 p. Butterworth and Co., London.

Kanazawa, J. 1975. Uptake and excretion of organophosphorus and carbamate insecticides by freshwater fish, Mustugo pseudorasboro Parva. Bull. Environ. Contam. Toxicol. 14(3):346-352.

Kanazawa, J., Allan R. Isensee, and Phillip C. Kearney. 1975. Distribution of carbaryl and 3,5-xylyl methylcarbamate in an aquatic model ecosystem. J. Agric. Food Chem. 23:760-763.

Karinen, J. F., J. G. Lamberton, N. E. Stewart, and L. C. Terriere. 1967. Persistence of carbaryl in the marine estuarine environment: chemical and biological stability in aquarium systems. J. Agric. Food Chem. 15:148-156.

Katz, Max. 1961. Acute toxicity of some organic insecticides to three species of salmonids and to three-spine stickleback. Trans. Am. Fish. Soc. 90(3):264-268.

Kearney, P. C., T. J. Sheets, and J. W. Smith. 1964. Volatility of seven s-triazines. Weeds 12:83-87.

Kenaga, E. E. 1969. Tordon herbicides - evaluation of safety to fish and birds. Down to Earth 25(1):5-9.

Kenaga, Eugene E. 1975. Partitioning and uptake of pesticides in biological systems. In Environmental dynamics of pesticides. Environ. Sci. Res. Vol. 6, p. 217-273. R. Haque and V. H. Freed, eds. Plenum Press, New York.

Kenaga, Eugene E. 1980a. Correlation of bioconcentration factors of chemicals in aquatic and terrestrial organisms with their physical and chemical properties. Environ. Sci. and Technol. 14(5):553-556.

Kenaga, Eugene E. 1980b. Predicted bioconcentration factors and soil sorption coefficients of pesticides and other chemicals. Ecotoxicol. and Environ. Saf. 4:26-38.

Kincheloe, John W., Gary A. Wedemeyer, and David L. Koch. 1979. Tolerance of developing salmonid eggs and fry to nitrate exposure. Bull. Environ. Contam. Toxicol. 23(4/5):575-578.

Klaassen, Harold E., and Ahmed M. Kadoum. 1979. Distribution and retention of atrazine and carbofuran in farm pond ecosystems. Arch. Environ. Contam. Toxicol. 8(3):345-353.

Klingman, Glenn C., and Floyd M. Ashton. 1975. Weed science: principles and practices. 429 p. John Wiley & Sons, New York.

Konrad, J. G., G. Chesters, and D. E. Armstrong. 1969. Soil degradation of malathion, a phosphorodithioate insecticide. Soil Sci. Soc. Am. Proc. 33(2):259-262.

Korn, Sidney. 1973. The uptake and persistence of carbaryl in channel catfish. Trans. Am. Fish. Soc. 102(1):137-139.

Koski, K Victor. 1966. The survival of coho salmon (Oncorhynchus kisutch) from egg deposition to emergence in three Oregon coastal streams. M.S. thesis. 84 p. Oreg. State Univ., Corvallis.

LaFleur, Kermit S. 1976. Movement of carbaryl through congaree soil into groundwater. J. Environ. Qual. 5(1):91-92.

Larson, Gary E., Charles E. Warren, Floyd E. Hutchins, Larry P. Lamperti, David A. Schlesinger, and Wayne K. Seim. 1978. Toxicity of residual chlorine compounds to aquatic organisms. U.S. Environ. Prot. Agency Ecol. Res. Ser. EPA-600/3-78-023. 105 p. Duluth, Minn.

LeCren, E. D. 1969. Estimates of fish populations and production in small streams in England. In Symposium on salmon and trout in streams, p. 269-280. T. G. Northcote, ed. H. R. MacMillan Lectures in Fisheries, Univ. B.C., Vancouver.

Leonard, R. A., G. W. Bailey, and R. R. Swank, Jr. 1976. Transport, detoxification, fate and effects of pesticides in soil and water environments. In Land application of waste materials, p. 48-78. Soil Conserv. Soc. Am., Ankeny, Iowa.

Leonard, R. A., G. W. Langdale, and W. G. Fleming. 1979. Herbicide runoff from upland piedmont watersheds - data and implications for modeling pesticide transport. J. Environ. Qual. 8(2):223-229.

Lewis, Stephen L. 1969. Physical factors influencing fish populations in pools of a trout stream. Trans. Am. Fish. Soc. 98(1):14-19.

Liang, T. T., and E. P. Lichtenstein. 1972. The effect of light, temperature, and pH on the degradation of azinphosmethyl. J. Econ. Entomol. 65(12):315-321.

Lichtenstein, E. P., K. R. Schulz, R. F. Skrenteny, and Y. Tsukano. 1966. Toxicity and fate of insecticide residues in water. Arch. Environ. Health 12:199-212.

Liebmann, H. 1960. Handbuch der Frischwasser- und Abwasserbiologie - 2. Munich, Federal Republic of Germany.

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

Lipschuetz, M., and A. L. Cooper. 1961. Toxicity of 2-secondary-butyl-4-6-dinitrophenol to blacknose dace and rainbow trout. N.Y. Fish Game J. 8(2):110-121.

Livingston, Robert J. 1977. Review of current literature concerning the acute and chronic effects of pesticides on aquatic organisms. Critical Rev. in Environ. Control 7(4):325-351. CRC Press, Cleveland, Ohio.

Lloyd, R., and L. D. Orr. 1969. The diuretic response by rainbow trout to sublethal concentrations of ammonia. Water Res. 3:335.

Loeffler, W. W., Jr., G. W. Trimberger, F. H. Fox, R. L. Ridgeway, D. J. Lisk, and G. G. Gyrisco. 1966. Extent of residues in milk resulting from use of Guthion-treated forage. J. Agric. Food Chem. 14:46-47.

