Chapter 7

Forest Chemicals

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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 habitats. 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 salmonid fishes (particularly anadromous populations) and their habitats. 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 salmonids and their habitats.

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 7.1). The common, chemical, and trade names of forest chemicals used in this chapter are listed in Table 7.2.

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 for use in agriculture, fewer than 10 have substantial use in forestry. Forestry uses account for less than 1% of the total pesticides used in the USA.

[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 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.

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TABLE 7.1.—Comparative annual use of chemicals in agriculture and forestry.

Chemical	Agriculture	Forestry
	Pesticides, 1980 (10^3 kg)[a]	
Insecticides	138,924	71[b]
Herbicides	202,030	169[b]
Fungicides	22,700	9[b]
	Fertilizers, 1978 (10^3 tonnes)[c]	
Nitrogen	9,636	55
Phosphorus	2,273	5

[a]Agricultural data 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 U.S. Forest Service, National Forest System land, from Table E1, Pesticide-Use Advisory Memorandum 284 (2150 Pesticide-Use Management and Coordination, March 12, 1981), U.S. Forest Service, Washington, D.C.
[b]U.S. Forest Service, National Forest System land only.
[c]Bengtson (1979).

Before fiscal year (FY) 1987 (fiscal years of the U.S. government extend from October 1 of the previous year to September 30 of the year designated), herbicides and insecticides accounted for more than 80% of U.S. Forest Service applications, fumigants and fungicides accounting for most of the rest (Table 7.3). More recently, however (FY 1987, 1989), fumigants and fungicides have accounted for 20% to nearly 50% of total pesticide use; most of these chemicals are used on tree nurseries. The total amount of pesticides used has varied from 137,000 kg (FY 1989) to 502,000 kg (FY 1983). The ratio of herbicide to insecticide applications has changed annually according to the needs for large-scale insect control and to court-imposed restrictions (which have been applied to herbicides since FY 1984). These figures underestimate the total use of pesticides in forestry because they do not include pesticides applied by other U.S. agencies or by state or private forest management groups.

Tables 7.4 and 7.5 give the herbicides and insecticides used on national forests and on other lands through federal assistance programs coordinated by the U.S. Forest Service. Picloram, alone or in combination with other chemicals, and 2,4-D accounted for about 70% of the herbicides applied in FY 1979–1981, but their use had declined to about 18% in 1989, probably because of a court-ordered ban on herbicides in Pacific northwestern states and of a U.S. Forest Service ban on aerial applications of herbicides nationwide. Uses of hexazinone, triclopyr, and glyphosate have increased as their registration has been granted and as experience with these chemicals has expanded. These three chemicals accounted for more than 75% of all herbicides used in FY 1987–1989.

Malathion and carbaryl accounted for nearly all the silvicultural insecticides used in FY 1979–1985, although the use of each has varied widely (Table 7.5). Since then, use of azinphos-methyl, in particular, has increased. *Bacillus thuringiensis*, a bacterial insecticide, is being used increasingly (Table 7.5) to control gypsy moth *Lymantria dispar* and western spruce budworm *Choristoneura* sp. Typical application rates of some forest chemicals are shown in Table 7.6.

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TABLE 7.2.—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 Fire-Trol 931L Fire-Trol 934L Phos-Chek Phos-Chek XAR Phos-Chek 202R 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-*s*-triazine	None
Dalapon	2,2-dichloropropionic acid	None
Dicamba	3,6-dichloro-*o*-anisic acid	None
Dinoseb	2-sec-butyl-4,6-dinitrophenol	None
DSMA	Disodium methanearsonate	None
Fosamine ammonium	Ammonium ethylcarbamoylphosphonate	Krenite
Glyphosate	N-phosphonomethylglycine	Roundup
Hexazinone	3-cyclohexyl-6-(dimethylamino)-1-methyl-1,3,5-triazine-2,4(1H,3H)-dione	Velpar
MSMA	Monosodium methanearsonic acid	None
Picloram	4-amino-3,5,6-trichloropicolinic acid (and various esters and salts)	Tordon 22K Tordon 101 (also contains 2,4-D)
SDMA	Sodium dimethyl arsonate	None
Silvex	2-(2,4,5-trichlorophenoxy)propionic acid	None
Triclopyr	[(3,5,6-trichloro-2-pyridinyl)oxy] acetic acid	Garlon
Insecticides		
Acephate	0,S-dimethyl acetylphosphoramidothioate	Orthene
Azinphos-methyl	0,0-dimethyl-S-[(4-oxo-1,2,3-benzotriazine-3-(4H)-yl) methyl]phosphorodithioate	Guthion
B.t.	*Bacillus thuringiensis*	None
Carbaryl	1-naphthyl-N-methylcarbamate	Sevin Sevin-4-Oil
Carbofuran	2,3-dihydro-2,2-dimethyl-7-benzofuranyl methylcarbamate	Furadan
Chlordecone	Decachloro-octahydro-1,3,4-metheno-2H-cyclobuta(cd)pentalene-2-one	Kepone
DDT	Dichlorodiphenyltrichloroethane	None
Malathion	0,0-dimethyl-S-(1,2-dicarbethoxyethyl)phosphorodithioate	None
Methoxychlor	2,2-bis(p-methoxyphenyl)-1,1,1-trichloroethane	None
NPV	Nuclear polyhedrosis virus	None

TABLE 7.3.—Pesticide applications by the U.S. Forest Service during six fiscal years in the period 1979–1989.[a] Dashes mean that no use was reported; empty cells mean data are unavailable.

Pesticide	Hectares	Kilograms (%)	Hectares	Kilograms (%)
	Fiscal year 1989		**Fiscal year 1987**	
Insecticides	67,296	3,702 (2.7)	255,953	106,763 (38.9)
Herbicides	48,597	65,748 (48.0)	60,458	101,484 (37.0)
Fumigants, fungicides	561	67,358 (49.2)	589	64,010 (23.3)
Repellants	10	16 (<0.1)	6,337	1,395 (0.5)
Rodenticides	23,585	154 (0.1)	23,187	689 (0.3)
Wood preservatives	—	—	—	—
Piscicides, predacides	16,766	11 (<0.1)	13,977	29 (<0.1)
Algicides	—	—	—	—
Behavioral chemicals	—	—	—	—
Total	156,815	136,989 (100)	360,501	274,370 (100)
	Fiscal year 1985		**Fiscal year 1983**	
Insecticides[b]	336,398	180,820 (51.5)	199,861	224,767 (44.8)
Herbicides[c]	61,200	126,113 (35.9)	99,174	238,894 (47.6)
Fumigants, fungicides	916	40,782 (11.6)	1,349	34,806 (6.9)
Repellants	6,108	1,984 (0.6)	11,237	1,940 (0.4)
Rodenticides	29,219	1,301 (0.4)	23,349	1,365 (0.3)
Wood preservatives	—	—	—	—
Piscicides, predacides		36 (<0.1)	12,230	135 (<0.1)
Algicides	—	—	7	29 (<0.1)
Behavioral chemicals	—	—	—	—
Total	433,841	351,036 (100)	347,207	501,936 (100)
	Fiscal year 1981		**Fiscal year 1979**	
Insecticides[b]	20,102	14,331 (6.3)	110,247	78,471 (23.2)
Herbicides[c]	79,742	172,741 (76.0)	74,483	213,725 (63.2)
Fumigants, fungicides	1,464	38,720 (17.0)	540	36,861 (10.9)
Repellants	2,517	580 (0.2)	3,845	4,144 (1.2)
Rodenticides	20,857	712 (0.3)	18,179	4,112 (1.2)
Wood preservatives		116 (<0.1)	—	—
Piscicides, predacides	37	13 (<0.1)	97	415 (0.1)
Algicides	3	160 (<0.1)	22	185 (<0.1)
Behavioral chemicals	—	—	919	8 (<0.1)
Total	124,722	227,373 (100)	208,332	337,921 (100)

[a]Fiscal years of the U.S. government begin on October 1 of the previous year and extend to September 30 of the year designated. Data sources are Pesticide-Use Advisory memoranda (2150 Pesticide-Use Management and Coordination) of the U.S. Forest Service, Washington, D.C.; 1989, Memorandum 450 (May 30, 1990); 1987, Memorandum 429 (July 7, 1988); 1985, Memorandum 388 (April 15, 1986); 1983, Memorandum 355 (May 18, 1984); 1981, Memorandum 316 (April 5, 1982); 1979, Memorandum 246 (June 5, 1980).

[b]Proportions of insecticide weights applied from aircraft: 97% in 1985, 96% in 1983, 29% in 1981, 87% in 1979.

[c]Proportions of herbicide weights applied from aircraft: 0% in 1985, 31% in 1983, 30% in 1981, 26% in 1979.

TABLE 7.4.—Herbicide applications by the U.S. Forest Service during six fiscal years in the period 1979–1989. Dashes mean that no use was reported or that use amounted to less than 0.1% of the total weight of herbicides applied. See Table 7.3, footnote a, for data sources.

Herbicide	Hectares	Kilograms (%)	Hectares	Kilograms (%)
	Fiscal year 1989		**Fiscal year 1987**	
Hexazinone	8,670	16,611 (28.6)	23,191	46,233 (47.9)
2,4-D + picloram	4,604	4,012 (6.9)	5,170	4,955 (5.1)
2,4-D	2,115	3,045 (5.2)	4,100	8,333 (8.6)
Glyphosate	3,194	5,456 (9.4)	4,966	6,101 (6.3)
Picloram	3,180	1,238 (2.1)	4,022	2,317 (2.4)
Triclopyr	17,172	22,276 (38.4)	10,987	17,894 (18.6)
2,4-D +2,4-DP	18	42 (<0.1)	53	654 (0.7)
2,4-D + dicamba	1,061	2,479 (4.3)	1,483	3,641 (3.8)
Fosamine ammonium	228	1,743 (3.0)	212	2,056 (2.1)
Dicamba	815	330 (0.6)	916	1,106 (1.2)
2,4-D[a]	90	93 (0.2)	991	1,721 (1.8)
MSMA	63	200 (0.4)	65	229 (0.2)
Atrazine	224	372 (0.6)	39	192 (0.2)
Simazine	2	10 (<0.1)	105	258 (0.3)
Dalapon	—	—	—	—
Ammonium sulfamate	25	102 (0.2)	212	748 (0.8)
Amitrole	—	—	2	5 (<0.1)
Sodium metaborate + sodium chlorate	—	—	—	—
Mineral spirits	3	75 (0.1)	—	—
Total	41,464	58,084 (100)	56,514	96,443 (100)
	Fiscal year 1985		**Fiscal year 1983**	
Hexazinone	21,226	44,195 (36.6)	14,515	30,756 (13.3)
2,4-D + picloram	10,454	16,445 (13.6)	14,031	22,544 (9.7)
2,4-D	6,815	16,128 (13.4)	28,852	73,975 (31.9)
Glyphosate	7,146	12,338 (10.2)	13,010	25,734 (11.1)
Picloram	3,638	11,480 (9.5)	9,308	11,635 (5.0)
Triclopyr	5,694	9,715 (8.0)	3,387	6,244 (2.7)
2,4-D + 2,4-DP	472	2,377 (2.0)	309	2,674 (1.2)
2,4-D + dicamba	901	1,986 (1.6)	800	1,995 (0.9)
Fosamine ammonium	205	1,793 (1.5)	484	3,697 (1.6)
Dicamba	1,030	1,370 (1.1)	1,741	2,822 (1.2)
2,4-D[a]	428	1,272 (1.1)	148	90 (<0.1)
MSMA	312	714 (0.6)	50	144 (<0.1)
Atrazine	215	249 (0.2)	5,217	21,327 (9.2)
Simazine	67	296 (0.2)	869	4,250 (1.8)
Dalapon	98	235 (0.2)	3,339	22,495 (9.7)
Ammonium sulfamate	12	96 (<0.1)	36	849 (0.4)
Amitrole	64	110 (<0.1)	291	881 (0.4)
Sodium metaborate + sodium chlorate	—	—	—	—
Mineral spirits	—	—	—	—
Total	58,777	120,799 (100)	96,387	232,112 (100)

TABLE 7.4.—Continued.

Herbicide	Hectares	Kilograms (%)	Hectares	Kilograms (%)
	Fiscal year 1981		**Fiscal year 1979**	
Hexazinone	1,841	2,942 (1.8)	155	381 (0.2)
2,4-D + picloram	27,988	40,435 (24.7)	23,068	61,374 (29.7)
2,4-D	29,376	65,986 (40.2)	29,724	84,061 (40.6)
Glyphosate	5,054	7,993 (4.9)	1,484	2,649 (1.3)
Picloram	6,147	15,296 (9.3)	6,416	11,316 (5.5)
Triclopyr	—	—	—	—
2,4-D + 2,4-DP	462	1,896 (1.2)	1,276	4,058 (2.0)
2,4-D + dicamba	652	1,552 (0.9)	2,522	6,791 (3.3)
Fosamine ammonium	689	3,036 (1.9)	789	3,601 (1.7)
Dicamba	1,703	2,171 (1.3)	429	637 (0.3)
2,4-D[a]	—	—	—	—
MSMA	380	280 (0.2)	1,440	8,439 (4.1)
Atrazine	2,415	9,854 (6.0)	2,144	8,580 (4.0)
Simazine	345	3,314 (2.0)	1,739	4,503 (2.2)
Dalapon	1,735	5,758 (3.5)	1,716	4,813 (2.3)
Ammonium sulfamate	105	1,361 (0.8)	182	1,588 (0.8)
Amitrole	399	1,058 (0.6)	356	776 (0.4)
Sodium metaborate + sodium chlorate	6	1,093 (0.7)	4	360 (0.2)
Mineral spirits	—	—	36	2,994 (1.4)
Total	79,297	164,025 (100)	73,480	206,921 (100)

[a]Applied in combinations not otherwise listed.

TABLE 7.5.—Insecticides most commonly applied by the U.S. Forest Service during six fiscal years in the period 1979–1989. Dashes mean that no use was reported or that use amounted to less than 0.1% of the total weight of insecticides applied; empty cells mean data are unavailable. See Table 7.3, footnote a, for data sources.

Insecticide	Hectares[a]	Kilograms (%)	Hectares[a]	Kilograms (%)
	Fiscal year 1989		**Fiscal year 1987**	
Malathion	448	251 (8.4)	3,026	149 (2.1)
Carbaryl	2,337	1,958 (65.5)	55	4,911[b] (68.8)
Azinphos-methyl[c]	168	557 (18.6)	279	1,437 (20.1)
Lindane	116	84 (2.8)	12	194 (2.7)
Carbofuran[c]		23 (0.8)		92 (1.3)
Diazanon[d]	62	103 (3.4)	101	91 (1.3)
Acephate	—	14 (0.5)	424	263 (3.7)
Ethylene dibromide[e]	—	—	—	—
Toxaphene[f]	—	—	—	—
Tetrachlorvinphos[f]	—	—	—	—
Bacillus thuringiensis	53,878	2,144,266[g]	75,453	2,441,686[g]
Total	57,009	2,990 (100)[h]	79,350	7,137 (100)[h]
	Fiscal year 1985		**Fiscal year 1983**	
Malathion	241,626	164,781 (91.6)	231	337 (0.2)
Carbaryl	10,220	9,005 (5.0)	188,711	213,205 (95.4)
Azinphos-methyl[c]	478	4,446 (2.5)	36	4,167 (1.9)
Lindane		1,293 (0.7)		327 (0.1)
Carbofuran[c]	9	173 (<0.1)	8	3,321 (1.5)
Diazinon[d]		130 (<0.1)	68	88 (<0.1)
Acephate	—	—	293	293 (0.1)
Ethylene dibromide[e]	—	—		1,740 (0.8)
Toxaphene[f]	—	—		18 (<0.1)
Tetrachlorvinphos[f]	—	—	9	30 (<0.1)
Bacillus thuringiensis	69,898	1,174,998[g]	5,955	78,798[g]
Total	322,231	179,828 (100)[h]	195,311	223,526 (100)[h]
	Fiscal year 1981		**Fiscal year 1979**	
Malathion	3,855	2,202 (19.3)	78,253	42,416 (54.7)
Carbaryl	2,017	2,051 (18.0)	20,711	22,910 (29.6)
Azinphos-methyl[c]		2,917 (25.6)		1,961 (2.5)
Lindane		74 (0.6)	150	140 (0.2)
Carbofuran[c]		2,500 (21.9)		2,481 (3.2)
Diazinon[d]		73 (0.6)	41	56 (<0.1)
Acephate	1,220	1,026 (9.0)	9,470	5,310 (6.9)
Ethylene dibromide[e]		347 (3.0)		1,144 (1.5)
Toxaphene[f]		218 (1.9)		1,041 (1.3)
Tetrachlorvinphos[f]	—	—	13	31 (<0.1)
Bacillus thuringiensis	—	—	—	—
Total	7,092	11,408 (100)	108,638	77,490 (100)

[a]Not all applications were per hectare. For control of seed and cone insects, for example, the pesticide-use memoranda give values as number of trees treated.
[b]The majority was applied to 12,593 individual trees.
[c]Control of seed and cone insects in seed production areas.
[d]Control of insects in forest tree nurseries.
[e]Control of bark beetles on cut logs.
[f]Control of ticks and lice on cattle.
[g]Billion international units (BIU), not kilograms.
[h]Total does not include *Bacillus thuringiensis* use.

TABLE 7.6.—Typical application rates of some forest chemicals.

Chemical	kg/hectare[a]	Method
	Herbicides	
2,4-D	1.12–4.48	
Picloram	≤1.12–5.0	
Hexazinone	0.55–3.36	Broadcast
	1.12–2.24	Basal treatment, stem injection
Atrazine	≤4.48	
Triclopyr	0.28–10.0	
MSMA	4.4–288	
Fosamine ammonium	3.36–6.72	
Glyphosate	<4.48	
Dalapon	0.46–7.6[b]	Ground[b]
	5.6–9.6[b]	Aerial[b]
	Insecticides	
Malathion	0.8	Aerial
Carbaryl	0.5–2.24	Agriculture
	<1.12	Forestry
Acephate	1.5	
	Fertilizers	
Urea-N	168–224[c]	

[a]Active ingredient.
[b]U.S. Forest Service (1984).
[c]Moore and Norris (1974).

TABLE 7.7.—Fire retardant use in the USA.[a]

Year	Quantity used (L)	User group
1956	87,000	All users
1961	28,400,000	All users
1966	22,500,000	U.S. Forest Service
1966	12,200,000	Calif. Division Forestry
1966	3,800,000	Bureau Land Management
1970	64,400,000	All users
1977[b]	56,669,902	U.S. Forest Service
1978[b]	24,371,221	U.S. Forest Service
1979[b]	54,795,771	U.S. Forest Service
1980[b]	39,348,023	U.S. Forest Service
1981[b]	44,712,371	U.S. Forest Service

[a]G. E. Cargill, U.S. Forest Service, Washington, D.C., personal communications, December 14, 1980, and September 21, 1982 (memorandums with attachments).
[b]Fiscal year: October 1 of the previous year through September 30 of the year designated. About 70% of this use is in Oregon, Washington, and California.

Fertilizers

Fertilizers are applied annually to only a small portion of commercial forest land (Table 7.1). Several private and public land-management groups, however, have been applying forest fertilizers for over 20 years, particularly in the northwestern USA where nitrogen deficiencies occur and, to a much lesser degree, in the southeastern states where phosphorus deficiencies may occur. Between 1965 and 1975, about 300,000 hectares of Douglas-fir forests were fertilized in western Oregon and Washington (Moore 1975b). Allen (1987) estimated that by 1986, more than 1 million hectares of Douglas-fir would have been fertilized. Bengtson (1979) and Allen (1987) wrote excellent articles on the use of fertilizers in American forestry.

Fire Retardants

The use of chemical fire retardants increased steadily after they were introduced in the 1930s and varied between 24 and 65 million liters during the 1970s and early 1980s (Table 7.7). Douglas (1974) and Norris et al.[2] summarized most of the literature through the mid-1970s on both the use and environmental effects of chemical fire retardants. 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 primarily of ammonium phosphate or ammonium sulfate and small amounts of several other chemicals such as dyes, wetting agents, thickeners, corrosion inhibitors, and bactericides.

Relation of Chemical Use to Salmonid Habitats

The quality of the water that forested watersheds yield reflects human activities and natural processes. Forest lands are only one-third of the total area of the USA, but they receive more than half of the total precipitation and yield more than three-fourths of the total streamflow. Forested watersheds in the USA 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 amounts from other lands (Storey 1965). Clearly, the possibility that chemical use in forest management may alter water quality, or some other aspect of fish habitat, deserves careful consideration.

The chemicals used in forestry may have direct or indirect effects or no effect on salmonids. 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

[2]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. Forest Service, Pacific Northwest Research Station, Forestry Sciences Laboratory, Corvallis, Oregon, 1978.

FIGURE 7.1.—A direct chemical effect on an organism requires a chain of events.

(Figure 7.1). Direct chemical effects can be evaluated by using traditional concepts of toxicology and dose–response relationships.

Indirect effects result from chemically induced modification of the habitat. Examples of indirect effects are insecticide-induced decreases in the biomass of terrestrial or aquatic insects that result in a decrease in the supply of food for salmonids, and reductions 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 risk 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 risk analysis requires equal consideration of both the likelihood of exposure and the toxicity of the chemical (Norris 1971b; Sanders 1979; U.S. Forest Service 1984).

Toxicity

Acute toxicity is the short-term response of organisms to one or 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 continuous or repeated, 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 from aircraft (Table 7.3, footnotes b and c), although a large proportion of herbicides is applied by ground-based equipment such as hand-held nozzles fed from either high- or low-pressure pumping systems, backpack sprayers, air-blast sprayers, or direct stem-injection equipment; occasionally, pelletized chemical may be scattered by hand. 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 characteristics. The concepts, principles, and practice of aerial pesticide application were 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 U.S. Department of Agriculture (1976) handbook.

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 stream's ratio of surface area to volume. The persistence of the chemical in surface water 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 (Norris and Montgomery 1975).

Drift from nearby spray areas is similar to direct application except that peak concentrations are lower and the probability that stream organisms will be affected 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.

Small and ephemeral stream channels are difficult to see from the air and may be sprayed along with the rest of the area. The problems 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. 1982, 1984).

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 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 1971a). 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 substantial precipitation; therefore, the concentrations in the water will be considerably 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 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 (Figure 7.2). This behavior largely determines the route of entry of chemicals into forest streams.

Chemicals on sediment.—Stream sediments may be contaminated with forest chemicals by deposition of soils carrying adsorbed chemicals from the land or by adsorption of chemicals from the water (Barnett et al. 1967).

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 occurs most frequently during intense winter storms from late November through February. Thus, appreciable quantities of a particular chemical must persist for 1–9 months for harmful amounts to be present in the soil at the time the first winter erosion is 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 close temporal and spatial proximity to 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 salmonid habitats is discussed in detail by Chamberlin et al. (1991, this volume).

FIGURE 7.2.—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 may be adsorbed from water by sediments already in the stream. Chemicals may bond to sediments by chemical or physical means (or both) according to the physicochemical properties of both chemical and sediment. The adsorption process 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 chapter.

Norris (1969) and Norris et al. (1982, 1984) believed that the discharge of pesticides in stream water during periods of heavy precipitation represents the mobilization of chemicals in ephemeral stream channels, though their research did not distinguish between pesticides in solution and those adsorbed on sediments carried in the streamflow.

Chemicals in the food chain.—Chemicals may be in or on the food of salmonids if the food substance is sprayed directly (for instance, if terrestrial insects that are sprayed fall into the water), or if food substances adsorb or 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, 1980b) and Geyer et al. (1980) provided good reviews and substantial data on bioaccumulation of organic chemicals, including many pesticides. The physicochemical properties of the compound and the organism are the predominant factors that determine the extent of bioaccumulation. The most important properties are the amount 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 ratio of fat to water solubilities. Pertinent examples are DDT or TCDD (tetrachlorodibenzodioxin) in fish. Chemicals that are highly water

FIGURE 7.3.—Relations of the water solubility of chemicals to their bioaccumulation.
(A) Aqueous solubilities and bioconcentration factors of organic chemicals in rainbow trout. Log_{10} (bioconcentration factor) = 3.41 − 0.508 $\log_{10}$ (water solubility); $r^2 = 0.93$. The bioconcentration factor is the concentration of a chemical in fish divided by its concentration in water. (From Figure 2 of Chiou et al. 1977.)
(B) Aqueous solubilities and bioaccumulation factors of organic chemicals in adipose tissues of rats. Log_{10} (bioaccumulation factor) = 1.20 − 0.56 $\log_{10}$ (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 Figure 1 of Geyer et al. 1980.)

soluble, like picloram or glyphosate, show little tendency to bioaccumulate. The relation of water solubility to bioaccumulation is illustrated in Figure 7.3, and data for specific chemicals are given in Table 7.8. Bioconcentration factors greater than 1,000 indicate a need for precise risk analysis, whereas values less than 100 do not warrant experimental verification (Kenaga 1980b).

TABLE 7.8.—Water solubilities of forest chemicals and measured bioconcentration factors (BCF = concentration in organisms/concentration in exposure medium).

Chemical[a]	Solubility in water (mg/L)	Test organism[b]	Exposure, E (mg/kg or mg/L) or application, A (kg/hectare)	Duration	Amount detected (mg/kg)	BCF	Source[c]
			Herbicides				
2,4-D		*Scenedesmus* (alga)	E 0.022	8 h		2.2	1
		Fish[d]	E 2.5	4–14 d	<0.005		6
		Gastropod	E 0.0002–0.05		0		9
Acid	900					13	3
DES	300,000						
Esters	<500						
MP		Fish[d]	E 2.5	4–14 d	0.031–0.122		6
GR		Mussels	A 1.2		0.38–0.70		8
GR		Fish	A 1.2		<0.04		8
BOE		Bluegill	E 3.0	8 d	<0.05		7
Picloram	430					20	3
Hexazinone	33,000						10
Atrazine	33					86	3
		Annelids				3.5	4
		Mayfly				480	4
Triclopyr	430					20	3
MSMA	250,000						
Fosamine	1,790,000						
Glyphosate	12,000					3	3
		Catfish	E 10.0	14 d		0.55	5
		Bass	E 10.0	14 d		0.12	5
		Trout	E 10.0	14 d		0.11	5
		Trout					
		Fillet	E 2.0		80	40	2
		Eggs	E 2.0		60	30	2
		Midge	E 2.0		0		2
Dalapon	800,000					0.4	3
Dinoseb	50					68	3
			Insecticides				
DDT	0.002					22,500	3
Malathion	145					37	3
Carbaryl	40					77	3
Azinphos-m	29						
Carbofuran	415					21	3
Acephate	650,000					0.3	3
			Fertilizers				
Urea	1,000,000					0.5	3

[a]BOE = butoxyethyl ester; DES = diethylamine salt; GR = granules; MP = metabolic products; azinphos-m = azinphos-methyl; fosamine = fosamine ammonium.

[b]Bass = largemouth bass; catfish = channel catfish; mayfly = nymphs; midge = larvae; trout = rainbow trout.

[c]1 = Boehm and Mueller (1976); 2 = Folmar et al. (1979); 3 = Kenaga (1980b); 4 = Lynch et al. (1982); 5 = Sacher (1978); 6 = Schultz (1973); 7 = Sigmon (1979); 8 = Smith and Isom (1967); 9 = Streit (1979); 10 = U.S. Forest Service (1984).

[d]Three species.

Approaches to Risk Analysis

Several specific risk analysis methods have been used for aquatic species. Most have used a specified fraction (expressed as a decimal) of the LC50 (or similar measure of response) as an estimate of the no-toxic-effect exposure level (U.S. Environmental Protection Agency 1973b, 1976). The LC50 is the chemical concentration lethal to half the test organisms, and the specified fraction of it is called the "no-observable-effect level," or NOEL. When only acute exposures and survival were the primary interest, the estimates of NOEL ranged from 0.1 to 0.05 of the LC50 (Sprague 1971). For compounds that are more persistent in the environment or for estimates of chronic exposures, estimates of the NOEL have ranged from 0.1 to 0.01 of the LC50 (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 h or more). 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, in which the concentration of pollutant changes rapidly.

A more refined and realistic method has been published (U.S. Environmental Protection Agency 1980). It 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-h average permissible concentration. This 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 risk assessment in this chapter, we have selected an approach that combines these two approaches. We have used fractional LC50 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 risk analysis. 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 (risk analysis) 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) intensively reviewed this subject for many pesticides. Although their emphasis was on agriculture, many of the concepts and some of the data are relevant in forestry. Malik and Vanden Born (1986) reviewed herbicides as used in Canada.

In this section, we review what is known about the physicochemical properties, movement and persistence in soil, entry and fate in forest waters, bioaccumulation, and toxicity to aquatic species of 10 herbicides, 5 chemical insecticides, 2 biological 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 USA. To the degree possible, we have relied most heavily on field studies in the northwestern USA and laboratory studies involving species common (or representative of species that are common) in northwestern USA forest ecosystems. In many cases, however, it has been necessary to go beyond these in order to fill critical data gaps or to reinforce other data.

The common and scientific names of invertebrates mentioned in this chapter are in Table 7.9. Information on rates and methods of application and carriers for pesticides are in the "Pacific Northwest Weed Control Handbook" (William et al. 1987), the "Pacific Northwest Insect Control Handbook" (Capizzi et al. 1987), "Pesticide Uses for Forestry,"[3] and "Pesticide Background Statement" (U.S. Forest Service 1984).

Herbicides: 2,4-D

The herbicide 2,4-D is one member of a large family of phenoxy herbicides that have been reviewed by the National Research Council of Canada (1978) and Norris (1981). For many years 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. More specific information on the use of this herbicide was reviewed by National Forest Products Association,[3] U.S. Forest Service (1984), and Newton (1987).

Behavior in the environment.—The physicochemical 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 it 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 water solubility of 2,4-D in various forms is shown in Table 7.8. Many 2,4-D esters are available; those commonly used in forestry are low in water solubility (<500 mg/L) 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} mm Hg (high-volatile esters) to 10^{-6} mm Hg (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. The physicochemical properties of 2,4-D were reviewed in more detail by House et al.,[4] National Research Council of Canada (1978), U.S. Forest Service (1984), and Weed Science Society of America (1989).

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[3]Unpublished report, "Pesticide uses for forestry," prepared by National Forest Products Association, 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. Advanced Research Project Agency, Department of Defense, Midwest Research Institute Project 3103-B, Contract DAHC 15-68-C-0119, Kansas City, Missouri, 1967.

TABLE 7.9.—Common and scientific names of invertebrates referred to in text and tables.

Common name	Scientific name
	Phylum Arthropoda
	ORDER Amphipoda
Scuds, amphipods	*Gammarus fasciatus* Say
	Gammarus lacustris Sars
	Gammarus 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.
Phanton midge	*Chaoborus* sp.
Midges	*Chironomus tenans* (Fabricius)
	Chironomus plumosus (Linnaeus)
	ORDER Ephemeroptera
Mayflies	*Hexagenia bilineata* (Say)
	Baetis sp.
	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.
	Phylum Mollusca
	ORDER Gastropoda
Snails	*Helisoma campanulata* (Say)
	Stagnicola emarginata (Say)

In soil, 2,4-D persists for only short periods (Table 7.10). Research reviewed by House et al.[4] indicates microbial decomposition is the predominant cause 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. More recent

research reviewed by National Research Council of Canada (1978), Norris (1981), and U.S. Forest Service (1984) supports these conclusions.

Soil organic matter adsorbs 2,4-D extensively (Norris 1970b), which tends to reduce the herbicide's mobility in soil. In light, sandy soils with a high pH, however, it may show substantial mobility.[4] Forest soils are usually high in organic matter and low in pH, which inhibits 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).

Norris (1981) reviewed the entry and fate of 2,4-D (and the other phenoxy herbicides) in forest waters. He concluded that direct application and drift to surface waters are the processes most likely to produce the highest residue levels, but that 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 stream concentrations of 2,4-D ranging from 0.001 to 0.13 mg/L during and shortly after application (Table 7.11). The time required to return to nondetectable levels (<0.001 mg/L) varied with the nature of the area and the maximum concentration observed. Times ranging from less than 1 h to more than 168 h have been noted, but they are usually less than 2 d. Application to marshy areas can lead to higher than normal levels of stream contamination; in one instance, 2,4-D concentrations approaching 0.9 mg/L were found in water flowing from a marshy area. In other areas, long-term outflow of 2,4-D was not 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 that 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 from whole watersheds have been conducted. In two separate studies, Norris et al. (1982) and Suffling et al. (1974) found that less than 0.02% of the 2,4-D applied to a watershed appeared in streamflow.

When operational applications of 2,4-D have been monitored, the results have largely agreed with research findings. The U.S. Forest Service[5] summarized data on phenoxy herbicides in streams after 304 applications in northwestern forests over 4 years; 84% of the applications resulted in no detectable stream contamination, and only 1% led to herbicide concentrations exceeding 0.01 mg/L.

Few field data are available on 2,4-D levels in sediments 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) all occur. Streit (1979) reported that concentrations of 2,4-D on aquatic sediments were no greater than in the water. Results of some other studies are summarized in Table 7.10. Nesbitt and Watson (1980a,

[5]Memorandum, "Summary of phenoxy herbicides in water," (2150, Pesticide-Use Management), from F. J. Kopechky to the Chief, U.S. Department of Agriculture, Forest Service, June 23, 1980.

Text continues on page 229

TABLE 7.10.—Persistence of forest chemicals in soil and water.

Chemical[a]	Substrate[b]	Initial amount in soil or water (mg/kg, mg/L, or kg/hectare*)	Time interval[c]	% remaining	Time to non-detection[c]	Source[d]
		Herbicides				
2,4-D	Forest floor (L)		10–20 d	50		25–27
	Oak forest (L)		30 d	0		1
	Forest (F)		31 d	10		40
	Chaparral (F)		15 d	60		30
Picloram	Hardwood forest (NC)	5.0	4 w	50	~28 w	23
Hexazinone	Agricultural (S)		<6 m	50		
	Blueberry fields (NS)	2.0–4.0*	1 y	<5		16
	Loam forest soil (S)		<4 w	50		
	Clay forest soil (S)		6 w	50		
Hexazinone P	Sandy forest soil (S)		14 w	50		
Atrazine	Soil		5 d	33		4
	Agricultural soil		1 y	<10		5
Triclopyr	Soil (WV)	4.4–18	14–16 d	50	28 d	20
	Hill pasture (OR)	3.4, 10.1*	75–81 d	50		28
MSMA	Water		5 d	10–50		42
Fosamine	Greenhouse		10 d	50		14
	DE, IL, FL (F)		7 d	50		14
Glyphosate	Foliage and litter		10–27 d	50		24
	Soil		29–40 d	50		24
	Soil		28 d	55		35,36
	Static water		12 d	50		34
	Soil "A"		32 d	60		22
	Soil "B"		32 d	90.5		22
	Soil "C"		32 d	97		22
Dalapon			<30 d	0		3
Dinoseb	Warm, moist soils		3–5 w	0		18
		Insecticides				
Malathion	Sterile, nonsterile soils		24 h	10–50		19
		8.6*	6 m	0		32
	River water		7 d	20	28 d	9
	pH = 7; 37°C (L)		1.3 d	50		10
	pH = 7; 20°C (L)		11 d	50		10
	pH = 6.1		160 d	50		10,19
	Natural aqueous system		230 h	<10		19
	Fresh water		11 d	50		41
	Saline water		<2 d	50		41
Carbaryl	Soil	3.36–30.2*	8 d	50		39
	Soil		1.5–6 m	50		13

TABLE 7.10.—Continued.

Chemical[a]	Substrate[b]	Initial amount in soil or water (mg/kg, mg/L, or kg/hectare*)	Time interval[c]	% remaining	Time to non-detection[c]	Source[d]
	River water	0.01	7 d	5	14 d	9
	Farm pond (water)	6.7*			2 d	33
	Sediment				4 d	6
	Brooks and streams	0.84*	23–28 h	50		37
Azinphos-m	Ponds, pH 7.2–8	1.0	2 d	50	14 d	21
	Muck soils (FL)		1 m	<50		2
	Clay soils (LA)		3 m	>50		2
	Clay soils (KN)		2–3 m	50		2
	Silty clay loam		105 d	1		15
Carbofuran	Loam, sandy soils with oats		14 d	10–40		11
	Soil		46–117 d	50		7
	Sterile, unsterile soils[e]		3–50 w	50		12
Acephate	Soils (PA); 0.56 kg/hectare applied	5.5	20 d	0.5		8
		5.5	70 d	<0.4		8
	Open forest floor (PNW)		10 d	<10		38
	Semiopen or densely covered area		10 d	<30	30 d	38
		0.1			6 w	31
B.t.	Foliage, cool, cloudy		3.9 d	50		29
	Foliage, hot, sunny		7.7 d	50		29
	White pine		1 d	20		17
	White pine		14 d	1		17
	White pine		28 d	<0.1		17

[a]Azinphos-m = azinphos-methy; *B.t.* = *Bacillus thuringiensis*; fosamine = fosamine ammonium; P = pellets.

[b]DE = Delaware; F = field study; FL = Florida; KN = Kansas; IL = Illinois; L = laboratory study; LA = Louisiana; NC = North Carolina; NS = Nova Scotia; OR = Oregon; PA = Pennsylvania; PNW = Pacific Northwest; S = southeastern USA; WV = West Virginia.

[c]d = day; m = month; w = week; y = year.

[d]1 = Altom and Stritzke (1972); 2 = Anderson et al. (1974); 3 = Ashton (1982); 4 = Axe et al. (1969); 5 = Birk and Roadhouse (1964); 6 = California Department of Fish and Game (1963, unpublished; see text footnote 9); 7 = Caro et al. (1973); 8 = Devine (1975); 9 = Eichelberger and Lichtenberg (1971); 10 = Freed et al. (1979); 11 = Fuhremann and Lichtenstein (1980); 12 = Getzin (1973); 13 = Goring et al. (1975); 14 = Han (1979b); 15 = Iwata et al. (1977); 16 = Jensen and Kimball (1987); 17 = Kearby et al. (1972); 18 = Klingman and Ashton (1975); 19 = Konrad et al. (1969); 20 = McKellar et al. (1982); 21 = Meyer (1965); 22 = Moshier and Penner (1978); 23 = Neary et al. (1985); 24 = Newton et al. (1984); 25 = Norris (1966); 26 = Norris (1970a); 27 = Norris and Greiner (1967); 28 = Norris et al. (1987); 29 = Pinnock et al. (1971); 30 = Plumb et al. (1977); 31 = Rabeni and Gibbs (1977, unpublished, U.S. Forest Service Report NA-FR-7, Broomall, Pennsylvania), 32 = Roberts et al. (1962); 33 = Romine and Bussian (1971, unpublished; see text footnote 8); 34 = Sacher (1978); 35 = Sprankle et al. (1975a); 36 = Sprankle et al. (1975b); 37 = Stanley and Trial (1980); 38 = Szeto et al. (1978); 39 = Union Carbide (1968); 40 = U.S. Forest Service (1977b); 41 = Walker (1978); 42 = Woolson et al. (1976).

[e]Losses were 7–10 times faster in alkaline soils (pH 7.9) than in acid or neutral soils (pH 4.3–6.5).

TABLE 7.11.—Peak concentrations of forest chemicals in soils, lakes, and streams after application.