Lorz, Harold W., Susan W. Glenn, Ronald H. Williams, Clair M. Kunkel, Logan A. Norris, and Bobby R. Loper. 1979. Effects of selected herbicides on smolting of coho salmon. U.S. Environ. Prot. Agency Ecol. Res. Ser. EPA 600/3-79-071. 103 p. Corvallis, Oreg.

Lynn, G. E. 1965. A review of toxicological information on Tordon herbicides. Down to Earth 20:6-8.

McCorkle, Fred M., Janice E. Chambers, and James D. Yarbrough. 1977. Acute toxicities of selected herbicides to fingerling channel catfish, Ictalurus punctatus. Bull. Environ. Contam. Toxicol. 18(3) :267-270.

Macek, K. J. 1968. Growth and resistance to stress in brook trout fed sublethal levels of DDT. J. Fish. Res. Board Can. 25(11):2443-2451.

Macek, Kenneth J., Kenneth S. Buxton, Scott Sauter, Sarah Gnilka, and Jerry W. Dean. 1976. Chronic toxicity of atrazine to selected aquatic invertebrates and fishes. U.S. Environ. Prot. Agency Ecol. Res. Ser. EPA-600/3-76-047. 50 p. Duluth, Minn.

Macek, Kenneth J., and William A. McAllister. 1970. Insecticide susceptibility of some common fish family representatives. Trans. Am. Fish. Soc. 99(1):20-27.

McIntire, C. David. 1966. Some factors affecting respiration of periphyton communities in lotic environments. Ecology 47(6):918-930.

McIntire, C. David, and Jonathan A. Colby. 1978. A hierarchical model of lotic ecosystems. Ecol. Monogr. 48: 167-190.

McIntire, C. David, and Harry K. Phinney. 1965. Laboratory studies of periphyton production and community metabolism in lotic environments. Ecol. Monogr. 35:237-258.

McKee, J. E., and H. W. Wolf. 1971. Water quality criteria. Calif. State Water Resour. Control Board Publ. 3–A. Sacramento, Calif.

McLeay, D. J., and M. R. Gordon. 1980. Toxicity studies with the brush–control herbicide "Krenite" and salmonid fish. B.C. Res. Proj. Rep. 1–01–305. 42 p. Vancouver, B.C.

Maier–Bode, Hans. 1972. Behavior of herbicides in water, muck and fish after application in fish ponds. Schriftenr. ver Wassen-Boden-Lufthyg. (Berlin–Dahlem) 37:67-75.

Marriage, P. B., W. J. Saidak, and F. G. Von Stryuk. 1975. Residues of atrazine, simazine, linuron, and diuron after repeated annual applications in a peach orchard. Weed Res. 15:373-379.

Matida, Yoshihiro, Yoshihisa Furuta, Hiroshi Kumada, Hikaru Tanaka, Motoyoshi Yokote, and Sekio Kimura. 1975. Effects of some herbicides applied in the forest to the freshwater fishes and other aquatic organisms. 1. Survey on the effects of aerially applied sodium chlorate and a mixture of 2,4-D and 2,4,5-T on the stream community. Bull. Freshwater Fish. Res. Lab. (Tokyo) 25(1):41-53.

Matida, Yoshihiro, Sekio Kimura, Hikaru Tanaka, and Motoyoshi Yokote. 1976. Effects of some herbicides applied in the forest to the freshwater fishes and other aquatic organisms. 3. Experiments on the assessment of acute toxicity of herbicides to aquatic organisms. Bull. Freshwater Fish. Res. Lab. (Tokyo) 26(2):79–84.

Meehan, William R., Logan A. Norris, and Howard S. Sears. 1974. Toxicity of various formulations of 2,4-D to salmonids. J. Fish. Res. Board Can. 31(4):480-485.

Melnikov, N. N. 1971. Chemistry of pesticides. Residue Rev. 36:1–480.

Meyer, Fred P. 1965. The experimental use of Guthion as a selective fish eradicator. Trans. Am. Fish. Soc. 94(3):203-209.

Mills, Derek H. 1969. The survival of juvenile Atlantic salmon and brown trout in some Scottish streams. In Symposium on trout and salmon in streams, p. 217–228. T. G. Northcote, ed. H. R. MacMillan Lectures in Fisheries, Univ. B.C., Vancouver.

Moore, D. G. 1970. Forest fertilization and water quality in the Pacific Northwest. Am. Soc. Agron. Abstr. 1970:160-161.

Moore, D. G. 1975a. Effects of forest fertilization with urea on stream water quality--Quilcene Ranger District, Washington. USDA For. Serv. Res. Note PNW–241. 9 p. Pac. Northwest For. and Range Exp. Stn., Portland, Oreg.

Moore, Duane G. 1971. Fertilization and water quality. In Proceedings, 1971 Annual Meeting, West. For. and Conserv. Assoc., p. 28–31. Portland, Oreg.

Moore, Duane G. 19758, Impact of forest fertilization on water quality in the Douglas–fir region--a summary of monitoring studies. In Forestry issues in urban America. Proceedings - 1974 Natl. Conv. Soc. Am. For., p. 209–219. Soc. Am. For., Washington, D.C.

Moore, Duane G., and Logan A. Norris. 1974. Soil processes and introduced chemicals. In Environmental effects of forest residues management in the Pacific Northwest: a state-of-knowledge compendium. USDA For. Serv. Gen. Tech. Rep. PNW–24, p. C-1 to C–27. Pac. Northwest For. and Range Exp. Stn., Portland, Oreg.

Morgan, L. S., and R. W. Kremer. 1952. Some observations on streams from the DDT spraying for the hemlock looper. Penn. For. and Water 4(6):138-140.

Moshier, Loren J., and Donald Penner. 1978. Factors influencing microbial degradation of $^{14}$C-glyphosate to $^{14}CO_2$ in soil. Weed Sci. 26(6):686-691.

Mount, Donald I., and Charles E. Stephan. 1967. A method for establishing acceptable toxicant limits for fish - malathion and the butoxyethanol ester of 2,4-D. Trans. Am. Fish. Soc. 96(2):185-193.

Mount, Michael E., and Frederick W. Oehme. 1981. Carbaryl: a literature review. Residue Rev. 80:2-64.

Mrazik, Brian R., Donald L. Mader, and William P. MacConnell. 1980. Integrated watershed management: an alternative for the Northeast. Res. Bull. 664. 50 p. Mass. Agric. Exp. Stn., Univ. Mass., Amherst.

Mulla, Mir S., and Lal S. Mian. 1981. Biological and environmental impacts of the insecticides malathion and parathion on nontarget biota in aquatic ecosystems. Residue Rev. 78:101-135.

Murphy, Michael Lewis. 1979. Predator assemblages in old-growth and logged sections of small Cascade streams. M.S. thesis. 72 p. Oreg. State Univ., Corvallis.

National. Academy of Sciences-National Academy of Engineering. 1973. Water quality criteria. 1972. A report of the committee on water quality criteria. Section 3. Freshwater aquatic life and wildlife. U.S. Environ. Prot. Agency Ecol. Res. Ser. EPA-R3-73-033. 594 p. Washington, D.C.

National Research Council of Canada. 1974. Picloram: the effects of its use as a herbicide on environmental quality. NRCC 13684. 128 p. Natl. Res. Counc. Can., Ottawa.

National Research Council of Canada. 1978. Phenoxy herbicides - their effects on environmental quality, with accompanying scientific criteria for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). NRCC 16075. 440 p. Natl. Res. Counc. Can., Ottawa.

Neave, Ferris. 1947. Natural propagation of chum salmon in a coastal stream. Prog. Rep. 70, p. 20-21. Pac. Coast Stn. Fish Res. Board Can., Nanaimo, B.C.

Nesbitt, Harry J., and John R. Watson. 1980. Degradation of the herbicide 2,4-D in river water. 1. Description of the study area and survey of rate determining factors. Water Res. 14(12):1683-1688.

Newbold, C. 1975. Herbicides in aquatic systems. Biol. Conserv. 7:97-118.

Newton, Michael. 1981. Herbicides in forestry. In Oregon weed control handbook, p. 78-93. R. E. Whitesides, ed. Oreg. State Univ., Corvallis.

Newton, Michael, and Joel A. Norgren. 1977. Silvicultural chemicals and protection of water quality. U.S. Environ. Prot. Agency EPA 910/9-77-036. 232 p. Seattle, Wash.

Norris, L. A. 1967. Chemical brush control and herbicide residues in the forest environment. In Proc., Herbic. and Veg. Manage. Symp., p. 103-123. Oreg. State Univ., Corvallis.

Norris, L. A. 1969. Herbicide runoff from forest lands sprayed in summer. In Research progress report, p. 24-26. West. Soc. Weed Sci.

Norris, L. A. 1971a. Chemical brush control--assessing the hazard. J. For. 69(10):715-720.

Norris, L. A., M. L. Montgomery, and Fred Gross. 1976. The behavior of picloram and 2,4-D in soil on western powerline rights-of-way. In Abstracts, 1976 Meeting of the Weed Sci. Soc. Am., p. 9-10. Weed Sci. Soc. Am., Champaign, Ill.

Norris, L. A., M. L. Montgomery, C. A. Troendle, and J. M. Kochenderfer. 1978. 2,4,5-T persistence in a West Virginia watershed. In Abstracts, 1978 Meeting of the Weed Sci. Soc. Am., p. 38-39. Weed Sci. Soc. Am., Champaign, Ill.

Norris, L. A., M. L. Montgomery, L. E. Warren, and W. D. Mosher. 1982. Brush control with herbicides on hill pasture sites in southern Oregon. J. Range Manage. 35(1):75-80.

Norris, L. A., and D. G. Moore. 1971. The entry and fate of forest chemicals in streams. In Forest land uses and stream environment, p. 138-158. J. T. Krygier and J. D. Hall, eds. Oreg. State Univ., Corvallis.

Norris, Logan A. 1966. Degradation of 2,4-D and 2,4,5-T in forest litter. J. For. 64(7):475-476.

Norris, Logan A. 1968. Stream contamination by herbicides after fall rains on forest land. In Research progress report, p. 33-34. West. Soc. Weed Sci.

Norris, Logan A. 1970a. Degradation of herbicides in the forest floor. In Tree growth and forest soils, p. 397-411. C. T. Youngberg and C. B. Davey, eds. Oreg. State Univ. Press, Corvallis.

Norris, Logan A. 1970b. The kinetics of adsorption and desorption of 2,4-D, 2,4,5-T, picloram, and amitrole on forest floor material. In Res. Prog. Rep., p. 103-105. West. Soc. Weed Sci.

Norris, Logan A. 1971b. The behavior of chemicals in the forest. In Pesticides, pest control and safety on forest and range lands, p. 90-106. J. Witt and J. Capizzi, eds. Oreg. State Univ., Corvallis.

Norris, Logan A. 1978. Toxic materials in forest streams. In Toxic materials in the aquatic environment, p. 43-68. Water Resour. Res. Inst., Oreg. State Univ., Corvallis.

Norris, Logan A. 1981. The movement, persistence and fate of the phenoxy herbicides and TCDD in the forest. Residue Rev. 80:65-135.

Norris, Logan A., and David Greiner. 1967. The degradation of 2,4-D in forest litter. Bull. Environ. Contam. Toxicol. 2(2):65-74.