Chemical[a] and system[b]	Application rate (kg/hectare)	Concentration (mg/L or mg/kg*) Peak	Subsequent	Time interval[c]	Time to non-detection	Source[d]
		Herbicides				
2,4-D	2.24	0.001–0.13			1–168 h[e]	17
Marsh	2.24	0.09				17,18
2,4-D BE						
Built pond	23.0					1
Water		3.0	1.0	85 d		
			0.2	180 d		
Sediment		8.0*	4.0*	13+ d		
			0.4–0.6*	82–182 d		
Aquatic plants			206*	7 d		
			8*	82 d	182 d	
2,4-D AS						
Reservoir		3.6	0	13 d		7
Picloram						
Runoff		0.078				19
Runoff		0.038				23
Ephemeral stream	2.8	0.32		157 d	915 d	9
Stream	0.37					3
Hexazinone						
Stream (GA)	1.68	0.044		3–4 m		11
Forest (GA)	1.68					14
Litter		0.177*	<0.01*	60+ d		
Soil		0.108*	<0.01*	90 d		
Ephemeral stream		0.514		3 d		
Perennial stream		0.442		3 d		
Atrazine						
Stream	3.0	0.42	0.02	17 d		16
Built ponds						10
Water		0.50	0.05	14 d		
			0.005	56 d		
Sediments		0.50*	0.9*	4 d		
		0.50*	0.25*	56 d		
Triclopyr						
Pasture (OR)	3.34	0.095*				20
Glyphosate						
Water	3.3	0.27	0.09	5.5 h		15
			<0.01	3 d		
Dalapon						
Field irrigation water		0.023–3.65	<0.01	Sev h		5
		Insecticides				
Malathion						
Streams	0.91					24
Unbuffered		0.037–0.042				
Buffered		0–0.017				
Carbaryl						
Streams and ponds (E)		0–0.03				24
Streams, unbuffered (PNW)		0.005–0.011			48 h	24
Water	0.84	0.026–0.042				8
Brooks with buffer	0.84	0.001–0.008				22
Rivers with buffer	0.84	0.000–0.002				22
Streams, unbuffered	0.84	0.016				22

TABLE 7.11.—Continued.

Chemical[a] and system[b]	Application rate (kg/hectare)	Concentration (mg/L or mg/kg*) Peak	Subsequent	Time interval[c]	Time to non-detection	Source[d]
Ponds	0.84					6
Water		0.254			100–400 d	
Sediment		<0.01–5.0*[f]				
Acephate						
Streams		0.003–0.961				4
Streams	0.56	0.113–0.135	0.013–0.065	1 d		21
Pond sediment and fish				14 d		2
		Fertilizers				
Urea	224					
Urea-N						
Forest stream (OR)		0.39	0.39	48 h		12
Dollar Cr (WA)		44.4				13
NH_4^+-N						
Forest stream (OR)		<0.10				12
Tahuya Cr (WA)		1.4				13
NO_3^+-N						
Forest stream (OR)		0.168		72 h		12
Elochoman R (WA)		4.0				13

[a]2,4-D BE = 2,4-D butoxyethanol ester; 2,4-D AS = 2,4-D amine salt + ester.

[b]E = eastern USA; Cr = Creek; GA = Georgia; PNW = Pacific Northwest; OR = Oregon; R = River; WA = Washington; buffer = wooded riparian strip.

[c]d = day; h = hours; m = months; sev h = several hours. Intervals are times from application to measurement of peak or subsequent concentration, whichever is the last measurement indicated.

[d]1 = Birmingham and Colman (1985); 2 = Bocsor and O'Connor (1975); 3 = Davis et al. (1968); 4 = Flavell et al. (1977); 5 = Frank et al. (1970); 6 = Gibbs et al. (1984); 7 = Hoeppel and Westerdahl (1983); 8 = Hulbert (1978); 9 = Johnsen (1980); 10 = Maier-Bode (1972); 11 = Mayack et al. (1982); 12 = Moore (1970); 13 = Moore (1975b); 14 = Neary et al. (1983); 15 = Newton et al. (1984); 16 = M. Newton (Oregon State University, personal communication, 1967); 17 = Norris (1967); 18 = Norris (1968); 19 = Norris (1969); 20 = Norris et al. (1987); 21 = Rabeni and Stanley (1979); 22 = Stanley and Trial (1980); 23 = Suffling et al. (1974); 24 = Tracy et al. (1977).

[e]Normally less than 48 h.

[f]One extreme case: 23.8 mg/kg peak concentration, 16 months to nondetection.

1980b) found that the number of live bacteria, nitrogen and phosphorus concentrations, sediment levels, and temperature all affected the persistence of 2,4-D in an Australian river.

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 that the chlorinated hydrocarbon insecticides do. Organisms exposed to phenoxy herbicides take up some of the chemical, but generally the bioaccumulation factor is low and the residence time is brief once exposure ceases (Table 7.8).

As part of a widespread survey of the Swedish environment for phenoxy herbicides, Erne (1975) reported only 3% 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). Sanborn (1974) did not detect unmetabolized 2,4-D in the components of a model aquatic–terrestrial ecosystem. 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% of samples did not contain detectable residues. Rodgers and Stalling (1972) noted that 2,4-D and its metab-

olites were rapidly eliminated from fish after exposure ceased. In Georgia, Hoeppel and Westerdahl (1983) found no 2,4-D in most samples of game fish after amine and ester formulations of 2,4-D were applied to a reservoir, although residues up to a maximum of 0.007 mg/kg were found in 18 of 20 gizzard shad. No residues were found 13 d after application.

Extensive data from Ellgehausen et al. (1980) support these findings. The lack of bioaccumulation evident in these results is consistent with the physicochemical properties of the herbicide.

Toxicity.—The toxicity of 2,4-D herbicides to fish varies; 96-h LC50s range from less than 1 to more than 400 mg/L, depending on formulation (National Research Council of Canada 1978). Most studies have incorporated static bioassays to determine lethal concentrations of the compounds, so their field applicability is somewhat limited. The test animals used in most studies have been bluegills, a species generally considered less sensitive than salmonids.

The 2,4-D dimethylamine (DMA) herbicides have relatively low toxicity to fish. Folmar (1976) reported a 96-h LC50 for rainbow trout of 100 mg/L, but he noted avoidance reactions at concentrations well below the 96-h LC50 value. Davis and Hughes (1963) and Hughes and Davis (1963) found considerable variation in the toxicity of different 2,4-D formulations to bluegills and even in the toxicity of a single formulation. The researchers believed these inconsistencies could be attributed to the different batch lots of chemical. The alkanolamine salt and the dimethylamine formulations were the least toxic formulations to bluegills; the isopropyl ester and butyl ester (not used in forestry) were the most toxic (Table 7.12). Davis and Hardcastle (1959) found differences in LC50 values for 2,4-D and other herbicides when waters from two different sources were used in toxicity tests. Results from other authors are summarized in Table 7.12.

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/L of the herbicide. Hiltibran (1967) observed that fertilized eggs of green sunfish developed normally when exposed to 1 mg/L 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-d duration of the test.

Cope et al. (1970) observed bioconcentration of the PGBE ester of 2,4-D in fish tissues 1–3 d after treatment. No detectable residues of the herbicide were found after 4 d in bluegills exposed to a 10-mg/L concentration of the PGBE ester, but histological and biochemical changes were observed in bluegills exposed to this ester at and above 5 mg/L in ponds in Oklahoma (Cope et al. 1970). The pathology included depletions of liver glycogen, globular deposits in the blood vessels, and stasis and engorgement of the brain circulatory system.

Much of the work on fish toxicity of the phenoxy herbicides has concerned the PGBE esters of 2,4-D or 2,4,5-T, but little has been done on mixtures of these compounds. Matida et al. (1975) noted no appreciable change in a stream community when a mixture of 2,4-D and 2,4,5-T as the butoxyethanol esters (commercially called "Brush Killer") was aerially spread over 9.5 hectares of forest at rates of 4.05 kg 2,4-D and 1.95 kg 2,4,5-T (active ingredient) per hectare. The authors were unable to detect the chemical in the stream during the 48-h observation period after spraying. Similarly, fishes (cherry salmon and dace

Text continues on page 235

TABLE 7.12.—Median lethal concentrations (LC50s) and no-observed-effect concentrations (NOEC) of forest chemicals for fish and invertebrates.

Chemical[a] and test species[b]	LC50 (mg/L)[c] 24 h	48 h	96 h	96-h NOEC (mg/L)	Exposure (mg/L)[c]	% mortality[d]	Source[e]
			Herbicides				
2,4-D AS							
Bluegill		800					6,19
2,4-D B, PGBE, BE							
Fish			<4.0				41
2,4-D BE							
Amphipod[f]	1.4						37
2,4-D IP							
Bluegill		0.8					6,19
2,4-D B							
Salmon				1.0	>1.0	~100	32
Bluegill		1.3					6,19
2,4-D SS							
Bluegill			66.0				41
2,4-D IO							
Salmon				1.0	1.5	Sig	32
Bluegill			160.0				41
Amphipod[f]	6.8						37
2,4-D Na borate							
Bluegill			90.0				41
2,4-D acid							
Salmon				50.0	<50.0	0[g]	32
2,4-D DM							
Coho salmon Y					200	0	25
Rainbow trout			100				10
Bluegill		166					6,19
2,4-D PGBE							
Coho salmon Fr					1.0 (96 h)[h]	26.7	32
Coho salmon Fi				<1.0			32
Cutthroat trout A				0.03	0.06, 0.124		47
Cutthroat trout Fr			0.06–1.0				45
Rainbow trout		1.1					5
Bluegill		2.1					19
Longnose killifish E		4.5					3
Amphipod[f]	2.1						37
2,4-D + 2,4,5-T							
Cherry salmon			0.6				29
Dace			1.3				29
isopod			1.6				29
2,4-D + 2,4,5-T PGBE							
Coho salmon Y					≤0.8 S	0	27
					0.21 F	0	27
Picloram							
Daphnia sp.				1.0			14
Daphnia sp.					380 (24 h)	0	26
Daphnia sp.					530 (24 h)	95	26
Stonefly N[i]	120		48				36
Picloram T							
Lake trout				<0.035			44
Amphipod[j]			0.027				20
Stonefly[k]			0.048				20

Table 7.12.—Continued.

Chemical[a] and test species[b]	LC50 (mg/L)[c] 24 h	48 h	96 h	96-H NOEC (mg/L)	Exposure (mg/L)[c]	% mortality[d]	Source[e]
Tordon 22K							
Coho salmon Y	17.5						25
Brook trout			91	69			22
Brown trout			52	22			22
Rainbow trout			58	22			22
Black bullhead			91	69			22
Bluegill			5.4				22
Fathead minnow			29	22			22
Green sunfish			91	39			22
Emerald shiner			30				22
Tordon 101							
Rainbow trout	20.0						25
Hexazinone							
Daphnia sp.			20–50				40
Fiddler crab			>1,000				40
Atrazine							
Coho salmon Y					15 (144 h)	25	25
Brook trout			6.3(4.1–9.7) F				28
Bluegill			6.0–8.0 F				28,42
					0.213	0	28
Fathead minnow			15 F				28
					0.095	0	28
Chubsucker, green sunfish, bluegill					10 (8 d)	NE	18
Triclopyr TE							
Rainbow trout, bluegill			>100				20
Shrimp			895				13
Crabs			>1,000				13
Daphnia magna		1,170					12
Oysters		56–87					13
Triclopyr BE							
Rainbow trout			0.74				7
Bluegill			0.87				7
Triclopyr U							
Rainbow trout			117				7
Bluegill			148				7
Fathead minnow			245 S				30
			120 F				30
MSMA							
Channel catfish Fi					10 (48 h)	<10	31
Amphipod[j]					100 (96 h)	0	35
Fosamine							
Coho salmon Y					200	0	25
Fosamine P							
Rainbow trout, fathead minnow			670				8
Glyphosate R							
Fathead minnow			2.3				11
Channel catfish			13				11
Amphipod[l]			43				11
Glyphosate T							
Rainbow trout			140				11
Glyphosate S							
Rainbow trout			2				11

TABLE 7.12.—Continued.

Chemical[a] and test species[b]	LC50 (mg/L)[c] 24 h	48 h	96 h	96-H NOEC (mg/L)	Exposure (mg/L)[c]	% mortality[d]	Source[e]
Dalapon							
Coho salmon			310[m]				2
Bluegill, fathead minnow		>310[n]	290[m]				38
Largemouth bass					1,000 (48 h) S	0	2
					1,000 (48 h) F	100	2
Grass carp			>30,000			0	39
Harlequin fish[o]		44					1
Emerald shiner					3,000 (72 h) S	0	23
Dinoseb							
Coho salmon Y	0.19[p]				0.06 (6 d)	93 (6 d)	25
	0.19[p]				0.06 (6 d)	100 (16 d)	25
	0.19[p]				0.04 (16 d)	94 (16 d)	25
Cutthroat trout			0.41–1.35				44
Lake trout			0.032–1.4				44
Dinoseb T							
Rainbow trout	0.30[q]						24
	0.073[r]						24
Blacknose dace	0.24[q]						24
Dinoseb BAD							
Redside shiner	0.16[s]						24
	0.24[t]						24
			Insecticides				
Malathion							
Chinook salmon			0.023				21
Coho salmon			0.101–0.17				20,27
Cutthroat trout			0.28				20
Rainbow trout			0.20				20,27
Lake trout			0.076				20
Brown trout			0.101–0.20				20,27
Fathead minnow			8.65–23				15,20,34
Walleye			0.064				20
Yellow perch			0.263				20
Bluegill			0.09–0.103				16,20,34
					0.066 (15 d)	100	9
					0.028 (54 d)	100	9
Black bullhead			12.9				20
Daphnia sp. I1		0.001–0.0018					20
Asellus sp. M			3.0				20
Amphipod[j]			0.00076				20
Isoperla sp. Y1			0.00069				20
Limnephilus sp. J			0.0013				20
Carbaryl							
Coho salmon[u]			0.764–4.34				20,21,27
Cutthroat trout			6.7–7.1				20,46
Rainbow trout[u]			1.35–1.95				21,27
Fathead minnow[u]			6.7–14.6				17,20,27
Yellow perch			5.1				20
Bluegill[u]			5.3–6.76				17,20,27
			39[v]				20
Daphnia pulex I1			0.064				20
Asellus sp. M			0.28				20
Amphipod[j] M			0.026				20
Stonefly[k] Y2			0.0048				36

TABLE 7.12.—Continued.

Chemical[a] and test species[b]	LC50 (mg/L)[c] 24 h	48 h	96 h	96-H NOEC (mg/L)	Exposure (mg/L)[c]	% mortality[d]	Source[e]
Azinphos-methyl							
Coho salmon[u]			0.0042–0.017				20,21,27
Rainbow trout[u]			0.0014–0.0043				20,21,27
Fathead minnow[u]			0.0093–0.235				17,20,27
Yellow perch			0.04[w]				20
			0.0024[x]				20
Bluegill[u]			0.0052–0.022				17,20,27
Largemouth bass[u]			0.0048–0.005				20,27
Asellus sp. M			0.021				20
Amphipod[j] M			0.00015				20
Stonefly[k] Y2			0.0019				20
Carbofuran							
Salmonids			0.164–0.560				20
Fathead minnow			0.872				20
Sheepshead minnow			0.386				33
Yellow perch			0.147				20
Acephate							
Rainbow trout			1,000				43
Goldfish			9,550				4
Plecoptera			9.5				20
Diptera L			1,000				20
Acephate T (94%)							
Rainbow trout			1,100				20
Acephate SP							
Rainbow trout			730				20
Fire retardants							
Phos-Chek							
Coho salmon			160–320				20
Rainbow trout			160–320				20
Amphipod			40–52				20
Phos-Chek 202							
Salmonids			650				20
Fathead minnow			840				20
Phos-Chek 259							
Salmonids			300				20
Bluegill			350				20

[a]AS = Alkanolamine salt; B = butyl ester; BAD = secondary butyl dinitrophenol + secondary amylbutyl dinitrophenol; BE = butoxyethanol ester; DM = dimethylamine; fosamine = fosamine ammonium; IP = isopropyl ester; IO = isooctyl ester; P = product; PGBE = propylene glycol butyl ether ester; R = Roundup; S = surfactant; SP = soluble product; SS = sodium salt; T = technical grade; TE = triethylamine salt; U = unformulated.

[b]A = alevins; E = estuarine; Fi = fingerlings; Fr = fry; I1 = first instar; J = juvenile; L = larvae; M = mature; N = nymph; Y = yearling; Y1 = first year; Y2 = second year.

[c]F = flow-through (continuous-flow) system; S = static (no-flow) system.

[d]NE = no effect; sig = significant.

[e]1 = Alabaster (1969); 2 = Bond et al. (1960); 3 = Butler (1965); 4 = Chevron (1976, Orthene technical information); 5 = Cope (1966); 6 = Davis and Hughes (1963); 7 = Dow Chemical Company (1983); 8 = Du Pont de Nemours Company (1979, unpublished); 9 = Eaton (1970); 10 = Folmar (1976); 11 = Folmar et al. (1979); 12 = Gersich et al. (1984); 13 = Ghassemi et al. (1982); 14 = Hardy (1966); 15 = Henderson and Pickering (1958); 16 = Henderson et al. (1959); 17 = Henderson et al. (1960); 18 = Hiltibran (1967); 19 = Hughes and Davis (1963); 20 = Johnson and Finley (1980); 21 = Katz (1961); 22 = Kenaga (1969); 23 = Lawrence (1962); 24 = Lipschuetz and Cooper (1961); 25 = Lorz et al. (1979); 26 = Lynn (1965); 27 = Macek and McAllister (1970); 28 = Macek et al. (1976); 29 = Matida et al. (1976); 30 = Mayes et al. (1984); 31 = McCorkle et al. (1977); 32 = Meehan et al. (1974); 33 = Parrish et al. (1977); 34 = Pickering et al. (1962); 35 = Sanders (1970); 36 = Sanders and Cope (1968); 37 = Sanders (1969); 38 = Surber and Pickering (1962); 39 = Tooby et al. (1980); 40 = U.S. Environmental Protection Agency (1982); 41 = Walker (1964a); 42 = Walker (1964b); 43 =

Table 7.12.—Continued.

Willcox and Coffey (1977, U.S. Forest Service, Pennsylvania, unpublished); 44 = Woodward (1976); 45 = D. F. Woodward (1977, U.S. Fish and Wildlife Service, personal communication); 46 = Woodward and Mauck (1980); 47 = Woodward and Mayer (1978).
[f]*Gammarus lacustris*.
[g]Except for pink salmon fry.
[h]Water hardness ranged from 10.0 to 33.6 mg/L as Ca and Mg.
[i]*Pteronarcys californica*.
[j]*Gammarus fasciatus*.
[k]*Pteronarcys* sp.
[l]*Gammarus pseudolimnaeus*.
[m]96-h median tolerance limit.
[n]48-h median tolerance limit.
[o]*Rasbora heteromorpha*.
[p]At 10°C and pH 7.
[q]At pH 8.0.
[r]At pH 6.9.
[s]Water hardness 18 mg/L; pH 7.6.
[t]Water hardness 105 mg/L; pH 8.2.
[u]Various stages or weights.
[v]Carbaryl contained in an oil dispersion, 49% active ingredient.
[w]At 7°C.
[x]Ar 22°C.

[genus not identified] fingerlings) showed no mortality or abnormal behavior, and the standing crop of invertebrates appeared to be 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 (Table 7.12). Exposures of cherry salmon fingerlings to "Brush Killer" at concentrations of 0.47 and 0.62 mg/L for 96 h caused histological changes of liver parenchyma, which the authors considered a nonspecific response to a toxic agent.

Sanders (1969) studied the effect of several 2,4-D formulations on the amphipod *Gammarus lacustris*. The butoxyethanol ester was most toxic, followed by the PGBE ester and the isooctyl ester (6.8 mg/L). The dimethylamine salt was not toxic at 100 mg/L (96 h). 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. The least toxic was 2,4-D-dimethylamine (DMA). Crayfish were less sensitive in this test than *Daphnia* sp., 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. Johnson and Finley (1980) summarized the results of studies (1965–1978) at the U.S. Fish and Wildlife Service's laboratory in Columbia, Missouri, providing a useful table of acute toxicity values for various formulations of 2,4-D applied to a variety of invertebrate and fish species.

Herbicides: 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 forms used in forestry are potassium and amine salts. It is often applied in combination with 2,4-D (Weed Science Society

of America 1989). Picloram may be applied as pellets or, more commonly, as a diluted spray mixture. Picloram may also be used in stem-injection treatments. National Forest Products Association (see footnote 3), U.S. Forest Service (1984), and Newton (1987) reviewed uses of picloram in forestry.

Behavior in the environment.—Amine and potassium salts of picloram are highly water-soluble and have negligible vapor pressure ($<10^{-6}$ mm Hg). The physicochemical properties of picloram were reviewed in detail by the National Research Council of Canada (1974) and the Weed Science Society of America (1989).