Norris, Logan A., and Marvin L. Montgomery. 1975. Dicamba residues in streams after forest spraying. Bull. Environ. Contam. Toxicol. 13(1):1-8.

Norris, Logan A., and Duane G. Moore. 1976. Forests and rangelands as sources of chemical pollutants. In Non-point sources of water pollution, p. 16-35. Water Resour. Res. Inst., Oreg. State Univ., Corvallis.

Nuttal, P. M. 1972. The effects of sand deposition upon the macroinvertebrate fauna of the River Camel, Cornwall. Freshwater Biol. 2:181-186.

Ogilvie, D. M., and J. M. Anderson. 1965. Effect of DDT on temperature selection by young Atlantic salmon, Salmo salar. J. Fish. Res. Board Can. 22(2):503-512.

Paris, Doris F., David L. Lewis, John T. Barnett, Jr., and George L. Baughman. 1975. Microbial degradation and accumulation in aquatic systems. U.S. Environ. Prot. Agency EPA-660/3-75-007. 45 p. Corvallis, Oreg.

Parrish, Patrick R., Elizabeth E. Dyar, Mark A. Lindberg, Chiara M. Shanika, and Joanna M. Enos. 1977. Chronic toxicity of methoxychlor, malathion and carbofuran to sheepshead minnows (Cyprinodon variegatus). U.S. Environ. Prot. Agency. Ecol. Res. Ser. EPA 600/3-77-059. 37 p. Gulf Breeze, Fla.

Phillips, Robert W. 1964. The influence of gravel size on survival to emergence of coho salmon and steelhead trout. In Proc. of the 15th Northwest Fish Culture Conf., p. 90. Oreg. State Univ., Corvallis.

Pickering, Q. H., C. Henderson, and A. E. Lemke. 1962. The toxicity of organic phosphorus insecticides to different species of warmwater fishes. Trans. Am. Fish. Soc. 91(2):175-184.

Plumb, T. R., L. A. Norris, and M. L. Montgomery. 1977. Persistence of 2,4-D and 2,4,5-T in chaparral soil and vegetation. Bull. Environ. Contam. Toxicol. 17(1):1-8.

Rabeni, Charles F., and Jon G. Stanley. 1979. Operational spraying of acephate to suppress spruce budworm has minor effects on stream fishes and invertebrates. Bull. Environ. Contam. Toxicol. 23(3):327-334.

Ray, Bibhas. 1975. Fate of organic arsenicals in soil and plants. Int. Pest. Control 17(1):9-14.

Reiser, D. W., and T. C. Bjornn. 1979. Influence of forest and rangeland management on anadromous fish habitat in western North America: 1. Habitat requirements of anadromous salmonids. W. R. Meehan, tech. ed. USDA For. Serv. Gen. Tech. Rep. PNW-96. 54 p. Pac. Northwest For. and Range Exp. Stn., Portland, Oreg.

Rice, R. M., J. S. Rothacher, and W. F. Megahan. 1972. Erosional consequences of timber harvesting: an appraisal. In Proceedings, National Symposium on Watersheds in Transition, p. 321-329. Am. Water Resour. Assoc., Urbana, Ill.

Roberts, J. E. 1962. Persistence of insecticides in soils and their effects on cotton in Georgia. Rev. Appl. Entomol. 50(A):567 (Part 11).

Roberts, Richard B. 1976. Pesticide spray application, behavior and assessment: workshop proceedings. USDA For. Serv. Gen. Tech. Rep. PSW-15. 68 p. Pac. Southwest For. and Range Exp. Stn., Berkeley, Calif.

Robinson, Edward L. 1975. Arsenic in soil with five annual applications of MSMA. Weed Sci. 23(5):341-343.

Rodgers, Charles A., and David L. Stallings. 1972. Dynamics of an ester of 2,4-D in organs of three fish species. Weed Sci. 20(1):101-105.

Rothacher, Jack, and William Lopushinsky. 1974. Soil stability and water yield and quality. In Environmental effects of forest residues management in the Pacific Northwest: a state-of-knowledge compendium. USDA For. Serv. Gen. Tech. Rep. PNW-24, p. D-1 to D-19. Pac. Northwest For. and Range Exp. Stn., Portland, Oreg.

Rueppel, Melvin L., Blanche B. Brightwell, Jacob Schaefer, and John T. Marvel. 1977. Metabolism and degradation of glyphosate in soil and water. J. Agric. Food Chem. 25(3):517-528.

Russell-Hunter, W. D. 1970. Aquatic productivity: an introduction to some basic aspects of biological oceanography and limnology. 306 p. The MacMillan Company, London.

Sacher, Reuven M. 1978. Safety of Roundup® in the aquatic environment. In Proc. European Weed Research Council, 5th Symposium on Aquatic Weeds, p. 315–322. Amsterdam, Netherlands.

Sanborn, James R. 1974. The fate of select pesticides in the aquatic environment. U.S. Environ. Prot. Agency Ecol. Res. Ser. EPA 660/3-74–025. 83 p. Corvallis, Oreg.

Sanders, Herman O. 1969. Toxicity of pesticides to the crustacean Gammarus lacustris. USDI Bur. Sport Fish. and Wildl. Tech. Pap. 25, p. 3–18. Washington, D.C.

Sanders, Herman O. 1970. Toxicities of some herbicides to six species of freshwater crustaceans. J. Water Pollut. Control Fed. 42(8):1544-1550.

Sanders, Herman O,, and Oliver B. Cope. 1968. The relative toxicities of several pesticides to naiads of three species of stoneflies. Limnol. and Oceanogr. 13(1):112-126.

Sanders, W. M., III. 1979. Exposure assessment: a key issue in aquatic toxicology. In Aquatic toxicology, ASTM STP 667, p. 271–283. L. L. Marking and R. A. Kimerle, eds. Am. Soc. Test. and Mater., Philadelphia, Penn.

Schultz, Donald P. 1973. Dynamics of a salt of 2,4-dichlorophenoxyacetic acid in fish, water and hydrosol. J. Agric. Food Chem. 21(2):186-192.

Schultz, Donald P., and Paul D. Harman. 1974. A review of the literature on the use of 2,4-D in fisheries. USDI Fish and Wildl. Serv. LR74–18. 94 p. Warm Springs, Ga.

Schultz, Donald P., and Eugene W. Whitney. 1974. Monitoring 2,4-D residues at Loxahatchee National Wildlife Refuge. Pestic. Monit. J. 7(3/4):146-152.

Sedell, James R., Frank J. Triska, and Nancy S. Triska. 1975. The processing of conifer and hardwood leaves in two coniferous forest streams: 1. Weight loss and associated invertebrates. Verh. Int. Ver. Limnol. 19:1617–1627.

Sergeant, M., D. Blazek, J. H. Elder, C. A. Lembi, and D. J. Moore. 1970. The toxicity of 2,4-D and picloram herbicides to fish. Proc. Indiana Acad. Sci. 80:114-123.

Sigmon, Catherine. 1979. Oxygen consumption in Lepomis machrochirus exposed to 2,4-D or 2,4,5-T. Bull. Environ. Contam. Toxicol. 21:826-830.

Smith, A. E., R. Grover, G. S. Edmond, and H. C. Korven. 1975. Persistence and movement of atrazine, bromacil, monuron and simazine in intermittently-filled irrigation ditches. Can. J. Plant Sci. 55:809-816.

Smith, C. E., and R. G. Piper. 1975. Lesions associated with chronic exposure to ammonia. In The pathology of fishes, p. 497–514. W. E. Ribelin and G. Migaki, eds. Univ. of Wis. Press, Madison.

Smith, Gordon E., and Billy G. Isom. 1967. Investigation of the effects of large-scale application of 2,4-D on aquatic fauna and water quality. Pestic. Monit. J. 1(3):16–21.

Sollins, P., K. Cromack, Jr., F. M. McCorison, R. H. Waring, and R. D. Harr. 1981. Changes in nitrogen cycling at an old-growth Douglas-fir site after disturbance. J. Environ. Qual. 10(1):37-42.

Solomon, Howard M., and Judith S. Weis. 1979. Abnormal circulatory development in medaka caused by the insecticides carbaryl, malathion, and parathion, Teratology 19:51–62.

Solomon, Howard Mark. 1978. The tetragenic effects of the insecticides DDT, carbaryl, malathion, and parathion on developing medaka eggs (Oryzias latipes). Ph. D. Diss. Abstr. 39:2176–2177B.

Spehar, Robert L., James T. Fiandt, Richard L. Anderson, and David L. DeFoe. 1980. Comparative toxicity of arsenic compounds and their accumulation in invertebrates and fish. Arch. Environ. Contam. Toxicol. 9:53-63.

Sprague, J. B. 1971. Measurement of pollutant toxicity to fish - 3. Sublethal effects and "safe" concentrations. Water Res. 5(6):245–266.

Sprankle, Paul, W. F. Meggitt, and Donald Penner. 1975a. Rapid inactivation of glyphosate in the soil. Weed Sci. 23(3):224-228.

Sprankle, Paul, W. F. Meggitt, and Donald Penner. 1975b. Adsorption, mobility and microbial degradation of glyphosate in the soil. Weed Sci. 23(3):229–234.

Stanley, Jon G., and Joan G. Trial. 1980. Disappearance constants of carbaryl from streams contaminated by forest spraying. Bull. Environ. Contam. Toxicol. 25(5):771-776.

Stavinoha, W. B., J. A. Rieger, Jr., L. C. Ryan, and P. W. Smith. 1966. Effects of chronic poisoning by an organophosphorous cholinesterase inhibitor on acetylcholine and norepinephrine content of the brain. In Organic pesticides in the environment. Advances in Chemistry vol. 60, p. 79–88. R. F. Gould, ed. Am. Chem. Soc., Washington, D.C.

Stay, F. S., A. Katko, K. W. Malueg, M. R. Crouse, S. E. Dominguez, and R. E. Austin. 1979. Effects of forest fertilization with urea on major biological components of small Cascade streams, Oregon. U.S. Environ. Prot. Agency, Res. and Dev., EPA 600/3-79-099. 60 p. Corvallis, Oreg.

Stewart, D. K. R., and Sonia O. Gaul. 1977. Persistence of 2,4-D, 2,4,5-T and dicamba in a dykeland soil. Bull. Environ. Contam. Toxicol. 18(2):210-218.

Stewart, Nelson E., Raymond E. Millemann, and Wilbur P. Breese. 1967. Acute toxicity of the insecticide Sevin and its hydrolytic product 1-napthol to some marine organisms. Trans. Am. Fish. Soc. 96(1):25-30.

Stockner, John G., and K. R. S. Shortreed. 1976. Autotrophic production in Carnation Creek, a coastal rainforest stream on Vancouver Island, British Columbia. J. Fish. Res. Board Can. 33(7):1553-1563.

Storey, H. C. 1965. Watershed management research. In Who's responsible for water resources research?, p. 55–64. Water Resour. Res. Inst., Oreg. State Univ., Corvallis.

Streit, Bruno. 1979. Uptake, accumulation, and release of organic pesticides by benthic invertebrates. 2. Reversible accumulation of lindane, paraquat, and 2,4-D from aqueous solution by invertebrates and detritus. Arch. Hydrobiol./Suppl. (Stuttgart) 55(3/4):349–372.

Suffling, Roger, David 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.

Swanston, Douglas N. 1980. Influence of forest and rangeland management on anadromous fish habitat in western North America: 2. Impacts of natural events. W. R. Meehan, tech. ed. USDA For. Serv. Gen. Tech. Rep. PNW-104. 27 p. Pac. Northwest For. and Range Exp. Stn., Portland, Oreg.