Picloram is both persistent and mobile in soil. These characteristics were reviewed in detail by House et al. (see footnote 4), Goring and Hamaker (1971), and National Research Council of Canada (1974). Norris (1970a, 1970b) noted, however, that picloram is adsorbed by organic matter and is degraded by microbial action. In forest soils, which characteristically have high organic matter and low pH, picloram is substantially less mobile and persistent than in agricultural soils.

Movement of the herbicide in soils is governed by the net water flow; maximum losses occur under warm, humid conditions, after heavy rainfall, and in light soils that are low in organic content. The leaching of picloram by rainfall is one of the major factors governing its dissipation under field conditions (National Research Council of Canada 1974). Leached picloram may be transported to aquatic ecosystems. Residues in surface runoff have reached 2 mg/L after applications of 1.1 kg/hectare (National Research Council of Canada 1974). Studies have indicated, however, that usually only small proportions (<5%) of the picloram applied to a watershed are transported in surface runoff.

Norris et al. (1976) determined the persistence and leaching 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 zones of 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 the 30-cm soil horizon was detected (relatively little herbicide was detected below 15 cm). When a layer of decaying forest litter was present, nearly all the herbicide was found in this layer. At another site, Norris et al. (1982) reported that picloram and 2,4-D disappeared from the soil within 29 months without significant leaching. 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 exceeding 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. Some of the peak concentrations are summarized in Table 7.11. Mayeux et al. (1984) studied picloram discharge from an 8-hectare watershed (Bermuda grass pasture, Texas) treated in its entirety at 1.12 kg/hectare in late April and again a year later. The maximum amount of picloram in storm-generated runoff the first year was 38 mg/m^2. The storm occurred 46 d after application. In the second year, six storm runoffs occurred 20–48 d after application; again, the highest concentration

occurred in the first (250 mg/m^2). The concentration of herbicide decreased 50 to more than 90% with travel downstream. Of the total amount of picloram applied to the watershed, 1.2% and 6% were recovered in streamflow in the first and second years, respectively. When picloram was intentionally added to flowing water in the study area, 73% remained in the water at 90 m, 16% at 1,170 m, and 0.13% at 5,400 m downstream from the point of addition. Norris et al. (1982) found a similar pattern on a hill pasture site in Oregon.

In these studies, the concentrations were highest with the first runoff events and decreased rapidly. At one site, 0.35% of the picloram applied to a 7-hectare 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 after application that were sufficient to generate streamflow. Suffling et al. (1974) found about 0.22% of the picloram applied to a Great Lakes forest opening (a powerline right-of-way) was contained in runoff water during 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 picloram (1.1 kg/hectare), 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/L) in stream samples only during the first substantial 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/hectare picloram reached 1 mg/L (National Research Council of Canada 1974). Dissipation of the herbicide in ponds appears to be rapid. One study found an initial decline of 14–18% of the picloram per day, then a decline of less than 1%/d 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 d, 7 μg/kg was detected in the pond sediments and 0.1 μg/kg picloram in the water. Dennis et al. (1977) measured picloram residues in water and sediment from ponds and streams after extensive use of the herbicide for control of woody vegetation on pastures in West Virginia. Picloram residues reached higher levels in pond water (up to 0.437 mg/L) than in streams (up to 0.011 mg/L), 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 ponds and streams. No picloram was detected in stream sediments whatever the concentrations in water.

Johnsen and Warskow (1980) injected picloram and 2,4-D into a small stream (discharge, 0.036 m^3/s) for 50 min to achieve a concentration of 6.26 mg/L picloram. The highest concentration outside the treatment zone was 2.4 mg/L at the first sampling station, 0.4 km downstream. Peak concentrations at other downstream locations were 0.94 mg/L at 0.8 km; 0.32 mg/L at 1.6 km; 0.014 mg/L at 3.2 km; and 0.001 mg/L at 6.4 km. The herbicide was not detected after 2 d. Stream water, originally containing 1,280 mg picloram/L, contained only 0.544 mg/L (a 57% reduction) after exposure to direct sunlight for 8.8 h.

The physicochemical 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 by organisms as exposure decreases. Residue analyses indicate that picloram is not bioconcentrated by aquatic invertebrates or other food-chain organisms (National Research Council of Canada 1974). *Daphnia* sp. exposed to 1 mg/L 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/L for 18 d (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-d exposure to picloram was adequate to achieve a steady-state level of accumulation in the mosquitofish. Kenaga (1980a) reported a bioconcentration factor of 31 for organisms in a flowing-water system compared to a factor of 0.02 in a static system.

Toxicity.—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%) was 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 trout and lake trout by a factor of 2. 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 with 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). The LC50s reported for this formulation are about 1 mg/L for sensitive species. Tordon 22K (potassium salt) is considerably less toxic to several fish species (Table 7.12).

Green sunfish exposed to the 99% analytical-grade picloram (1.2 mg/L) were not affected, but the technical grade or the 22% commercial formulation of picloram (for up to 1 h) caused immobilization but not death (Sergeant et al. 1970). Recovery of normal swimming response followed transfer of the fish 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 et al. (1970) 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, affect fish development and growth (Woodward 1976) and swimming response and liver histopathology (Sergeant et al. 1970). Most deaths occurred during yolk absorption, which took 4–5 d longer in picloram-treated fish.

Lorz et al. (1979) estimated the 24-h LC50 of Tordon 22K and Tordon 101 (a 4:1 mixture of 2,4-D:picloram) as 17.5 and 20 mg/L, 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 as much as 70%. Reasons for the deaths in seawater after low herbicide exposure are unknown. When coho salmon yearlings were exposed for 96 and 360 h 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

Aerial application of herbicide to control grass in a logged area recently replanted with forest seedlings.

(0.3 mg/L). In well-planned spray operations in forestry, similar concentrations (those that might cause inhibition of migration) are unlikely to occur in streams.

Herbicides: Hexazinone

Hexazinone is a relatively new forestry herbicide used selectively for site preparation and release of conifers for uninhibited growth and nonselectively for control of weeds and woody plants. The level of use has increased sharply from 2,994 kg in 1980 to 46,233 kg in 1987, when it was the most extensively used herbicide. The most common trade name is Velpar. U.S. Forest Service (1984) reviewed the uses of hexazinone in forestry.

Behavior in the environment.—In its pure form, hexazinone has a relatively low vapor pressure (6.4×10^{-5} mm Hg at 86°C, which extrapolates to 2×10^{-7} mm Hg at 25°C). Thus the potential for hexazinone to volatilize into the atmosphere is quite small. It is highly soluble in water (3.3 g/100 g water), but is substantially more soluble in a wide array of organic solvents (U.S. Forest Service 1984).

In soil, hexazinone is dissipated by photodegradation, biodegradation, and leaching. Loss from soil by volatilization is minimal, but hexazinone apparently is subject to photodegradation while the residues are confined to the soil surface. The half-lives of hexazinone in field trials are summarized in Table 7.10. Biodegradation occurs in soil under aerobic conditions, but not under anaerobic conditions. Based on studies involving radioactive herbicide, it is apparent that microbial activity, particularly fungal activity, plays a prominent role in the biological dissipation of hexazinone from soil.

Hexazinone is quite mobile. It is readily leached in laboratory soil studies and

field studies in southern forests confirm its mobility (U.S. Forest Service 1984). In Nova Scotia soils, detectable residues were found down to 45 cm, the lowest depth sampled; however, except in a sand soil, most of the recovered residues were in the top 15-cm layer (Jensen and Kimball 1987).

Neary et al. (1983) studied the off-site movement of hexazinone in four 1-hectare watersheds in the upper piedmont of Georgia after application at 1.68 kg active ingredient/hectare (10% active ingredient pellets) in April (Table 7.11). Their results show that both decomposition and leaching reduced concentrations in the forest floor and soil. By 90 d after application, however, the residue level in litter had increased to 3.42 mg/kg as foliage from treated plants fell to the forest floor. These added residues had not entered the soil when the 90-d measurements were made, but likely did so later.

Three days after hexazinone was applied to a Georgia forest, residues appeared in both storm-generated flow from ephemeral streams and baseflow in the nearest perennial stream (Neary et al. 1983; Table 7.11). All subsequent measurements were much lower, averaging 0.033 mg/L for 26 storms that produced runoff during 13 months. Flow from five of the last seven storms did not contain detectable residues. Hexazinone appeared in base flow in pulses 90–110 d after application; the peak concentration was 0.023 mg/L and subsequent pulse levels were 0.01 mg/L or less. Overall, 0.53% of the hexazinone applied was discharged from the four 1-hectare Georgia watersheds; 71% of the discharge occurred during the first storm. The amount of hexazinone discharged was 34.9% of the amount that fell directly into ephemeral stream channels. Nearly all was discharged in the dissolved phase.

Hexazinone degrades rapidly in water exposed to sunlight, and its degradation in natural waters is not greatly reduced in the presence of suspended sediments. In dark laboratory conditions, degradation was quite slow, although the test waters may not have contained many microbes. Decomposition is 4–7 times faster in natural water than in distilled water exposed to sunlight (Rhodes 1980; U.S. Forest Service 1984), indicating that photodegradation is only one means by which hexazinone decomposes.

Hexazinone is rapidly metabolized by animals and excreted in urine or eliminated in feces. It does not tend to bioaccumulate and the clearance rate from tissues of exposed animals is rapid once exposure ceases. Bluegills exposed to hexazinone for 4 weeks at concentrations up to 1.0 mg/L had hexazinone residues that reached maximum values of 2.1 mg/kg in the carcass and 6.7 mg/kg in the viscera. After 2 weeks in clean water, no hexazinone was detected in the fish (Rhodes 1980). Animals pretreated with hexazinone clear themselves of residues from subsequent hexazinone exposures more rapidly than animals not pretreated. This indicates some adaptation to more rapid metabolism and excretion as the result of the pretreatment (Rhodes and Jewell 1980). These results indicate little potential for bioaccumulation.

Toxicity.—Hexazinone in its various formulations (soluble powder, pellets, dry flowable and liquid end-use products) is practically nontoxic to aquatic invertebrates (U.S. Environmental Protection Agency 1982); LC50s or no-effect levels for invertebrates and microorganisms are above 10 mg/L (Table 7.12). Over a period of 8 months following application (16.8 kg/hectare) of hexazinone pellets to a forested watershed, there were no major alterations in the composition or

diversity of aquatic invertebrate species and no changes in the community composition of small terrestrial arthropods (Mayack et al. 1982).

Available data indicate that hexazinone is only slightly toxic to fish. The LC50s were greater than 100 mg/L in all studies reported (U.S. Forest Service 1984).

At least some aquatic plants are vulnerable to hexazinone. Algal growth, for example, was inhibited by concentrations as low as 0.5 mg/L (U.S. Environmental Protection Agency 1982).

Herbicides: Atrazine

Atrazine is one of a large group of compounds called triazine herbicides. It is widely used, at rates up to 4.48 kg/hectare, as a selective herbicide for control of broadleaf and grassy weeds in both agriculture and forestry. At higher rates of application, it can be used for nonselective control of vegetation on noncroplands. National Forest Products Association (see footnote 3), U.S. Forest Service (1984), and Newton (1987) 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/L) but substantial solubility in several organic solvents (chloroform, 52,000 mg/L; methanol, 18,000 mg/L; diethyl ether, 12,000 mg/L). Although its vapor pressure is low (3×10^{-7} mm Hg), it is reported to evaporate 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 (Table 7.10). Birk and Roadhouse (1964) reported 90% loss of atrazine from agricultural soils within 1 year. In the same study, they found that 85% of the atrazine was in the top 2.5-cm layer of soil and 5.7% was in the 2.5–5.0-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/hectare in nine consecutive years. Measurable residues of atrazine were confined to the upper 15 cm of the soil profile, and most of them were in the 0–5-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% of the applied atrazine within the first season after application. About 90% of the loss occurred within the first month after application. The magnitude, frequency, and intensity of precipitation largely determined the amount of atrazine in runoff. Runoff of water in this study ranged from 17 to 68% of the incident precipitation, resulting in loss of as much as 10,000 kg soil/hectare (silty clay loam soil, 14% slope). In two small (2.3- and 1.4-hectare) agricultural watersheds in Georgia, 0.2 and 1.9% of the atrazine applied were recovered in storm-generated runoff during the first 90 d after application (1.45 and 4.03 kg/hectare). The first runoff events were 6 and 24 d after application. Most of the atrazine recovered (83 and 99%) was in solution (Leonard et al. 1979).

Frank and Sirons (1979) monitored streams in 11 agricultural watersheds (average size, 4,279 hectares) for atrazine in both water and sediment. The herbicide or its metabolites were found in 80% of the streams; the mean concentration was 0.0014 mg/L and the peak concentration did not exceed 0.032 mg/L. About 62% of the atrazine discharge was associated with storm runoff, 21% was in baseflow, and an additional 22% resulted from chemical spills. Atrazine

was detected in 4 of 10 sets of stream-bottom sediment samples at concentrations up to 20 mg/kg.

Smith et al. (1975) analyzed water samples from irrigation ditches and basins that had been sprayed with atrazine when the 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 significant degradation of atrazine in groundwater, although the rate of degradation was slower than would be expected for the same herbicide in soil.

In a model stream ecosystem that received atrazine (0.25 mg/L) for 30 d followed by a 60-d depuration phase four times in one year, Lynch et al. (1982) found no significant accumulation of the herbicide. Residues greater than 0.1 mg/kg were found in only a few samples of substrate, and these showed no discernible pattern relative to treatment or depuration phases. Bioaccumulation factors during the treatment phase ranged from 3.5 in annelids to 480 in mayfly nymphs. Residues declined to pretreatment levels within a few days during the depuration phase. The authors noted that the sensitivity of detection was limited by the low level of initial uptake of the atrazine; however, the results are consistent with other reports of atrazine persistence in biota. Based on its physicochemical properties, atrazine would be expected to show little tendency for bioaccumulation. Boehm and Mueller (1976) noted that increasing the 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 contaminated algae were transferred into an atrazine-free medium, the herbicide was rapidly desorbed except for about 10% of the residue that apparently was 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 atrazine/L for 24 h. 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, respectively; depuration halftimes of 0.03 h, 9.5 h, and 1.5 d; and biomagnification factors of less than 10. The intensive study reported by these authors indicates that no significant bioaccumulation of atrazine will occur in aquatic environments in the forest. A mollusk accumulated atrazine to a level 3–4 times greater than the concentration in water (0.05 mg/L) during a 72-h exposure period. Most of the accumulation occurred in the first 12 h. Similar results were obtained with whitefish. Water rather than food appeared to be the major source of the herbicide for these animals (Gunkel and Streit 1980).

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

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 (Table 7.12). Parental survival, egg production, and hatchability of brook trout appeared to be unaffected by exposure to 0.72 mg/L (Macek et al. 1976). Survival and growth of brook trout fry, however, were significantly reduced after 90 d of exposure to 0.72, 0.45, and 0.24 mg atrazine/L. Analysis of muscle tissue from bluegills, fathead minnows, and brook trout indicated that these fish did not bioconcentrate detectable amounts of atrazine after prolonged exposure (Macek et al. 1976).

Walker (1964a) observed no fish mortality after application of 2.0–6.0 mg atrazine/L 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, caddisflies, leeches (Hirudinea), and gastropods (*Musculium* sp.). 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 atrazine/L 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 atrazine/L significantly reduced production of *Daphnia magna*. Development to the seventh instar of the F_1 generation of gammarids exposed to 0.14 mg atrazine/L was reduced 25% below that of animals exposed to lower concentrations and of controls.

Herbicides: Triclopyr

Triclopyr is marketed in two principal formulations: Garlon 3A, a triethylamine salt; and Garlon 4, the butoxyethyl ester. These formulations have increased substantially in use in recent years—to more than 22,000 kg in 1989 (Table 7.4)—and are most widely used for site preparation and conifer release. Rates of application range from 0.28 to 10 kg/hectare. Most aerial applications do not exceed 3.36 kg/hectare, but ground application rates may average higher; rates to more than 7 kg/hectare have been reported (U.S. Forest Service 1984).

Behavior in the environment.—Triclopyr is only moderately soluble in water (430 mg/L at 25°C), but is highly soluble in a wide array of organic solvents. Specific information on vapor pressure is lacking but, based on their structures, the amine salt and the acid form are likely to have quite low vapor pressures. The vapor pressure of the ester is likely to be higher, but is probably less than 1×10^{-4} mm Hg at 25°C. The acid form resists hydrolysis, but the ester form rapidly hydrolyzes to the acid, which then is converted to a salt at normal environmental pH (U.S. Forest Service 1984; Weed Science Society of America 1989).

Triclopyr dissipates relatively rapidly in soil, apparently by microbial activity; however, triclopyr photodegrades in water and may also in soil. The average half-life in soil is reported to be 30 d, but the half-life can be affected by soil type and other environmental conditions such as moisture, nutrients, and temperature (Table 7.10). In Sweden, triclopyr residues were reported to last more than 2 years in some cases. The reason for this unusually long persistence is not known (Torstensson and Stark 1982).

Aerial application of herbicide to control competing shrubs in a recently forested area.

Triclopyr has the potential to leach in soil, but this is minimized by its rapid dissipation by microbial and photochemical means. In soils of increasing organic matter content, mobility is decreased and dissipation is enhanced. The leaching of triclopyr and its two primary metabolites (trichloropyridinol and trichloromethoxy pyridine) was studied in six soils around the USA; only small amounts were found in the 15–30-cm and the 30–46-cm portions of the soil profile (Ghassemi et al. 1982). In a laboratory study, Choon et al. (1986) applied triclopyr as the acid or ethylene glycol butyl ether ester to packed columns of loam soil collected after duff removal from a cedar–hemlock forest in western British Columbia. Water was added at a rate of 2.5 cm every other day. After 54 d, 65% of the original amount of herbicide added to the columns was recovered as triclopyr (5%) or two metabolites (95%). Residues were found only in the top 10 cm of the column. No residues were detected in the leachate, indicating little leaching under these test conditions. The authors concluded there is little likelihood that triclopyr will leach from forest application sites into water.

There have been few studies of triclopyr entry to water in forest settings. McKellar et al. (1982) monitored triclopyr residues in streams flowing from small West Virginia watersheds that had been treated at 11.2 kg/hectare. Triclopyr concentrations in water samples collected about 61 m downstream ranged from nondetectable to 0.02 mg/L. In an Oregon hill pasture stream, Norris et al. (1987) found the maximum concentration of triclopyr to be 0.095 mg/L within 1 h after

the entire 1.74-hectare watershed had been sprayed (3.34 kg/hectare). The intermittent stream was dry during the summer months, but when fall rains recharged the stream, maximum concentrations of 0.015 mg/L were found during the first storm that generated streamflow (6 months after application). The last detectable residue occurred 4 d later. Altogether, 0.003% of the herbicide applied to this watershed was discharged in streamflow.

Photodegradation is a major reason for the disappearance of triclopyr from water; a half-life as short as 10 h has been reported (Weed Science Society of America 1989). The long-term persistence of triclopyr in water does not appear to be a significant problem in forest environments of the northwestern USA.

Toxicity.—There are not many data on the toxicity of triclopyr to invertebrates, microorganisms, or fishes; much of the available data was generated by Dow Chemical Company for its registration of the triethylamine salt (Garlon 3A). These data indicate that the triethylamine salt of triclopyr is only slightly toxic or practically nontoxic to organisms tested. Garlon 4, the butoxyethyl ester of triclopyr, is highly toxic to both rainbow trout and bluegills, whereas unformulated triclopyr is only slightly toxic to both species (Table 7.12).

Herbicides: MSMA

MSMA is a pentavalent organic arsenical herbicide. In forestry, its principal use has been stem injection for precommercial thinning and to aid in control of certain bark beetles. These uses provide only limited opportunity for MSMA to enter the aquatic environment. National Forest Products Association (see footnote 3), U.S. Forest Service (1984), and Newton (1987) reviewed the use of MSMA in forestry. Norris reported the results of a major study of the behavior and impact of 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 that 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 to 288 kg/hectare. Elemental arsenic did not increase in any plot receiving MSMA at rates less than 36 kg/hectare. The mechanisms of loss in these studies were not determined.