Swanston, Douglas N., and Frederick J. Swanson. 1976. Timber harvesting, mass erosion, and steepland forest geomorphology in the Pacific Northwest. In Geomorphology and Engineering, p. 299-321. D. R. Coates, ed. Dowden, Hutchinson & Ross, Stroudsburg, Penn.

Swingle, H. S. 1947. Experiments on pond fertilization. Agric. Exp. Stn. Bull. 264. Ala. Polytechnic Inst., Auburn, Ala.

Symons, P. E. K. 1973. Behavior of young Atlantic salmon (Salmo salar) exposed to or force-fed fenitrothion, an organophosphate insecticide. J. Fish. Res. Board Can. 30(5):651-655.

Symons, P. E. K. 1977. Dispersal and toxicology of the insecticide fenitrothion; predicting hazards of forest spraying. Residue Rev. 68:1-36.

Szeto, S. Y., H. R. MacCarthy, P. C. Oloffs, and R. F. Shepherd. 1978. Residues in Douglas-fir needles and forest litter following an aerial application of acephate (Orthene®). J. Environ. Sci. Health B13(2):87-103.

Tarrant, Robert F., and James M. Trappe. 1971. The role of Alnus in improving the forest environment. In Plant and Soil, Spec. Vol., 1971, p. 335-348. Martinus Niejhoff, The Hague, Netherlands.

Tarzwell, C. M. 1966. Water quality requirements for aquatic life. In Proc. Nat. Symp. on Water Quality. Ann Arbor, Mich. Univ. Mich. School of Public Health, Continued Educ. Ser. 161:185-197.

Tarzwell, Clarence M. 1958. Toxicity of some organic insecticides to fishes. In Proc. 12th Annual Conf., p. 233. Southeast Assoc. Game and Fish Comm., Columbia, S.C.

Thut, Rudolph N., and Eugene P. Haydu. 1971. Effects of forest chemicals on aquatic life in surface waters. In Forest land uses and stream environment, p. 159-171. J. T. Krygier and J. D. Hall, eds. Oreg. State Univ., Corvallis.

Torstensson, N. T. L., and A. Aamesepp. 1977. Detoxification of glyphosate in soil. Weed Res. 17:209-212.

Tracy, Harry B., John Bernhardt, Douglas Freeman, and Bill Purvis. 1977. A report on aquatic monitoring of the 1976 spruce budworm control project in Washington State. State Wash., Dep. Ecol., Tech. Rep. DOE 77-3. 50 p. Olympia, Wash.

Tu, C. M., and J. R. W. Miles. 1976. Interactions between insecticides and soil microbes. Residue Rev. 64:17-65.

Union Carbide. 1968. Technical information on Sevin carbaryl insecticide. Union Carbide Corp. ICG-0449A Bookl. 56 p. Salinas, Calif.

Upchurch, R. P., and D. D. Mason. 1962. The influence of soil organic matter on the phytoxicity of herbicides. Weeds 10:9-14.

U. S. Department of Agriculture. 1976. Aerial application of agricultural chemicals. USDA Agric. Handb. 287. 25 p. Washington, D.C.

U. S. Department of Agriculture, Animal and Plant Health Inspection Service. 1980. Rangeland grasshopper cooperative management program. USDA Final Environ. Impact Statement. 36 p. USDA, APHIS, Washington, D.C.

U. S. Department of Agriculture, Forest Service. 1976. Cooperative western spruce budworm pest management plan. USDA Environ. Statement. 143 p. USDA For. Serv., Pac. Northwest Reg., Portland, Oreg.

U. S. Environmental Protection Agency. 1975. Substitute chemical program. Initial scientific review of MSMA/DSMA. U.S. Environ. Prot. Agency, EPA/540/1-75-020. 124 p. Washington, D.C.

U. S. Environmental Protection Agency. 1976. Quality criteria for water. 256 p. U.S. Environ. Prot. Agency, Washington, D.C.

U.S. Environmental Protection Agency. 1980. Appendix B - Guidelines for deriving water quality criteria for the protection of aquatic life and its uses. Fed. Regist. 45(231):79341-79347.

Van Meter, Wayne P., and Charles E. Hardy. 1975. Predicting effects on fish of fire retardant in streams. USDA For. Serv. Res. Pap. INT-166. 16 p. Intermt. For. and Range Exp. Stn., Ogden, Utah.

Von Endt, D. W., P. C. Kearney, and D. D. Kaufman. 1968. Degradation of monosodium methanearsonic acid by soil microorganisms. J. Agric. Food Chem. 16(1):17-20.

Walker, Charles R. 1964a. Toxicological effects of herbicides on the fish environment. Part 1. Water Sewage Works 111:113-116.

Walker, Charles R. 1964b. Simazine and other s-triazine compounds as aquatic herbicides in fish habitats. Weeds 12(2):134-139.

Walker, William M. 1978. Insecticide persistence in natural sea water as affected by salinity, temperature, and sterility. U.S. Environ. Prot. Agency Ecol. Res. Ser. EPA-600/3-78-044. 25 p. Ocean Springs, Miss.

Warner, Kendall, and Owen C. Fenderson. 1962. Effects of DDT spraying for forest insects on Maine trout streams. J. Wildl. Manage. 26(1):86-93.

Warner, R. E., K. K. Peterson, and L. Borgman. 1966. Behavioral pathology in fish: a quantitative study of sublethal pesticide toxication. Pesticides in the environment and their effects on wildlife: Proceedings of an Advanced Study Institute, sponsored by the North Atlantic Treaty Organization, Monks Wood Experimental Station, England, July 1-14, 1965. N. W. Moore, ed. In 3. Appl. Ecol., Suppl. to vol. 3, p. 223-247. Blackwell Scientific Publications, Oxford, England.

Wauchope, R. D. 1975. Fixation of arsenical herbicides, phosphate, and arsenate in alluvial soils. J. Environ. Qual. 4(3):355-358.

Webb, F. E., and D. R. MacDonald. 1958. Studies of aerial spraying against the spruce budworm in New Brunswick. 10. Surveys of stream-bottom fauna in some sprayed and unsprayed streams, 1955-57. Can. Dep. Agric. Interim Rep. 1957-3. 13 p. For. Biol. Lab., Fredericton, N.B.

Weber, Walter J., Jr., and Egon Matijevic. 1968. Adsorption from aqueous solutions. Advances in Chemistry Series vol. 79. 212 p. Am. Chem. Soc., Washington D.C.

Weed Science Society of America. 1979. Herbicide handbook! 4th Ed. 479 p. Weed Sci. Soc. Am., Champaign, Ill.

Weidner, C. W. 1974. Degradation in groundwater and mobility of herbicides. M.S. thesis. 77 p. Nebraska Univ., Lincoln.

Weiss, C. M., and J. H. Gakstatter. 1964. Detection of pesticides in water by biochemical assay. J. Water Pollut. Control Fed. 36:240-253.

Westin, Deborah T. 1974. Nitrate and nitrate toxicity to salmonid fishes. Prog. Fish-Cult. 36(2):86.

Whitesides, Ralph E. 1981. Oregon weed control handbook. 162 p. Oreg. State Univ., Corvallis.

Wilbur, R. L., and E. W. Whitney. 1973. Toxicity of the herbicide Kuron (silvex) to bluegill eggs and fry. Trans. Am. Fish. Soc. 102(3) :630-633.

Wildish, D. J., W. G. Carson, T. Cunningham, and N. J. Lister. 1971, Toxicological effects of some organophosphate insecticides to Atlantic salmon. Fish. Res. Board Can. MS Rep. No. 1157. 22 p. St. Andrews, New Brunswick.

Willcox, H., III. 1972. Environmental impact study of aerially applied Sevin-4-oil on a forest and aquatic ecosystem. LOTEL Rep. 55 p. Lake Ont. Environ. Lab., Oswego, N.Y.

Williams, A. K., and C. R. Sova. 1966. Acetyl-cholinesterase levels in brains of fishes from polluted waters. Bull. Environ. Contam. Toxicol. 1:198-204.

Woodward, D. F. 1976. Toxicity of the herbicides dinoseb and picloram to cutthroat trout (Salmo clarki) and lake trout (Salvelinus namaycush). J. Fish. Res. Board Can. 33(8):1671-1676.

Woodward, D. F., and W. L. Mauck. 1980. Toxicity of five forest insecticides to cutthroat trout and two species of aquatic invertebrates. Bull. Environ. Contam. Toxicol. 25(6):846-853.

Woodward, D. F., and F. L. Mayer, Jr. 1978. Toxicity of three herbicides (butyl, isooctyl and propylene glycol butyl ether esters of 2,4-D) to cutthroat trout and lake trout. USDI Fish and Wildl. Serv. Tech. Pap. 97, p. 1-6. USDI Fish and Wildl. Serv., Washington, D.C.

Woolson, E. A., A. R. Isensee, and P. C. Kearney. 1976. Distribution and isolation of radioactivity from $^{74}$As-arsenate and $^{14}$C-methanearsonic acid in an aquatic model ecosystem. Pestic. Biochem. and Physiol. 6:261-269.

# APPENDIX

Table 18--Common, chemical, and trade names of chemicals referred to in text and tables

| Common name | Chemical name | Trade name used in text |
|---|---|---|
| Fertilizer: | Urea | None |
| Fire retardants: | None | Fire-Trol 100<br>Fire-Trol 931L<br>Fire-Trol 934L<br>Phos-Chek<br>Phos-Chek XAR<br>Phos-Chek 202R<br>Phos-Chek 259R |
| Herbicides: | | |
| 2,4-D | 2,4-dichlorophenoxyacetic acid (and various esters and salts) | None |
| 2,4,5-T | 2,4,5-trichlorophenoxyacetic acid | None |
| Amitrole | 3-amino-1,2,4-triazole | Amitrole-T |
| Atrazine | 2-chloro-4-ethylamino-6-isopropyl-amino-?-triazine | None |
| Dicamba | 3,6-dichloro-o-anisic acid | None |
| Dinoseb | 2-sec-butyl-4,6-dinitrophenol | None |
| DSMA | Disodium methanearsonate | None |
| Fosamine ammonium | Ammonium ethy1carbamoy1-phosphonate | Krenite |
| Glyphosate | N-phosphonomethylglycine | Roundup |
| MSMA | Monosodium methanearsonic acid | None |
| Picloram | 4-amino-3,5,6-trichloropicolinic acid (and various esters and salts) | Tordon 22K<br>Tordon 101 (also contains 2,4-D) |
| SDMA | Sodium dimethyl arsonate | None |
| Silvex | 2-(2,4,5-trichlorophenoxy)-propionic acid | None |
| isecticides: | | |
| Acephate | 0,S-dimethy1 acety1phosphor-amidothioate | Orthene |
| Azinphos-methyl | 0,0-dimethyl-S-[(4-oxo-1,2,3-benzotriazine-3-(4H)-yl)methyl] phosphorodithioate | Guthion |
| Carbary1 | 1-naphthyl-N-methylcarbamate | Sevin<br>Sevin-4-Oil |
| Carbofuran | 2,3-dihydro-2,2-dimethyl-7-benzofuranyl methylcarbamate | Furadan |
| Chlordecone | Decach1oro-octahydro-1,3,4-metheno-2H-cyclobuta(cd)pentalene-2-one | Kepone |
| DDT | Dichloro diphenyl trichloroethane | None |
| Malathion | 0,0-dimethy1-S-(1,2-dicarbethyoxyethy1)-phosphorodithioate | None |
| Methoxychlor | 2,2-bis(p-methoxyphenyl)-1,1,1-trichloroethane | None |