Dickens and Hiltbold (1967) conducted column leaching studies in which, 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[6] determined that MSMA was rapidly leached through the forest-floor material, but was not leached in the three forest soils, by 86.4 cm of water applied over a 20-d period. In tests with 2.54 cm

of undisturbed forest-floor material, as little as 2.5 cm of water delivered over an 8-d period was sufficient to move about half of the surface-applied MSMA through that 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.[6]

Norris et al. (1983) 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.

Norris[6] looked for arsenic in four streams flowing from areas that had been precommercially thinned with MSMA. Samples were collected at various intervals after treatment; special emphasis was given to storm periods when runoff might occur and to the spring runoff. Only five samples contained detectable quantities of arsenic; four of these were at the minimum level of detection, and 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 that 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 (Table 7.10). One system contained sandy loam soil as the sediment and was stocked with channel catfish and crayfish *Procambarus clarki*; the second system contained sediment, algae, daphnids, mosquitofish, and crayfish. Channel catfish showed little tendency to bioaccumulate arsenic from MSMA (the bioaccumulation factor was 4 and showed substantial reduction in bioaccumulation level after 14 d in fresh water. Crayfish showed higher levels of accumulation (bioaccumulation factors of 80–480) but also a 50% decrease in arsenic concentration after 18 d in fresh water. The second experiment was conducted similarly, and different results were obtained. Mosquitofish had bioaccumulation ratios of about 100, but crayfish showed bioaccumulation ratios of less than 10. Daphnids and algae had bioaccumulation factors of 5 and 34, 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.—Few data are available on the toxicity of MSMA to fish. The 96-h LC50 ranges from 12 to more than 100 mg/L, depending on species, test conditions, and the amount of active ingredient in the formulation tested (Midwest Research Institute 1975; Johnson and Finley 1980). Additional toxicity data for MSMA are shown in Table 7.12. Spehar et al. (1980) conducted experiments on the comparative toxicity of arsenic compounds and their accumulation in invertebrates and fish. These investigators noted that a concentration of 1 mg arsenic/L 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 amphi-

[6]Unpublished report, "The behavior and impact of organic arsenical herbicides in the forest: final report on cooperative studies," by L. A. Norris, U.S. Forest Service, Pacific Northwest Research Station, Forestry Sciences Laboratory, Corvallis, Oregon, 1974.

Backpack application of herbicide to control competing vegetation in a young forest plantation.

pods (*Gammarus pseudolimnaeus*) and *Daphnia magna* after 2 weeks of exposure or of stoneflies, snails, and rainbow trout after 28 d.

Herbicides: 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. National Forest Products Association (see footnote 3), U.S. Forest Service (1984), and Newton (1987) discussed the use of fosamine ammonium in more detail.

Behavior in the environment.—Fosamine ammonium is highly soluble in water (179 g/100 g at 25°C) but substantially less so in nonpolar organic solvents (0.02 g in 100 g *n*-hexane at 25°C). It has little vapor pressure (4×10^{-6} mm Hg at 25°C).

Fosamine ammonium is not persistent in soils; laboratory studies indicate that soil microorganisms rapidly decompose it (Han 1979a; Table 7.10). Fosamine ammonium showed only limited mobility in column leaching studies. After 56 cm of leaching water had been applied, 60–80% of the herbicide was contained in the top 10 cm of the soil column. After 1 year and 165 cm of rain in the field, 93% of the chemical present was in the top 10 cm of soil. Thus, 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 by which the chemical enters streams, the data base for 2,4-D is probably applicable. Fosamine ammonium decomposes in water. In laboratory tests at pH 5, fosamine ammonium was completely degraded in 2 weeks; the compound was quite stable in water closer to pH 7, however (Han 1979a). A strong interaction of fosamine ammonium with soil suggests it is likely to be adsorbed on suspended or bottom sediments where it enters the forest streams. Stream-bottom sediments lose fosamine in 3 months or less.[7]

Specific information on the bioaccumulation of fosamine ammonium is limited; as with other pesticides of high water solubility, however, the probability of bioaccumulation is not great. Laboratory tests have demonstrated that fosamine ammonium is not bioaccumulated. Concentrations of the herbicide in fish tissues were similar to those in water (Newton and Norgren 1977). Residues in channel catfish exposed to a 1.1-mg/L concentration of ^{14}C-carbonyl-labeled fosamine ammonium in water for 4 weeks reached a plateau in 2–3 weeks and indicated an accumulation factor of less than 1. In a separate experiment, channel catfish were placed for 4 weeks in a tank containing soil treated with ^{14}C-fosamine ammonium (15 mg/L); the system had been aged for 30 d before it was flooded and fish were exposed to the chemical. The residue levels in this group of channel catfish also reached a plateau in 2–3 weeks with an accumulation factor of less than 1. After the 4-week exposures in both experiments, the fish were transferred to fresh water for 2-week depuration periods, during which residue levels dropped 50–90%. No effects on the fish were observed during these experiments (Han 1979b). In rats, fosamine ammonium was rapidly excreted and only 0.05% of the chemical remained in the body beyond 72 h (Chrzanowski et al. 1979).

Toxicity.—McLeay and Gordon (1980) conducted partial life-cycle studies of coho salmon (egg through smolt) and rainbow trout (egg through fingerling) to assess the toxicity of Krenite (the commercial formulation of fosamine ammonium) on early life stages of fish. For both fish species, the alevin was the stage most sensitive to fosamine ammonium; 96-h LC50s (postexposure mortality was included) were 618 mg/L (coho salmon) and 367 mg/L (rainbow trout). Eggs and embryos generally were very tolerant of Krenite. Swim-up fry and young fingerlings of both species had tolerances between those of eggs and alevins. Yearling coho salmon presmolts were slightly more tolerant than coho salmon fingerlings—96-h LC50s were 7,014 and 5,361 mg/L, respectively—and coho salmon smolts were slightly more sensitive to the herbicide than presmolts. Although all tested life stages suffered some mortality after 96-h exposures to fosamine ammonium, no groups surviving previous exposure to the chemical showed any latent effects throughout the observation period in fresh water. Four-day LC50 values for swim-up fry varied 12-fold when the diluent waters varied in pH, hardness, and alkalinity; toxicity increased with increases in these variables. Overall, the acute toxicity of fosamine ammonium to salmonid fish was

[7]Unpublished data of J. Harrod, Biochemicals Department, E. I. du Pont de Nemours and Company, 1007 Market Street, Wilmington, Delaware, 1979.

2–4 orders of magnitude less than those of the brush-control herbicides 2,4-D, 2,4,5-T, silvex, picloram, amitrole, and glyphosate.

Herbicides: Glyphosate

Glyphosate is a relatively 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/hectare. National Forest Products Association (see footnote 3) and Newton (1981) discussed the use of glyphosate in more detail; Chykaliuk et al. (1981) published an extensive bibliography on this chemical.

Behavior in the environment.—Glyphosate is highly soluble in water (12,000 mg/L at 25°C) but much less so in organic solvents. It has negligible vapor pressure.

In general, glyphosate is very immobile in soil, being rapidly adsorbed by soil particles, and subject to some degree of microbial degradation. Sprankle et al. (1975a, 1975b) showed that 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 soil was reversible, phosphate ions competing for binding sites. Thus, the initial rapid inactivation of glyphosate in soil probably results from rapid adsorption rather than degradation, although some microbial degradation of the herbicide also occurs (Sprankle et al. 1975a, 1975b). The authors also showed, by thin-layer chromatography, that glyphosate is immobile in soil.

Moshier and Penner (1978) reported that the decomposition of glyphosate differed substantially among soils (Table 7.10). Rueppel et al. (1977) also found that the degree of glyphosate decomposition varied among soil types, ranging from 5 to 50% in 28 d. In two of three soils examined, 90% of the chemical was dissipated in less than 12 weeks. Aminomethylphosphonic acid (AMPA) was the only significant soil metabolite of glyphosate, and it degraded 16–35% in 60 d 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 Aamisepp (1977) and Hance (1976).

Newton et al. (1984) conducted a thorough study of glyphosate in a forest ecosystem after it was aerially applied (3.3 kg/hectare) to an 8-hectare area in the Oregon Coast Range. The study site contained two beaver ponds and a small (50 L/min) perennial stream. No buffer strips existed and the ponds and stream received direct application of herbicide. Glyphosate residues, and in many cases metabolites, were measured for 55 d after application at various depths in the canopy, on foliage, and in litter, soil, stream water, sediments, and wildlife (Tables 7.10, 7.11). Glyphosate and AMPA reached maximum concentrations of about 0.5 and 0.1 mg/L about 15 d after application. After 55 d, AMPA was no longer detectable, but glyphosate remained at about 0.1 mg/L. None of the fish collected during the 55-d study had detectable residue levels of glyphosate or AMPA (<0.05 mg/kg) despite detectable levels of glyphosate in water for at least 3 d and in the sediment for 55 d.

Glyphosate was applied to an agricultural watershed at rates of 1.10, 3.36, and 8.96 kg/hectare, and runoff from natural rainfall after treatments in early spring

was measured and analyzed to define concentration and transport (Edwards et al. 1980). The highest concentration (5.2 mg/L) was found in runoff occurring 1 d after treatment at the highest rate. Glyphosate (0.004 mg/L) 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/L for events occurring 9–10 d after application, and decreased to 0.002 mg/L within 2 months of treatment. The maximum amount transported by runoff was 1.85% of the amount applied, most of which occurred during a single storm on the day after application of the highest rate of glyphosate. In each of the 3 study years, herbicide transported in the first runoff event after treatment accounted for 99% 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 they did in the runoff water.

Most of the data on the fate of glyphosate in water come from canals in which glyphosate was used to control weeds 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/hectare to the 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 glyphosate and 0.78 mg metabolite per kilogram in the 0–10-cm layer. When glyphosate was added to flowing canal water (sufficient to achieve 150 μg/L), about 30% of the herbicide was lost in 1.6 km of travel. Thereafter, the rate of disappearance diminished; about 58% was present 8 and 14 km downstream from the introduction sites in two study canals, which implies interaction between the concentration and the mechanism of loss. Rueppel et al. (1977) reported that less than 0.02% of applied glyphosate was removed by runoff from soil after artificial rain was applied at the rate of 1.9 cm/h 1, 3, and 7 d after application of chemical.

Relatively little has been done on the bioaccumulation of glyphosate, primarily because its physicochemical properties are such that bioaccumulation is not expected to be substantial. Studies of fish metabolism demonstrated that glyphosate has a very low bioaccumulation factor (Table 7.8). No residues of glyphosate or its primary metabolite (AMPA) were detected in the fillets or eggs of rainbow trout exposed to the isopropylamine salt (Folmar et al. 1979).

Toxicity.—Folmar et al. (1979) determined the acute toxicities to four aquatic invertebrates and four species of fish of glyphosate, the isopropylamine salt of glyphosate, the formulated herbicide Roundup, and the Roundup surfactant. Technical-grade glyphosate, the active ingredient in Roundup, was less toxic than Roundup or the surfactant (Table 7.12). 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. Rainbow trout did not avoid concentrations of the isopropylamine salt up to 10.0 mg/L; mayfly nymphs avoided Roundup at concentrations of 10 mg/L, but not at 1.0 mg/L.

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 applications of 2.2, 22, and 220 kg/hectare.

Herbicides: Dalapon

Dalapon is usually formulated as the sodium and magnesium salts. In forestry it is used primarily for site preparation, conifer release, right-of-way maintenance, and grass control.

Behavior in the environment.—Dalapon and its salts are highly soluble in water (800,000 mg/L), but have little solubility in organic solvents. The acid form is relatively volatile, but dalapon is expected to exist as a salt at normal environmental acidities. The sodium and magnesium salts are not volatile; thus, volatilization of this material is unlikely in the field.

Kenaga (1974) and Foy (1975) extensively reviewed the behavior of dalapon in soil. Dalapon is highly mobile in soil because it has little affinity for soil particles in clay and clay loam soils; in muck soils, however, 20% of the dalapon may be adsorbed (Foy 1975). Laboratory studies indicate that leaching from soils should occur readily. In field tests reviewed by Kenaga (1974), however, dalapon did not leach through the soil as expected, indicating that microbial degradation may occur more rapidly than leaching. Numerous studies have indicated that dalapon is subject to microbial degradation; field persistences of less than 1 month have been commonly noted (Ashton 1982). Both dalapon and its salts undergo hydrolysis in soil, but the rates are relatively slow compared to the microbial degradation rate.

Site-specific data are not available for dalapon that may enter forest water as the result of forest vegetation management. The pattern of entry into forest streams is expected to be similar to that for 2,4-D. Dalapon will not likely adsorb strongly or extensively on sediments in aquatic systems. The primary means of inactivation in water will be microbial action, as in soil. One of the important uses of dalapon is for the control of vegetation on ditch banks. As a consequence of this use, dalapon is likely to appear in water near applications of this type. Folmar (1976, 1978) indicated that the expected dalapon concentration in water from ditch bank applications would be 0.2 mg/L.

As a result of its high water solubility and low solubility in organic solvents, dalapon shows virtually no tendency for bioaccumulation. Mammals excreted dalapon rapidly via urine (Kenaga 1974).

Toxicity.—Dalapon is only slightly toxic to fish and amphibians (Table 7.12). Fish toxicity studies of dalapon and its sodium salt formulation were reviewed by Kenaga (1974), who had access to Dow Chemical Company documentation.

Herbicides: Dinoseb

Dinoseb is a contact-action herbicide available in two forms: free phenol and amine or ammonium salt of the phenol. It was registered for use in forestry as a desiccant before lands are burned for forest-site preparation (Oregon only), but all uses are currently suspended, pending hearings by EPA. The likelihood of continued registration in forestry is remote. We include it in this chapter because of its high toxicity to aquatic species and the potential for its use in areas of important anadromous fish habitat in other countries.

Behavior in the environment.—The phenol form of dinoseb has substantial vapor pressure (0.01 mm Hg at 78°C) and is soluble to 52 mg/L in water, 23.4% in ethyl alcohol, and 8.7% 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 can volatilize from soil. Hollingsworth and Ennis (1953) showed that this process depends on ambient temperature, moisture content of the soil, and the formulation applied. Volatilization 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 3–5 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 that 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 30 cm of the acid forest soils of the northwestern USA in the first year after application. Substantial decomposition by microbial action takes place within the first year after 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 dinoseb/L for 384 h. Most fish sampled, however, did not contain detectable residues. In tests with fathead minnows, Call et al. (1984) reported a whole-body concentration factor for dinoseb of 1.4 (although if based on total radioactive carbon, the value would be about 60). When placed in clean water, fathead minnows eliminated 67% of the dinoseb in 24 h and 95% in 14 d. Rainbow trout injected with dinoseb eliminated 90% in 24 h (50% was dinoseb and the balance was in the form of metabolites).

Toxicity.—Dinoseb is more toxic to humans, animals, and fish than are most herbicides. The acute and chronic effects of dinoseb on cutthroat trout and lake trout were investigated by Woodward (1976), who found that the toxicity of a given exposure was greatly influenced by water quality. Decreasing the pH of the water increased the 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 water hardness also enhance the toxicity of dinoseb to fish, but to a lesser extent than pH (Webb as cited by Lipschuetz and Cooper 1961; Woodward 1976).

Woodward (1976) observed no cumulative mortality of lake trout and cutthroat trout chronically exposed (8–12 d) to dinoseb. Prolonged exposures of 0.005–0.010 mg dinoseb/L, however, affected yolk absorption time and fry growth. Yolk

Coho salmon fingerlings in a static bioassay to determine the acute toxicity of a forest chemical.

absorption time increased by 6–9 d over that of the controls, and fry growth was reduced at all concentrations of dinoseb tested.

Lorz et al. (1979) calculated the 24-h LC50 of dinoseb to be 0.19 mg/L for yearling coho salmon under static conditions at 10°C and pH 7.0 (Table 7.12). When survivors of this bioassay were challenged with seawater, no mortalities occurred. In a flowing-water system, the toxicity of dinoseb appeared to be greater. Releasing dinoseb-exposed coho salmon and monitoring their downstream movement showed that groups exposed to 0.040 and 0.060 mg/L for 48 h were less migratory than the controls. Yearling coho salmon exposed to 0.100 mg/L for 114 h showed extensive necrosis of the liver, kidney, and gill lamellae; however, fish exposed to 0.040 and 0.060 mg/L showed only minor degenerative changes.

Insecticides: Malathion

Malathion is an organophosphate insecticide that is extensively used in both agriculture and forestry. It has been available for use since 1959. Information on the use and effect of malathion in the forest is in two environmental impact statements (U.S. Forest Service 1977b; U.S. Animal and Plant Health Inspection Service 1980). The most recent uses of malathion on lands managed by the U.S. Forest Service have been for control of western spruce budworm and grasshoppers on western forests and ranges.

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 4×10^{-5} mm Hg (30°C) and a water solubility of 145 mg/L. It is soluble in most organic solvents, but is of limited solubility in petroleum oils.

Malathion disappears rapidly from soil, even at high application rates, probably by both chemical and biological means (Table 7.10). Both the persistence and mobility of malathion were determined at terrestrial wastewater disposal sites where the chemicals were applied (0.1 mg/L) 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/L 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).

Tracy et al. (1977) detected low or no malathion concentrations in stream water 48 h after applications of the insecticide for spruce budworm control in Washington in 1976 (Table 7.11). No residues were found in fish or benthic organisms from these streams.

Eichelberger and Lichtenberg (1971) determined the persistence of malathion in river water (Table 7.10). 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% disappearance in 180 h, and a more rapid phase accounted for more than 50% disappearance in the next 60 h. Degradation in the aquatic system would have represented both chemical degradation (the slow phase) and microbial degradation (the rapid phase). Walker (1978) reported that malathion was the shortest lived of the insecticides tested in both fresh and salt water (Table 7.10).

Malathion is expected to show little bioaccumulation. Kenaga (1980a, 1980b) predicted a bioconcentration factor of 37. Paris et al. (1975) found no measurable adsorption 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 h after they were sprayed at rates several times the normal rate used in aerial applications in forestry. Only trace amounts were found 3 d after treatment. The rapid disappearance of the insecticide from milk was attributed to its rapid excretion by the animal. The short persistence of malathion in the aquatic environment also limits its bioaccumulation.

Toxicity.—Hoffman (1957), Stavinoha et al. (1966), and Livingston (1977) all noted that organophosphate insecticides generally are short-lived in the environment, do not significantly bioaccumulate or biomagnify, and have 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 determinations of environmental persistence (Benson 1969). Although acute toxicities of these compounds to aquatic organisms are generally lower than those of the organochlorines, they vary widely (Tarzwell 1959; Pickering et al. 1962; Macek and McAllister 1970; Johnson and Finley 1980).

Toxic effects on various species have been associated with synergistic or antagonistic effects of the parent compounds and hydrolysis products. Numerous

studies have shown that cholinesterase activity is the primary locus of organophosphate attack. 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 (1967b) on the fathead minnow (Table 7.12). Reproduction and early fry survival were unaffected by the 7.4-μg/L concentration that crippled adult fish after exposure for several months.

Mulla and Mian (1981) and Mulla et al. (1981) synthesized and interpreted much of the available information on the effect of malathion and parathion on nontarget flora and fauna in aquatic ecosystems, as well as on the persistence and distribution of these chemicals in aquatic habitats. Malathion had 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 that enters surface waters in various ways, interpretation of residue concentrations is difficult because of the toxicity of a "persistent" metabolite (malaoxon) that is not easily identified in tissues. Cook et al. (1976) suggested alternative 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 two hydrolysis byproducts of malathion, which showed a pronounced synergistic effect with malathion, were 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 0.09–0.24 mg/L (the LC50s 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* (LC50, 0.003 mg/L) 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 7.12 summarizes some of the available data on malathion's acute toxicity to important invertebrate and fish species.

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 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 malathion concentrations of 0.120–0.300 mg/L showed 70–80% inhibition of acetylcholinesterase (AChE), and activity indexes were reduced by 50–70% of those 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 h caused severe damage to gill tissues and minor nonspecific liver lesions. Ponds given four semimonthly treatments up to 0.02 mg/L 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 by

Laboratory facility for flow-through, chronic toxicity tests with fish and other aquatic species.

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.

Insecticides: 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 0.5–2.24 kg/hectare, active ingredient, often in repeated spray treatments. In forestry, it is used to control defoliating insects (U.S. Forest Service 1977b). Forest application rates of more than 1.12 kg/hectare are uncommon. In fiscal year 1980, most of the carbaryl used in U.S. 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 the chemistry, toxicity, metabolism, environmental degradation, and persistence of carbaryl.

Behavior in the environment.—Carbaryl is soluble in most polar organic solvents and to about 0.01% 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 d was noted in several of their systems. Carbaryl was detected in soil and the forest floor for 64 and 128 d, respectively, after application (Willcox 1972). Other values for persistence of carbaryl are presented in Table 7.10.

LaFleur (1976) studied carbaryl movement and loss in the soil profile and its accumulation in underground water over 16 months. Rainfall during the study was 182 cm. The upper 1 m of soil contained about 6% of the applied carbaryl 16 months after application. None was found in the 10–20-cm layer after the fourth 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 groundwater, carbaryl appeared within 2 months after application and persisted through the eighth month. The maximum groundwater concentration was 0.3 μM/L at the end of the second month.

No carbaryl was detected in the field plot or in soil water at a land wastewater disposal site that received carbaryl (0.1 mg/L 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 that carbaryl will leach less than 20 cm in a soil profile that receives an annual rainfall over 150 cm.

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

Paris et al. (1975) indicated that carbaryl is 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/L or less in water about 10 cm deep may be expected to degrade completely or disappear in 1 or 2 d (Lichtenstein et al. 1966).[8,9]

Karinen et al. (1967) reported that the concentration of carbaryl in estuarine water decreased 50% in 38 d. When mud was present, more than 90% loss occurred in 10 d. The carbaryl was adsorbed where decomposition continued at a slower rate. The principal metabolite of carbaryl, 1-napthal, was less persistent. Carbaryl applied to a tidal mud flat (11.2 kg/hectare) disappeared rapidly. The initial residue level of 10.7 mg/kg decreased rapidly the first day when tidal flow 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 d after treatment to 0.1 mg/kg by day 42.

Several authors have measured peak concentrations of carbaryl in water in connection with spraying for control of the spruce budworm (Table 7.11). The rate constant (0.028 h^{-1}) reported by Stanley and Trial (1980) for carbaryl disappearance in streams was similar to the decay constants determined in the laboratory for carbaryl in river water (0.017 h^{-1}) and pond water (0.028 h^{-1}) (Eichelberger

[8]Unpublished report, "The degradation of carbaryl after surface application to a farm pond," Project Report 111A13, by R. R. Romine and R. A. Bussian, Union Carbide Corporation, Salinas, California, 1971.

[9]Unpublished report, "An investigation into the effect on fish of Sevin (carbaryl) used in rice culture," Pittman-Robertson Project W-52-R, prepared by Resource Agency, Wildlife Investigations Laboratory, California Department of Fish and Game, Sacramento, 1963.

and Lichtenberg 1971; Kanazawa 1975). Marancik[10] noted that Atlantic salmon, brook trout, and slimy sculpins did not contain detectable residues of carbaryl 24, 48, or 168 h after aerial application of 1.12 kg/hectare to forests in the eastern USA.

Bernhardt et al. (1978) conducted an intensive study of carbaryl in six streams in Washington; peak concentrations of 0.005, 0.013, 0.014, 0.020, 0.029, and 0.121 mg/L were observed. Residues typically declined from peak levels within a few hours after application. Residue levels were much lower in downstream locations. In Squilchuck Creek, the stream that received the greatest exposure, residues of 100–120 mg/kg were measured in benthic organisms, 131–152 mg/kg in cutthroat trout, and 32–335 mg/kg in sediment. Residues were not found in these ecological components at most other locations and, with the exception of sediment, were not found 30 d 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 factors of 2,000–4,000 for algae and duckweed, but values of only 1,000–5,000 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* sp., 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 d.

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 d to ^{14}C-carbaryl in the diet (2.8 mg/kg) or by bath (0.25 mg/L) produced whole-body residues of 9 and 11 μg/kg, respectively. Within 28 d, 78% of these residues were eliminated by the fish exposed via the diet, but only 11% were eliminated by fish exposed to carbaryl baths (Johnson and Finley 1980). Korn (1973) found that channel catfish did not accumulate carbaryl because they metabolize or excrete the compound. Marancik,[10] citing Tompkins (1975), reported that pumpkinseeds exposed to the commercial product Sevin at 5 mg/L for 2 h accumulated 12.6 mg carbaryl/kg in the tissue by the end of the exposure period, but eliminated 99.8% within 24 h after exposure ended. These data suggest that carbaryl bioaccumulation is limited and that its persistence is brief.

Toxicity.—Table 7.12 summarizes the toxicity of carbaryl to several invertebrate and fish species. Courtemanch and Gibbs (1980) noted short- and long-term effects of carbaryl sprayings on stream invertebrates. The initial postspray response was an increase in drift, and the benthos showed significant declines among Plecoptera, Ephemeroptera, and Trichoptera. Plecopterans did not repopulate any treated stream by 60 d after treatment. These findings are similar to those of Burdick et al. (1960), who reported a reduced standing crop of total stream invertebrates after forest spraying with Sevin. The long-term effect of the

[10]Unpublished report, "Effect of insecticides used for spruce budworm control in 1975 on fish," pages 11–34 in "1975 Cooperative Pilot Control Project of Dylox, Matacil, and Sumithion: forest spruce budworm control in Maine," by J. Marancik, U.S. Forest Service, State and Private Forestry, Northeastern Area, Upper Darby, Pennsylvania, 1976.

chemical was most apparent on plecopterans, especially in streams treated for 2 consecutive years.

Following the aerial application of carbaryl (0.84 kg/hectare) for control of spruce budworm in Maine, Gibbs et al. (1984) observed woodland ponds for 30 months. The most severe and persistent effects were on amphipods; *Hyallela azteca* and *Crangonyx richmondensis* were reduced to near $0/m^2$ and they failed to recolonize in some of the ponds 30 months after treatment. Numbers of immature Ephemeroptera and Trichoptera were reduced immediately following spray application but this effect did not persist throughout the season or into the following year. Numbers of immature Odonata were reduced following treatment and remained low during the following year. Chironomids did not appear to be affected either as immatures or emerging adults.

Stewart et al. (1967) studied the acute effects of carbaryl and its hydrolytic product 1-naphthal 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 1-naphthal. Carlson (1972) found that long-term exposure of fathead minnows to carbaryl at a concentration of 0.68 mg/L 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 increases in circulatory anomalies were produced at concentrations of 5 mg carbaryl and 20 mg malathion per liter.

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

> Little or no alteration in toxicity resulted when temperatures were increased from 10°C to 21°C for daphnids or from 7°C to 17°C 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 8.5-9.0) were 1.4–11.4 times more toxic to trout, salmon, and yellow perch than were test solutions with lower pH (6.5–7.5). Variations in hardness (12–300 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.

Insecticides: 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 to control seed and cone insects in seed production areas. Because of this pattern of use, the chemical is unlikely to enter aquatic systems and contaminate aquatic organisms.

Coho salmon fingerlings from a test of chronic chemical toxicity. The top fish, a control, was unaffected. The middle and bottom fish show the effects of increasing toxicant concentration on growth.

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 about forestry uses can be drawn from the agricultural experience. Azinphos-methyl is soluble to 29 mg/L in water (25°C) and is readily soluble in most organic solvents (except aliphatics).

The Chemagro Division of BayChem Corporation conducted soil persistence studies of azinphos-methyl (Anderson et al. 1974; Table 7.10). The average half-life of the compound was reported to be about 3 months, although it varied substantially in different soil types and in different geographic locations. Haque and Freed (1974) estimated that azinphos-methyl would leach less than 20 cm in soils receiving 150 cm of rainfall. Results of these tests suggest that the persistence of azinphos-methyl and its mobility are not sufficient to result in either buildup of the compound in the soil or its transfer into groundwater.

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 soil in the southeastern USA. Its

predominant pattern of use in forestry minimizes the likelihood that this insecticide will enter forest surface waters in western North America.

Meyer (1965) reported that the half-life of azinphos-methyl would be about 2 d in the aquatic environment (Table 7.10). Flint et al.[11] reported a half-life of 1.2 d 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 that azinphos-methyl is subject to photodecomposition in aquatic systems. These tests suggest that the decomposition of azinphos-methyl in an aquatic environment is relatively rapid and that accumulation is not to be expected.

Azinphos-methyl should show little potential for bioaccumulation. Dairy cattle appear to excrete it rapidly (Everett et al. 1966; Loeffler et al. 1966). No residues were found in milk 1–2 d after treated feed was withdrawn.

Toxicity.—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; Table 7.12). It is 2–10 times more toxic than malathion.

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 (Table 7.12). Variations in water hardness from 12 to 300 mg/L produced no change in toxicity to scuds or fish. Alkaline solutions (pH 8.5–9.0) were slightly less toxic to fish than more acidic solutions (pH 6.5–7.5). Aqueous degradation from 1 to 3 weeks produced a 1.3- to 2-fold increase in 96-h LC50s for Atlantic salmon and yellow perch. Atlantic salmon eggs were highly tolerant of the chemical (11-d LC50 > 50 mg/L). The susceptibility of yolk-sac fry equaled that of fingerlings. Time-independent LC50s (TILC50) were 0.00023, 0.00029, and 0.00032 mg/L for Atlantic salmon, bluegills, and yellow perch, respectively. The TILC50 is a statistical estimate of the toxicant concentration at which 50% of the test population would be expected to survive in 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-h LC50 to the TILC50 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 0.00051 mg azinphos-methyl/L, but not 0.00033 mg/L, drastically reduced egg production by fathead minnows, but caused no other apparent adverse effects.

Insecticides: 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. It is used in forestry to control seed and cone insects in nurseries and seed orchards and as a root dip at time of planting (see footnote 3).

[11]Unpublished report, "Soil runoff, leaching, and adsorption and water stability studies with Guthion," Report 28936, by D. R. Flint, D. D. Church, H. R. Shaw, and J. Armour, Chemagro Corporation, Kansas City, Missouri, 1970.

Behavior in the environment.—Carbofuran is soluble to 700 mg/L in water (25°C) and has a vapor pressure of 2×10^{-5} mm Hg (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 (Table 7.10). Goring et al. (1975) included carbofuran in the group of pesticides that is "moderately persistent in soil" (half-lives of 1.5–6 months). Sanborn (1974) did not detect a bioaccumulation of carbofuran in a multicomponent model ecosystem, although each component gave evidence that carbofuran became tightly bound and underwent substantial degradation. Additional specific data on this chemical are lacking, but carbofuran 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 USA. The way it is used in forestry minimizes the likelihood that carbofuran will enter forest surface waters in the western USA.

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 (Table 7.12). Adult sheepshead minnows exposed to concentrations of 0.049 mg/L or more showed significantly greater mortality than control fish during a 131-d study (Parrish et al. 1977). Hatching success of eggs spawned by fish exposed to 0.049 mg/L was significantly less than that of eggs of unexposed fish. Mortality of fry hatched from eggs spawned by fish exposed to 0.23 and 0.049 mg/L was significantly greater than control fry mortality. Davey et al. (1976) noted that carbofuran was the least toxic of five rice-field pesticides to 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/L, but observed no adverse effects.

Insecticides: 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, where it is applied at a rate of 1.5 kg/hectare (see footnote 3).

Behavior in the environment.—Willcox and Coffey[12] summarized the behavior and the toxicity of acephate. It is degraded in soil by microbial action. Chevron[13] reported that, in soils from nine locations across the USA, acephate had a half-life ranging from 0.5 to 13 d when the soil was fortified to 1 or 10 mg/kg. The longest persistence was in a highly organic muck soil; in the other eight soils, the half-life ranged from 0.5 to 4 d. Other persistence values are shown in Table 7.10.

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[12]Unpublished report, "Environmental impact of acephate insecticide (Orthene)," by H. Willcox III and T. Coffey, Jr., U. S. Forest Service, State and Private Forestry, Forest Insect and Disease Management, Northeastern Area, Upper Darby, Pennsylvania, 1977.

[13]Unpublished report, "The impact of Orthene on the environment," prepared by Chevron Chemical Company, Richmond, California, 1973.

In laboratory studies, acephate (freshly added) was readily leached in soil. Aged soil residues were much less mobile. The short persistence of acephate in biologically active soils is believed to minimize the likelihood of significant movement to groundwater. According to Chevron, acephate is hydrolyzed slowly in water (half-life at 21°C: 55 d at pH 5.0, 46 d at pH 7.0, and 16 d 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 also adsorb acephate, but the residue levels decline rapidly.

Flavell et al. (1977) summarized the aquatic data collected during pilot-scale applications of acephate to control the western spruce budworm in three 405-hectare blocks in Montana in 1976 (Table 7.11). Concentrations decreased rapidly, typically to 10% of initial values in 2–6 h. Residues averaged 0.065 mg/kg (range, 0.026–0.139 mg/kg) in fish and 0.036 mg/kg (0.0–0.107 mg/kg) in insects.

Sanborn (1974) reported that acephate did not accumulate in algae, clams, crabs, *Daphnia* sp., *Elodea* sp., mosquitofish, or snails in a model ecosystem that had both terrestrial and aquatic components. The acephate was applied at a rate of 1.12 kg/hectare to the terrestrial portion of the system. The data also indicated more than 95% decomposition of acephate in the system in 33 d.

Bluegills were continuously exposed to 1.0- or 0.01-mg/L concentrations of ^{14}C-labeled acephate for 35 d, and tissue samples were 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 d.[14] The maximum tissue concentration of 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% of the residues in the edible flesh within 3 d. These data indicate a low potential for bioaccumulation.

Toxicity.—The effects on stream fishes and invertebrates of an operational acephate spraying to suppress spruce budworm were investigated by Rabeni and Stanley (1979). Acephate reached its maximum concentration of 0.14 mg/L in North Brook and 0.113 mg/L in South Brook, Maine, within 1 h of spraying, and residues remained in stream water for at least 2 d. 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 acetylcholinesterase activity was depressed in suckers but not in trout or salmon, and brook trout altered their diet but their 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 12) summarized the pertinent literature on environmental effects of acephate insecticide. Acephate has an extremely low toxicity to fish (Table 7.12); although it is more toxic to invertebrates, no effects on Plecoptera or Ephemeroptera in a Pennsylvania stream and pond were recognized after a treatment of 0.56 kg (active ingredient)/hectare.

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

Woodward and Mauck (1980) suggested that acephate would be the most acceptable of five forest insecticides tested from the standpoint of its effects 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 water after a spraying operation.

Insecticides: Bacillus thuringiensis

Bacillus thuringiensis (*B.t.*) is a naturally occurring bacterial insecticide first registered in the USA in 1961. It has found broad usage in agriculture and forestry and for mosquito control. It is currently registered for terrestrial food and nonfood crops, greenhouse food crops, forestry, and indoor uses.

Behavior in the environment.—Most of the environmental studies with *B.t.* have focused on the persistence of the material as it affects efficacy. On foliage and probably the surface soil, *B.t.* is rapidly inactivated by sunlight. The rate of inactivation varies from test to test; factors such as humidity, rainfall, and plant species are influential (Table 7.10).

Spores of *B.t.* germinated, grew, and sporulated in soil of neutral pH to which alfalfa or casein had been added. The number of viable spores increased 100-fold. In more acid soils, the spores germinated but the vegetative cells did not survive. It appears *B.t.* spores can remain viable for a long time in soil, and that the organism can compete successfully under conditions favoring the bacillus component of the microbial populations (Saleh et al. 1970; Petras and Casida 1985).

Field and laboratory studies have also examined the persistence of *B.t.* in water. Following aerial application of *B.t.* in eastern Canada to help control eastern spruce budworm, *B.t.* was recovered from rivers and public water distribution systems. Laboratory tests indicate that *B.t.* can survive for extended periods of time in both fresh and marine water at 20°C. The field tests did not reveal detectable quantities of the organism in oysters or clams, even though the water tested positive (Menon and De Mestral 1985).

Bacillus thuringiensis is ubiquitous in the natural environment. For this reason, and because toxicity tests show virtually no effect on most other organisms, little work has been done on the movement, persistence, and fate of *B.t.* for purposes of estimating exposure variables. The U.S. Environmental Protection Agency has no data on the environmental fate of *B.t.* but does not require them, probably because this material is not toxic to most nontarget species (U.S. Environmental Protection Agency 1988).

Toxicity.—Few toxic effects have been reported in studies of aquatic species exposed to *B.t.* A static bioassay with Dipel, a formulated product containing 3.2% *B.t.* variety *kurstaki*, suggested possible toxicity to mussels and brine shrimp. The LC50 for brine shrimp was 85 mg/L, but it was uncertain whether the deaths were caused by the microbe or other factors.

Toxicity studies on *B.t.* variety *israeliensis* were conducted by ToxiGenics for Abbott Laboratories. Rainbow trout and bluegills were subjected in static bioassays to concentrations of 300–370 mg/L (rainbow trout) and 300–600 mg/L (bluegills). One rainbow trout died between 72 and 96 h after exposure to 370 mg/L. Five of 30 bluegills subjected to 300 mg/L died within 96 h, as did 7 of 30 bluegills subjected to 600 mg/L. The LC50s were not calculated by ToxiGenics

(Study 410-0561 and Study 410-0563, Attachments 17 and 18 in U.S. Environmental Protection Agency 1988).

Insecticides: Nuclear Polyhedrosis Virus

Nuclear polyhedrosis virus (NPV) is a biological insecticide. Its use in forestry has been developed specifically for the control of several insects, including European pine sawfly *Neodiprion sertifer*, spruce budworm, and Douglas-fir tussock moth *Orgyia pseudotsugata*. The active ingredient is a nuclear polyhedrosis virus whose infection particles or virons are randomly occluded in an orthagonal crystalline matrix called polyhedral inclusion bodies, or PIBs. The dosage rate of NPV is usually expressed in PIBs per unit area (hectare or acre). A specific NPV is produced for each target organism. For instance, the NPV for the Douglas-fir tussock moth is isolated from millions of tussock moth larvae that have been infected with the virus under closely controlled conditions. The virus is purified, stored, and (when needed) formulated into a material that can be easily applied to the forest.

Behavior in the environment.—Little attention has been given to the movement, persistence, and fate of NPV in forest environments. Active NPV introduced into the forest floor undergoes little vertical movement in the soil, but remains active for at least 11 years. The NPV produced from early instars of insect hosts, however, appears to be largely inactivated before it reaches the forest floor (Thompson and Scott 1979). Jaques (1969) found that the abundance of NPV developed for cabbage looper *Trichoplusia ni* had not decreased significantly 231 weeks after it was applied to the soil, but little virus was detected at depths greater than 7.5 cm; this suggests that the viruses are unlikely to move into groundwater.

Exposure data developed in the traditional way are meaningless because NPV is part of the normal environment. Naturally occurring NPV can persist for up to 41 years after an epizootic of the disease. Concentrations typically are low (<45 PIB/cm^3), but they are sufficient in sheltered locations to infect tussock moth larvae (Thompson et al. 1981). Laboratory tests have shown that NPV is virtually nontoxic and nonpathogenic to mammals, birds, fish, and other nontarget organisms, indicating the highly specific action of this biological insecticide.

Toxicity.—Bluegills and rainbow trout showed no adverse effects when exposed to high doses of PIB. Freshwater crayfish showed no adverse effects when similarly exposed (MicroGeneSystem 1985).

Buckner et al. (1975) conducted an extensive study of the effects of NPV on a wide array of nontarget organisms in the forest. In this study, NPV was applied (247.5×10^9 PIB/hectare) to 160 hectares of forest on Manitoulin Island, Ontario, to control eastern spruce budworm. The area was surveyed for effects on songbirds, small mammals, honey bees, and many aquatic species. No immediate or short-term effects on any of these organisms were found.

Fertilizers

Nitrogen (N), as urea, is the element most commonly applied as a forest fertilizer in the northwestern USA. Application rates vary, but are usually 168–224 kg urea-N/hectare (Moore and Norris 1974). Bengtson (1979) reviewed the use of fertilizers in forestry.