Table 19--Common and scientific names of fishes referred to in text and tables

| Common name | Scientific name |
|---|---|
| | FAMILY Catostomidae |
| Lake chubsucker | Erimyzon sucetta (Lacèpéde) |
| | FAMILY Centrarchidae |
| Bluegi11 | Lepomis macrochirus Rafinesque |
| Green sunfish | Lepomis cyanellus Rafinesque |
| Largemouth bass | Micropterus salmoides (Lacèpéde) |
| Pumpkinseed | Lepomis gibbosus (Linnaeus) |
| Smallmouth bass | Micropterus dolomieui Lacèpéde |
| | FAMILY Cottidae |
| Slimy sculpin | Cottus cognatus Richardson |
| | FAMILY Cyprinidae |
| Blacknose dace | Rhinichthys atratulus (Hermann) |
| Carp | Cyprinus carpio Linnaeus |
| Fathead minnow | Pimephales promelas Rafinesque |
| Goldfish | Carassius auratus (Linnaeus) |
| Lake emerald shiner | Notropis atherinoides Rafinesque |
| Redside shiner | Richardsonius balteatus (Richardson) |
| Stoneroller | Campostoma anomalum (Rafinesque) |
| | FAMILY Cyprinodontidae |
| Longnose killifish | Fundulus similis (Baird and Girard) |
| Sheepshead minnow | Cyprinodon variegatus Lacèpéde |
| | FAMILY Ictaluridae |
| Black bullhead | Ictalurus melas (Rafinesque) |
| Channel catfish | Ictalurus punctatus (Rafinesque) |
| | FAMILY Oryziidae |
| Medaka | Oryzias latipes (Temminck and Schlegel) |
| | FAMILY Penidae |
| Yellow penh | Perca flavescens (Mitchill) |
| Walleye | Stizostedion vitreum (Mitchill) |
| | FAMILY Poeciliidae |
| Guppy | Poecilia reticulata Peters |
| Mosquitofish, gambusia | Gambusia affinis (Baird and Girard) |
| | FAMILY Salmonidae |
| Atlantic salmon | Salmo salar Linnaeus |
| Brook trout | Salvelinus fontinalis (Mitchill) |
| Brown trout | Salmo trutta Linnaeus |
| Cherry salmon | Oncorhynchus masou (Brevort) |
| Chinook salmon | Oncorhynchus tshawytscha (Walbaum) |
| Chum salmon | Oncorhynchus keta (Walbaum) |
| Coho salmon | Salmo clarki Richardson |
| Cutthroat trout | Salmo clarki henshawi Gill and Jordan |
| Lahontan cutthroat trout | Salvelinus namaycush (Walbaum) |
| Lake trout | Oncorhynchus gorbuscha (Walbaum) |
| Pink salmon | Salmo gairdneri Richardson |
| Rainbow (steelhead) trout | Oncorhynchus nerka (Walbaum) |
| Sockeye salmon | Prosopium sp. |
| Whitefish | Coregonus sp. |
| Whitefish | |
| | FAMILY Umbridae |
| Eastern mudminnow | Umbra pygmaea (DeKay) |

Table 20--Common and scientific names of invertebrates referred to in text and tables

| Common name | Scientific name |
|---|---|
| | ORDER Amphipoda |
| Scuds, amphipods | Gammarus fasciatus Say |
| | Gammarus lacustris Sars |
| | Gamnarus pseudolimnaeus Bousfield |
| | ORDER Cladocera |
| Daphnids, water fleas | Daphnia magna Straus |
| | Daphnia pulex Leydig |
| | ORDER Decapoda |
| Crayfishes | Orconectes nais (Faxon) |
| | Procambarus clarki (Girard) |
| Glass shrimp | Palaemonetes kadiakensis Rathbun |
| | ORDER Diptera |
| Crane fly | Tipula sp. |
| Phantom midge | Chaoborus sp. |
| Midges | Chironomus tenans (Fabricius) |
| | Chironomus plumosus (Linnaeus) |
| | ORDER Ephemeroptera |
| Mayflies | Hexagenia bilineata (Say) |
| | Baetis sp. |
| | ORDER Gastropoda |
| Snails | Helisoma campanulata (Say) |
| | Stagnicola emarginata (Say) |
| | ORDER Isopoda |
| Sowbugs, isopods | Asellus brevicaudus Forbes |
| | Asellus hilgendorffii |
| | ORDER Megaloptera |
| Dobsonfly | Nigronia sp. |
| | ORDER Odonata |
| Dragonfly | Macromia sp. |
| Damselfly | Ischnura venticalis (Say) |
| | ORDER Ostracoda |
| Seed shrimp | Cypridopsis vidua (Müller) |
| | ORDER Plecoptera |
| Stoneflies | Pteronarcys californica Newport |
| | Pteronarcys dorsata Say |
| | Pteronarcella badia (Hagen) |
| | Isoperla sp. |
| | Skwala sp. |
| | ORDER Trichoptera |
| Caddisflies | Hydropsyche sp. |
| | Limnephilus sp. |

The Forest Service of the U.S. Department of Agriculture is dedicated to the principle of multiple use management of the Nation's forest resources for sustained yields of wood, water, forage, wildlife, and recreation. Through forestry research, cooperation with the States and private forest owners, and management of the National Forests and National Grasslands; it strives — as directed by Congress — to provide increasingly greater service to a growing Nation.



Pacific Northwest Forest and Range
Experiment Station
809 NE Sixth Avenue
Portland, Oregon 97232