Aerial application of urea fertilizer.

Behavior in the environment.—Urea is highly soluble in water and is readily moved from surface deposits into the forest floor and soil. Hydrolysis to ammonium ion is usually complete in 2 weeks. Ammonium ions may be adsorbed by humic substances, held as exchangeable cations, incorporated by soil microorganisms, or taken up by forest vegetation. In addition, there is evidence for ammonia volatilization, which can be appreciable in some cases (Derome 1979, 1980; Marshall and DeBell 1980). Usually, the nitrogen is quickly distributed through the biomass and is cycled within the forest ecosystem (Moore and Norris 1974). Pang and McCullough (1982) monitored nutrient distribution in the forest floor and in soil over a 31-month period after urea fertilizer was applied (448 kg N/hectare) to a Douglas-fir forest. The increase in nutrient concentration (sampled with tension lysimeters) was greatest in the forest floor; concentrations up to 200 mg N/L persisted 5 months later, compared to 0.5 mg/L in untreated stands. There was no appreciable difference in nutrient levels between the forest floor and 10- and 30-cm soil depths in the fertilized stand. When the forest was thinned as well as fertilized, however, the concentration of nitrogen was about the same in the forest floor but increased to 80–100 mg/L at 10- and 30-cm depths in the soil. This illustrates the importance of vegetation density in the capture and cycling of nitrogen added to forest ecosystems.

Fertilizer nitrogen enters aquatic environments by the same routes described for pesticides. The highest concentrations of urea result from direct application to stream surfaces. Urea transformation products are mobilized in ephemeral stream channels and move through subsurface drainage networks to perennial streams.

Forest soils filter out plant nutrients very efficiently, but increased levels of

TABLE 7.13.—Nitrogen lost from treated watershed 2 during the first year after application of 224 kg urea-N/hectare and from untreated watershed 4 during the same period, South Umpqua Experimental Forest, Oregon. (From Moore 1971.)

Loss locus or statistic	Urea-N	NH_3-N	NO_3-N	Total
	Absolute loss (kg/hectare)			
Watershed 2 (treated)	0.65	0.28	27.09	28.02
Watershed 4 (untreated)	0.02	0.06	2.07	2.15
Net loss (2 − 4)	0.63	0.22	25.02	25.87
	Proportional loss			
Percent of total	2.44	0.85	96.71	100.00

various nitrogen species have been measured in several forest stream systems in the northwestern USA. 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 urea-N/hectare to 68 hectares of a southwestern Oregon forest (Table 7.11). Only 0.01% of the nitrogen applied to the watershed was found in streams up to 15 weeks after application. Over the next 24 weeks during the summer and fall, precipitation and hence streamflows were low and essentially no applied nitrogen was lost. November storms brought the soil moisture back to maximum storage capacity, and stream concentrations of nitrate-N reached a second peak of 0.177 mg/L in December. Both streamflow and nitrate-N levels remained high through December and January, during which time 23.8 kg of applied nitrogen were lost. This 2-month washout accounted for 92% of the total amount of fertilizer nitrogen lost during the first year—25.9 kg (Table 7.13). Over the same period, the total amount of soluble inorganic nitrogen lost from the 49-hectare control wastershed was 2.15 kg. Stream data on soluble organic nitrogen, total phosphorus, silica, and exchangeable cations (sodium, potassium, calcium, magnesium, iron, manganese, and aluminum) indicate that nitrogen fertilization did not accelerate losses of native soil nitrogen and other plant nutrients.

Similar data were reported by Moore (1975a, 1975b) for several other monitoring studies conducted throughout the Douglas-fir region. In one study, the concentrations of nitrogen after forest fertilization were determined in 29 streams in the northwestern USA and Alaska (Moore 1975b). The most extreme values from that study are shown in Table 7.11. Increases in the concentration of urea-N ranged from very low to a high of 44.4 mg/L. These increases resulted almost entirely from direct applications to surface water, and the peak concentrations reached were directly proportional to the amount of open surface water in the treated units. The high peak concentrations of urea-N measured in Dollar Creek were associated with the spring runoff of snowmelt.

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 3–5 d after application, urea-N in the streams returned to pretreatment concentrations.

Increases in ammonium-N levels also resulted from direct applications of urea fertilizer to open water. Urea is readily hydrolyzed to ammonium-N in the stream system. Urea applied to the forest floor and to soil surfaces does not reach streams

because it hydrolyzes rapidly to ammonium carbonate and is then held on cation-exchange sites in the soil and the 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/L in a tributary stream of the Elochoman River. The concentration of nitrate-N in stream samples usually reaches a peak 2–4 d after spring applications of fertilizer. Concentrations then decrease, but may remain above background levels for 6–8 weeks. Losses of applied nitrogen are very small because the maximum concentrations of nitrate-N are generally less than 1 mg/L, 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 d is from direct application and is measured as urea- and ammonium-N. The other half enters as nitrate. In the early fertilization projects, stream buffer strips were either very narrow or not used, and estimated total losses were 2–3% of the applied nitrogen. In later projects, however, direct application to open surface water was minimized by buffer strips along the main streams and tributaries, and measured losses were less than 0.5%.

When monitoring studies have 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 storms. 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 applications of fertilizer (September, October) are similar to those after spring applications. Peak concentrations measured during winter storms may not be as high, however, because shorter periods of warm weather mean less nitrogen is converted to nitrate. The initial peak in nitrate-N concentration after a fall fertilization occurs 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.

Hetherington (1985) reported that peak nitrogen concentrations in two small streams were 14 mg/L as urea, 1.9 mg/L as ammonia, and 9.3 mg/L as nitrate within the first 60 d after an early-September application. These values are consistent with the range of concentrations reported by Moore (1975b). However, the total amounts of nitrogen discharged from the study watersheds (228 hectares, 46% fertilized with urea at 224 kg N/hectare; and 78 hectares, 80% fertilized) were 5.9% and 14.5%, respectively, of the amounts applied, values that are substantially higher than the losses of about 1% summarized by Moore (1975b). From 53 to 61% of the discharge occurred in November, the third month after application; 92–98% of the nitrogen was discharged as nitrate. Hetherington concluded that fertilization did not lower water quality below drinking water standards or endanger fish, but he cautioned against direct applications of fertilizer to stream channels, open water, or swampy areas.

Toxicity.—Ammonia is one of the toxic breakdown products of fertilizers. U.S. Environmental Protection Agency (1976) summarized its toxic characteristics as follows.

> 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.

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 NH_3 for rainbow trout . . . (Liebmann, 1960). The concentration for Atlantic salmon smolts . . . (Herbert and Shurben, 1965) and for rainbow trout (Ball, 1967) was found to be only slightly higher. Although a concentration of NH_3 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 . . . 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 established a recommended standard for nitrate but not a mandatory one because nitrate has long been considered almost nontoxic to fish. Westin (1974) reported a 96-h medium tolerance limit (TLm) of 5,800 mg nitrate/L for chinook salmon fingerlings and 6,000 mg/L for rainbow trout fingerlings. Few data are available on other life stages, but Kincheloe et al. (1979) found that sodium nitrate was mildly toxic to the early life stages of several salmonids. Coho salmon eggs and fry were resistant to nitrate toxicity. Eggs and fry of chinook salmon, rainbow trout, steelhead, and Lahontan cutthroat trout exhibited mortalities during exposure to nitrate concentrations as low as 5 mg/L. A complication was that eggs were infested with the fungus *Saprolegnia* sp. The authors believed that nitrate levels of 10 mg/L (2 mg nitrate-N/L) 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 increase the productivity of fish ponds (Swingle 1947; Boyd and Sowles 1978). These fertilizers can lower the alkalinity of water (Hunt and Boyd 1981), so fertilized ponds may have to be limed to neutralize the acidity.

Stay et al. (1979) studied the effects of fertilizing a second-growth Douglas-fir forest with 224 kg urea-N/hectare. Although they found sharp increases of urea in a stream during fertilization because of direct application, all nitrogen forms

Aerial application of fertilizer to enhance the growth of young seedlings planted in a recently harvested area.

returned to near background levels shortly afterward. A 2-month rainbow trout bioassay showed no deaths 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 complex mixtures. The most abundant constituent (responsible for the fire-retarding 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 7.14, 7.15). 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 stimulation 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). One analysis, however, suggested that photolysis of the ferrocyanide in several Fire-Trol retardant formulations may yield sufficient cyanide to be the primary toxicant in these products.[15]

Behavior in the environment.—The behavior of ammonium and ammonia in the environment was described in the previous section on urea fertilizer. Fire-Trol

[15]Unpublished draft environmental assessment report, "Toxicity and environmental effects of fire retardant chemicals," prepared by U.S. Forest Service, Pacific Northwest Region, Portland, Oregon, 1979.

TABLE 7.14.—Typical composition of some chemical fire retardants.[a] Empty cells mean information is unavailable.

Retardant and constituent	Empirical formula	% by weight in dry powder or liquid concentration	mg/L in mixed retardant
Phos-Chek XAR,[b] Monsanto			
Diammonium phosphate	$(NH_4)_2HPO_4$	85–90	$1.02–1.08 \times 10^5$
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}	0.25–0.58	300–700
Thiosulfate	S_2O_3	0.01–5	1,200–6,000
2-Mercapto-benzothiazole	$C_6H_4SCSH:N$	0.0005–2	600–2,400
Flow conditioner (insoluble)		2–4	2,400–4,800
Phos-Chek 259R (0.14 kg/L), Monsanto			
Diammonium phosphate	$(NH_4)_2HPO_4$	92	111,000
Modified polysaccharide		2.5	3,000
Iron oxide	Fe_2O_3	0.75	902
Corrosion inhibitors: soluble salt of			
Silicofluoride	SiF_6^{-2}	1.47	1,768
Thiosulfate	S_2O_3	0.71	854
2-Mercapto-benzothiazole	$C_6H_4SCSH:N$	0.20	241
Flow conditioner (insoluble)		2.0	2,405
Phos-Chek 259R (0.19 kg/L), Monsanto			
Diammonium phosphate	$(NH_4)_2HPO_4$	92	148,000
Modified polysaccharide		2.5	4,024
Iron oxide	Fe_2O_3	0.75	1,207
Corrosion inhibitors: soluble salt of			
Silicofluoride	SiF_6^{-2}	1.47	2,366
Thisosulfate	S_2O_3	0.71	1,143
2-Mercapto-benzothiazole	$C_6H_4SCSH:N$	0.20	322
Flow conditioner (insoluble)		2.0	3,219
Fire-Trol 100,[c] 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,[d] 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[e]		—	—
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[e]			

[a]From Chemical Economics Handbook, January 1978, Menlo Park, California, Phosphorus Products, page L.

[b]U.S. Patent 3,024,100 (March 6, 1962), 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.

[c]U.S. Patent 3,196,108 (July 20, 1965), Fire Supressing Composition for Aerial Application, granted to Nelson and assigned to Arizona Agrochemical Corporation (now Chemical Industries).

[d]U.S. Patent 3,960,735 (June 1, 1976), Corrosion-Inhibited Polyphosphate Compositions, granted to Lacey and assigned to Early California Industries, Inc.

[e]The formulation and concentration of these compounds were furnished by Chemonics and are not included because of their proprietary natures.

TABLE 7.15.—Concentration of specific ions in some chemical fire retardants (estimated from data in Table 7.14).[a]

Retardant and specific ion	Formula	mg/L in mixed retardant
Phos-Chek XA, Monsanto		
Ammonium + ammonia[b]	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 (0.14 kg/L), Monsanto		
Ammonium + ammonia[b]	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 (0.19 kg/L), Monsanto		
Ammonium + ammonia[b]	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[b]	NH_3	43,600
Sulfate	SO_4^{-2}	122,900
Dichromate	$Cr_2O_7^{-2}$	2,500
Fire-Trol 931L, Chemonics		
Ammonium + ammonia[b]	NH_3	30,300
Phosphate	PO_4^{-3}	113,300
Ferrocyanide	$Fe(CN)_6^{-4}$	1,800–3,800
Fire-Trol 934L, Chemonics		
Ammonium + ammonia[b]	NH_3	31,370
Phosphate	PO_4^{-3}	117,000
Ferrocyanide	$Fe(CN)_6^{-4}$	2,720

[a]From Table 2, "Draft fire retardant environmental assessment," U.S. Forest Service, Pacific Northwest Region, Portland, Oregon, undated.

[b]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. Forest Service, Pacific Northwest Forest and Range Experiment Station, Forestry Sciences Laboratory, Corvallis, Oregon, 1978.

931L and 934L are ammonium-based fire retardants, but they contain ferrocyanide as a corrosion inhibitor. According to Burdick and Lipschuetz (1950, quoting Baudisch and Bass 1922), ferrocyanide solutions "are decomposable to some extent under the influence of light" (sunlight). The product of photolysis is cyanide:

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

The CN^- then reacts with water:

$$CN^- + H_2O \rightleftharpoons HCN + OH^-;$$

the equilibrium reaction is strongly pH dependent. The CN^- ion 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 is 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 19.3 km of river and was associated with an industrial discharge of ferrocyanides and ferricyanides (Burdick and Lipschuetz 1950). Investigators showed that the ferrocyanide and ferricyanide concentrations were below those generally accepted as lethal.

Studies of ferrocyanide conversion to cyanide were carried out by Burdick and Lipschuetz (1950) in open vessels exposed to sunlight during May and October. Initial potassium ferrocyanide concentrations ranged from 1 to 100 mg/L, and exposure time was 1–5 h. Results were inconsistent, which was attributed to varying light intensity and temperature. The highest percentage conversions to cyanide—up to 25%—occurred at the low initial concentrations of potassium ferrocyanide (1–5 mg/L).

In a more closely controlled experiment with 1-, 2-, and 3-mg/L concentrations of potassium ferrocyanide, the conversions ranged from 10 to 15% in 1 h, after which cyanide values decreased. The decrease was attributed to loss of HCN and recombination of reaction products. In any event, although the percentage conversions of potassium ferrocyanide to cyanide vary, the maximum value is about 25%.

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

- fire-retardant mixtures contain up to 5,400 mg $Na_4Fe(CN)_6$/L (equivalent to 3,800 mg $Fe(CN)_6^{-4}$/L);
- an air drop covers an area 75 m by 20 m and the rate of deposition is 2 L/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; and
- retardant mixes instantaneously in the stream.

Based on these assumptions, the instantaneous stream concentration of $Fe(CN)_6^{-4}$ (before it is diluted by normal flow) would be 38 mg/L. If 25% of the ferrocyanide were photolyzed to cyanide and 90% of the cyanide occurred as HCN, the instantaneous HCN concentration would be more than 8 mg/L.

With time, HCN disappears from water, as indicated by the reports of Burdick and Lipschuetz (1950) 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 heavy metals in the "d" block of the periodic table. Such metals typically are more abundant in lowland streams than upland forest waters. Reports of cyanide degradation in water are lacking; however, 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 from fire retardants is deposited in streams, some cyanide will be produced through photolysis. The concentration will 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 it volatilizes, becomes diluted, and forms complexes with metals.

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

The retardant was applied across streams at four western locations. Direct application of retardant to the surface of the stream produced detectable changes in water chemistry for distances as far as 1,000 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, described later in this section.)

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 virtually eliminated movement of retardant from the land to the stream, but the edge of the treated area was only 3 m from the stream at several points.

The principal chemicals that were elevated in the stream within the first 24 h after application were ammonium-nitrogen and total phosphorus. Ammonia is potentially toxic to aquatic species and phosphorus may contribute to downstream eutrophication. After 24 h, 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 next to streams can cause nitrogen to enter the streams in measurable quantities and in a form toxic to fish. The probability that toxic levels will occur is low, however, and can be further minimized if ammonium-based fire retardants are not used on shallow, rocky, poorly developed soils on steep slopes that drain directly into stream channels.

The computerized simulation studies used a combination of real and generated data (1) to develop methods for predicting the amount (concentration) of retardant in streams at the time it is directly applied to stream surfaces, (2) to develop methods for describing the dispersal of retardant in a stream, and (3) to integrate these techniques with data on retardant toxicity to evaluate the effects of various types of retardant application on fish mortality. These simulations suggested that (1) direct application of retardant to streams is likely to cause fish mortality, and that (2) the magnitude of the mortality and the distance over which it occurs vary with characteristics of the application, the site, and the streamflow.

Characteristics of the application (for a constant pattern of distribution) include orientation of the line of flight to the stream, size of each load dropped, number of loads dropped, and the timing and placement of subsequent loads relative to the first load. For instance, a much smaller zone of mortality results when the flight path is perpendicular to a stream than when it is centered on the stream's axis. 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 of the timing and placement of subsequent loads on the mortality zone, but we believe that the effects of sequential loads are at least additive. 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 effects on the stream.

Characteristics of the site include the width and depth of the stream and the density of overstream vegetation (leaf-area index). The simulation suggested that narrow, deep streams have a much shorter mortality zone than shallow, wide streams (for equivalent flow properties). 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 determine the degree and speed with which retardant is mixed and diluted as it travels downstream. For streams of roughly equal gradient (steepness), the simulations showed 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 groundwater inflows and contributions from side streams. Increased stream discharge dilutes the retardant. Managers can recognize streamflow characteristics and take them 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 showed varying results, mainly because of the multitude of conditions that may be encountered. Blahm (1978) demonstrated that commercial fire retardants were toxic to juvenile coho salmon and rainbow trout and attributed the mortality to ammonia in the retardants; increasing the pH of diluent water from 7 to 8 increased the toxicity. McKee and Wolf (1971) noted that ammonia concentrations as low as 0.3 mg/L were lethal to trout fry and 75 mg/L 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 mixture. Blahm et al.[16] found that two species of juvenile salmonids exposed to four commercial fire retardants had 96-h TLms of 120–940 mg/L.

Johnson and Finley (1980) found that warmwater fish species were less sensitive than salmonids to two Phos-Chek fire-retardant formulations (Table 7.12). Yolk-sac fry of coho salmon and rainbow trout were more sensitive than fingerlings.

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) stressed that the suitability of cyanide-polluted waters for aquatic life has to be

[16]Unpublished report, "Effect of chemical fire retardants on the survival of juvenile salmonids," by T. H. Blahm, W. C. Marshall, and G. R. Snyder, Bureau of Land Management, Contract 53500-CT2-85(N), National Marine Fisheries Service, Environmental Field Station, Prescott, Oregon, 1972.

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expressed as a concentration of free cyanide or molecular HCN, not of total cyanide. Free cyanide concentrations from 0.05 to 0.01 mg/L as CN have proved fatal to many sensitive fishes (Jones 1964), and levels above 0.2 mg/L are rapidly fatal for most species of fish. A level as low as 0.01 mg/L is known to have a pronounced, rapid, and lasting effect on the swimming ability of salmonid fishes (U.S. Environmental Protection Agency 1973b). Blahm (1978) 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 are 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 might be more toxic to aquatic life than other approved retardants, especially on sunny days.

Risk Assessment

Risk Assessment at the Organism Level

The toxicological risk of forest chemicals to anadromous fish may be manifested through direct action on the fish themselves or indirect action on fish food organisms. One means of expressing toxic risks is the margin of safety, i.e., the ratio of the "no-effect" level (concentration) to the actual exposure concentration. The no-effect level is the highest concentration that causes no mortality of test animals in acute toxicity tests. When the exposure level is equal to the no-effect level, the margin of safety is 1.0. Margins of safety less than 1.0 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 judgement. For many pharmaceuticals, caffeine, alcohol, and other materials many humans encounter daily, the margins of safety are as low as 1.5–15, 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. When the species likely to be exposed are extremely valuable or rare, a much larger margin of safety may be appropriate. These margins of safety usually reflect an assumption that long-term chronic exposure will occur. Some margin of safety is necessary because (1) the toxicity testing done thus far may not have identified the "lowest" no-effect level, (2) toxicity-testing conditions usually differ from field conditions, and (3) individuals in the population differ in susceptibility.

Forest chemicals have been investigated mostly for their acute 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 can reduce fish growth in the field and laboratory (Warner and Fenderson 1962; Cope et al. 1970; Woodward 1976). Several types of behavior (e.g., learning, swimming, temperature preference, predator avoidance) may be altered by exposure to pesticides (Ogilvie and Anderson 1965; Warner et al. 1966; J. M. Anderson 1968, 1971; Anderson and Peterson 1969; Hatfield and Anderson 1972; Hatfield and Johansen 1972; Symons 1973, 1977). Both DDT and 2,4-D can lower the reproductive success of fish (Macek 1968; Wilbur and Whitney 1973). Lorz et al. (1979) showed that diquat and picloram inhibited migration by coho salmon smolts in coastal Oregon streams. Many studies have demonstrated biochemical or physiological changes in fish exposed to pesticides (Weiss and Gakstatter 1964; Grant and Mehrle 1970; Wildish et al. 1971; Hiltibran 1972a, 1972b). One of the best-documented biochemical effects of a forest chemical is the inhibition of acetylcholinesterase activity by organophosphate pesticides (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 likely to be much lower than for acute or chronic toxicities. Our lack of knowledge prevents risk 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 salmonids.

Organisms can exhibit numerous kinds of responses when exposed to toxic chemicals. Changes in survival, growth, reproductive success, and behavior are probably the most important of these, but the bulk of the aquatic toxicology literature reports only survival during short-term acute exposures to toxicants. Although this deficiency in the data base is obvious, short-term acute exposures predominate in forest aquatic systems, 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 selected the concentration of 0.1(96-h LC50), or 10% of the 96-h LC50, as the no-effect level for survival after brief acute exposures to peak concentrations of a forest chemical. This value is a little more conservative than the 0.1(48-h LC50) 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 10% of the toxic units, or 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 several application factors had been proposed but "generally speaking, recommendations for maximum levels are 0.1 or 0.05 toxic units for non-persistent pollutants, and 0.1 or 0.01 toxic units for persistent chemicals and pesticides, mostly the lower figure." The U.S. Environmental Protection Agency (1973b) also recommended the use of application factors not exceeding 10% of the 96-h LC50, when materials are nonpersistent or have noncumulative effects, to estimate "safe" concentrations of toxic wastes discharged 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 chapter. The U.S. Environmental Protection Agency (1973b) recommended that no toxicant concentration should exceed 5% of the 96-h LC50 at any time or place, and that the 24-h average concentration of persistent or cumulative-action toxicants should not exceed 1% of the 96-h LC50.

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

Risk assessment for acute toxicity.—Numerous acute exposure values can be used to calculate margins of safety. We used both the single highest instantaneous field concentration we found in surveying the literature and a peak concentration of 0.02 mg/L (Table 7.16), which we believe is the maximum likely to occur if minimum buffer strips are used along streams and lakes and some direct application to surface water occurs. We used these values with 10% of the 96-h LC50 to calculate the margin of safety for acute exposures (Table 7.17). These calculations yield conservative estimates of the margin of safety because the instantaneous peak concentration in the field does not persist for the 96-h period used in toxicity tests and current "best management practices" in the use of forest chemicals will not produce exposure levels that approach the peak concentrations listed in Tables 7.16 and 7.17.

Risk assessment for chronic toxicity.—We calculated the margin of safety for chronic exposures using (1) integrals of concentration–time curves for chemicals in forest streams as estimates of exposure in the field and (2) integrals of concentration–time curves for exposures equal to 1% of the 96-h LC50 as estimates of no-effect exposure levels in toxicity tests. This concept is based on

TABLE 7.16.—Integral of concentration–time curves for 48 h for several pesticides and for 192 h for urea in forest streams after aerial application.[a]

Chemical	Actual peak concentration (mg/L)	Integral for actual peak concentration ([mg/L]h)	Integral for assumed peak concentration of 0.02 mg/L for pesticides and 7.02 mg/L[b] for urea ([mg/L]h)
2,4-D	0.014	0.116	0.167
Amitrole	0.110	0.498	0.091
Dicamba	0.037	0.310	0.167
Malathion	0.040	0.074	0.037
Carbaryl	0.121	0.343	0.057
Acephate	0.471	1.708	0.072
Urea[c]	1.389	38.2	193
Urea[d]	0.700	19.4	195

[a]Based on Figure 7.4.
[b]Mean peak concentration of 28 fertilizer-monitoring projects summarized by Moore (1975b).
[c]Based on Figure 7.4G.
[d]Based on Figure 7.4H.

the use of exposure units, i.e., the integral of duration and level of exposure, as developed by Allison (1977) and Larson et al. (1978).

The use of 0.01(96-h LC50) as the no-observed-effect concentration for chronic exposure (NOEC) is based on (a) the findings of Kenaga (1982), who calculated the acute:chronic no-effect levels for 135 compounds and found 93% of these values expressed as their log was 1.4 or less, and (b) the recent analysis by Slooff et al. (1986) who regressed 164 data pairs of $\log_{10}$(NOEC) versus $\log_{10}$(50%-effect concentration, L[E]C50) in standard acute toxicity tests. Slooff et al. (1986) found $\log_{10}(\text{NOEC}) = -1.28 + 0.95\log_{10}(\text{L[E]C50})$; $r = 0.89$. This equation yields L[E]C50:NOEC ratios with logs of about 1.3, suggesting the actual NOEC value is closer to 0.05(96-h LC50) than to the 0.01(96-h LC50) value we used in our calculations. Thus, our estimates of margins of safety for no-chronic-effect levels are conservative, erring on the side of safety.

Numerous data exist on 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%) at 3 h after application (Figure 7.4A–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 first day after application). The areas under the curves were measured to give a time–concentration expression ([mg/L]h) of contamination for the first 48 h after application; we used both the actual peak concentration observed and the assumed instantaneous peak concentration of 0.02 mg/L (Table 7.16). 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 the related discussion in the section

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TABLE 7.17.—Estimated margins of safety for survival of salmon, trout, and other sensitive aquatic species to "short-term" exposure to selected forest chemicals. LC50 is median lethal concentration in laboratory studies; NOEC is no-observed-effect concentration (10% of the LC50)[a]; NOEE is no-observed-effect exposure (integration of time and concentration)[b]; HOC is highest observed concentration (field applications)[c]; STE is short-term exposure integrated over time and concentration (field applications; peak concentration assumed to be 0.02 mg/L).[d]

Formulation and test species	96-h or 48-h* LC50 (mg/L)	NOEC (mg/L)[a]	Margin of safety $\left(\frac{NOEC}{HOC}\right)$	Margin of safety $\left(\frac{NOEC}{0.02}\right)^{e}$	48-h field exposures: NOEE ([mg/L]h)[e]	48-h field exposures: Margin of safety $\left(\frac{NOEE}{STE}\right)$
Herbicides						
2,4-D: HOC = 0.84 mg/L; STE = 0.334 (mg/L)h[f]						
Dimethylamine						
Rainbow trout	100	10	11.9	500	48	144
Daphnia sp.	4	0.4	0.5	20	1.92	5.7
Glass shrimp	0.15	0.015	<0.1	0.7	0.072	0.2
Butyl ester						
Cutthroat trout	0.9	0.09	0.1	4.5	0.432	1.3
Pteronarcella sp.	1.5	0.15	0.2	7.5	0.72	2.2
PGBE ester						
Cutthroat trout	1.0	0.1	0.1	5	0.480	1.4
Daphnia sp.	1.2*	0.12	0.1	6	0.576	1.7
Glass shrimp	0.4	0.04	<0.1	2	0.192	0.6
Picloram: HOC = 2.0 mg/L; STE = 0.083 (mg/L)h[g]						
Technical						
Cutthroat trout	3.5	0.35	0.2	17	1.68	20
Pteronarcys sp.	0.048	0.0048	<0.1	0.2	0.023	0.3
Potassium salt						
Cutthroat trout	1.5	0.15	<0.1	7.5	0.72	8.7
Tordon 101[h]						
Rainbow trout	8.6	0.86	0.4	43	4.13	50
Hexazinone: HOC = 0.044 mg/L; STE = 0.5 (mg/L)h[i]						
Rainbow trout	322	32.2	727	1,600	153	306
Bluegill	952	95.2	2,114	4,600	442	884
Daphnia sp.	20	2	45	100	9.6	19
Fiddler crab	>1,000	>100	>2,272	>5,000	>480	>960
Atrazine: HOC = 0.42 mg/L; STE = 0.668 (mg/L)h[j]						
Chironomous tenans	0.72*	0.072	0.2	3.6	3.46	5.2
Daphnia sp.	6.9*	0.69	1.6	34	3.31	5.0
Bluegill	6.7*	0.67	1.6	34	3.22	4.8
Brook trout	4.9*	0.49	1.2	24	2.35	3.5
Triclopyr: HOC = 0.095 mg/L; STE = 0.5 (mg/L)h[i]						
Butoxyethyl ester (Garlon 4)						
Rainbow trout	0.74	0.074	0.8	3.7	0.35	0.7
Bluegill	0.87	0.087	0.9	4.3	0.42	0.8
Triethylamine salt (Garlon 3A)						
Rainbow trout	552	55.2	579	2,750	264	528
Bluegill	891	89.1	937	4,450	427	854
Triethylamine salt						
Fathead minnow	120	12	126	600	57.6	115
Formulation unknown						
Rainbow trout	117	11.7	125	600	57.6	115
Bluegill	148	14.8	158	750	72	144

TABLE 7.17.—Continued.

Formulation and test species	96-h or 48-h* LC50 (mg/L)	NOEC (mg/L)[a]	Margin of safety (NOEC/HOC)	Margin of safety (NOEC/0.02)[c]	48-h field exposures: NOEE ([mg/L]h)[b]	48-h field exposures: Margin of safety (NOEE/STE)
MSMA: HOC = 0.01 mg/L; STE = ?[k]						
Liquid formulation						
Cutthroat trout	100	10	1,000	500	48	[1]
Gammarus fasciatus	100	10	1,000	500	48	[1]
Bluegill	12	1.2	120	60	5.76	[1]
Plus surfactant						
Bluegill	49	4.9	490	245	23	[1]
Fosamine ammonium: HOC = ?[m]; STE = 0.668 (mg/L)h[j]						
Coho salmon, fingerling	5,361	536	[1]	>10,000	2,573	3,852
Rainbow trout, yolk-sac fry	528	52.8	[1]	2,640	253	379
Glyphosate: HOC = 2.6 mg/L; STE = 0.668 (mg/L)h[j]						
Technical						
Rainbow trout	130	13.0	5	650	62.4	93
Liquid formulation						
Rainbow trout, fingerling	8.3	0.83	0.3	41	3.98	6.0
Rainbow trout, swim-up fry	2.4	0.24	<.1	12	1.15	1.7
Daphnia sp.	3	0.3	0.1	15	1.44	2.2
Dalapon: HOC = 3.65 mg/L; STE = 1.4 (mg/L)h[n]						
Rainbow trout	>100	>10	3	500	48	34
Bluegill	115	11.5	3	600	57.6	41
Dinoseb: HOC = ?[m]; STE = 0.334 (mg/L)h[g]						
Cutthroat trout	0.041	0.004	[1]	0.2	0.019	<0.1
Insecticides						
Malathion: HOC = 0.042 mg/L; STE = 0.037 (mg/L)h[o]						
Daphnia sp.	0.001*	0.0001	<0.1	<0.1	0.0005	<0.1
Pteronarcys sp.	0.01	0.001	<0.1	<0.1	0.0048	0.1
Coho salmon	0.17	0.017	0.4	0.8	0.082	2.2
Carbaryl: HOC = 0.121 mg/L; STE = 0.057 (mg/L)h[o]						
Daphnia sp.	0.006*	0.0006	<0.1	<0.1	0.0029	<0.1
Pteronarcys sp.	0.0017	0.00017	<0.1	<0.1	0.0008	<0.1
Coho salmon	4.34	0.434	3.6	22	2.08	36
Azinphos-methyl: HOC = ?[m]; STE = ?[k]						
Gammarus fasciatus	0.15	0.015	[1]	0.7	0.072	[1]
Pteronarcys sp.	0.0019	0.00019	[1]	<0.1	0.0009	[1]
Coho salmon	0.006	0.0006	[1]	<0.1	0.003	[1]
Carbofuran: HOC = ?[m]; STE = ?[k]						
Coho salmon	0.530	0.053	[1]	2.6	0.25	[1]
Rainbow trout	0.380	0.038	[1]	1.9	0.18	[1]
Acephate: HOC = 0.961 mg/L; STE = 0.072 (mg/L)h[o]						
Pteronarcella sp.	9.5	0.95	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

TABLE 7.17.—Continued.

Formulation and test species	96-h or 48-h* LC50 (mg/L)	NOEC (mg/L)[a]	Margin of safety $\left(\frac{NOEC}{HOC}\right)$	Margin of safety $\left(\frac{NOEC}{0.02}\right)^{e}$	48-h field exposures: NOEE ([mg/L]h)[b]	48-h field exposures: Margin of safety $\left(\frac{NOEE}{STE}\right)$
Fertilizer						
Urea: HOC = 44.4 mg/L; STE = 193.0 (mg/L)h[o]						
Ammonia: HOC = 0.014 mg/L[p]; STE = 0.83 (mg/L)h[q]						
Coho salmon	0.2	0.02	1.4	1	0.096	0.1
Fire retardant						
Ammonia: HOC = 1.30 mg/L[r]; STE = ?[s]						
Coho salmon	0.2	0.02	<0.1	1	0.096	[l]

[a]0.1(LC50), or 10% of the 96-h or 48-h LC50, is assumed to be the no-effects concentration for survival during short-term acute exposures.

[b]The no-effects exposure is the integral of the 0.01(LC50) curve over 48 h.

[c]The highest observed concentration is the single highest instantaneous concentration reported in the literature for field applications. Some values have been adjusted to reflect registered rates of application in forestry.

[d]Short-term exposures are 48-h integrals of the time–concentration curves in Figure 7.4 for assumed peak concentrations of 0.02 mg/L (see also Table 7.16). Based on operational monitoring, 0.02 mg/L is the maximum instantaneous concentration likely to result during field applications. It is assumed that streams in treated areas have minimum buffer strips and that there is some direct application of chemical to stream surfaces.

[e]Margin of safety for assumed peak concentrations of 0.02 mg/L for pesticides, 7.02 mg/L for urea fertilizer (0.02 mg/L for the ammonia component), and 130 mg/L for fire retardant (0.02 mg/L for the ammonia component).

[f]From Table 7.16, adjusted to a 2.24-kg/hectare application rate (based on data for 2,4-D, Figure 7.4A) and a peak concentration of 0.02 mg/L.

[g]From Table 7.16, adjusted to a 0.56-kg/hectare application rate (based on data for 2,4-D, Figure 7.4A) and a peak concentration of 0.02 mg/L.

[h]Tordon 101 is a 4:1 mixture of 2,4-D and picloram. The risk-assessment calculations were made with exposure data for picloram only, See 2,4-D for relevant data on 2,4-D.

[i]From Table 7.16, adjusted to a 3.36-kg/hectare application rate (based on data for 2,4-D, Figure 7.4A) and a peak concentration of 0.02 mg/L.

[j]From Table 7.16, adjusted to a 4.48-kg/hectare application rate (based on data for 2,4-D, Figure 7.4A) and a peak concentration of 0.02 mg/L.

[k]Data not available. Normal use is not expected to result in stream contamination.

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

[m]Data not available.

[n]From Table 7.16, adjusted to a 9.6-kg/hectare application rate (based on data for 2,4-D, Figure 7.4A) and a peak concentration of 0.02 mg/L.

[o]From Table 7.16, for an assumed peak concentration of 0.02 mg/L except for urea, which is based on an assumed concentration of 7.02 mg/L.

[p]From Table 7.13; assumes 1% un-ionized ammonia (25°C, pH 7.5).

[q]Based on the proportion of ($NH_3 + NH_4^+$) to urea, Table 7.13, and 1% un-ionized ammonia (25°C, pH 7.5).

[r]Assumes 1% ammonia in 130-mg/L retardant in stream water, from Norris et al. (1978).

[s]No estimate because of high variability in patterns of retardant use. Applications directly into streams will produce levels of ammonia >1 mg/L.

"Chemicals in water," and U.S. Animal and Plant Health Inspection Service 1980).

The results showed reasonably good agreement among the herbicides: 0.167 (mg/L)h for 2,4-D, 0.091 (mg/L)h for amitrole, and 0.167 (mg/L)h for dicamba (Table 7.16). Based on this analysis, we decided to use the 48-h time–concentra-

tion expression of exposure of 0.167 (mg/L)h derived from the 2,4-D data from Preacher Creek (Figure 7.4A), adjusted for rate of application for all the aerially applied herbicides in Table 7.17. MSMA was excluded because it usually is not 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 chapter.

Data for the concentrations of malathion, carbaryl, acephate, and urea (from fertilizer) in streams at various times after application were plotted, and the areas under the curves were integrated in the same way as for the herbicides (Figures 7.4D–H; Table 7.16). The normal uses of azinphos-methyl and carbofuran—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 h) of the time–concentration curve equivalent to 0.01(96-h LC50) for that chemical. These values are expressed as (mg/L)h for 48 h, just as the exposure data from field studies are expressed. For example, the 96-h LC50 of carbaryl for coho salmon is 4.34 mg/L and 0.01(96-h LC50) is 0.0434 mg/L. Because the exposure level is constant over the 48-h period we are interested in, the integral of the time–concentration curve is 0.0434 mg/L $\times$ 48 h = 2.08 (mg/L)h. The ratio of the no-effect exposure integral to the field exposure integral is the margin of safety (Table 7.17).

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 peak exposures are quite transitory. For instance, if we were to extend the period of evaluation from 48 h to 30 d, 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.

Risk Assessment at the Ecosystem Level

Assessments of risk to individual organisms rest on a reasonably adequate data base, but they focus on individual organisms and do not take into account time, 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 by greater individual tolerance for the chemical) and that repopulation of the affected portion of the stream will occur (by migration from unaffected areas or by 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

A
B
C
D
E
F
G
H
Percent of peak concentration
Hours after application
Actual
Estimated

assessments in Table 7.17 are conservative; the true margins of safety for salmonids from exposure to forest chemicals on the large watershed or ecosystem scales are greater than we have calculated.

Indirect Effects of Forest Chemicals

Toxic effects of forest chemicals on aquatic organisms have been investigated for several decades and are an integral component of environmental risk assessment. The intended uses of insecticides, herbicides, fertilizers, and fire retardants alter the structure and biological processes of both terrestrial and aquatic ecosystems, and these indirect effects of forest chemicals may have more profound influences on communities of fish and other aquatic organisms than direct lethal or sublethal toxic effects. Ecological effects of forest chemicals must be assessed from an ecosystem perspective rather than from the more simple perspective of direct toxicity, either lethal or sublethal, to an organism (Barnthouse et al. 1986).

Alteration of terrestrial vegetation and invertebrate communities may change both allochthonous inputs into streams and 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 Bjornn and Reiser (1991, this volume) and Murphy and Meehan (1991, this volume). Land

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FIGURE 7.4.—Concentrations of chemicals in forest streams at different times after aerial application. Concentration is expressed as a percentage of the peak concentration. Time intervals are 5 h in panels A–F and 10 h in panels G and H.

(A) 2,4-D in Preacher Creek, Oregon, with a partial buffer strip of streamside vegetation. Actual peak concentration was 0.0139 mg/L after an aerial application of 2,4-D at 1.12 kg/hectare. (From Table 10 of Fredriksen et al. 1975.)

(B) Amitrole in Wildcat Creek, Oregon, with no stream buffer. Actual peak concentration was 0.110 mg/L after an aerial application of amitrole at 2.24 kg/hectare. (From Table 10 of Fredriksen et al. 1975.)

(C) Dicamba in Farmer Creek, Oregon, with no stream buffer. Actual peak concentration was 0.037 mg/L after an aerial application of dicamba at 1.12 kg/hectare. (From Figure 2 of Norris and Montgomery 1975.)

(D) Malathion in Hansel Creek, Washington, with no stream buffer. Actual peak concentration was 0.040 mg/L after an aerial application. (From Figure 2 of Tracy et al. 1977.)

(E) Carbaryl in Squilchuck Creek, Washington, with no stream buffer. Actual peak concentration was 0.121 mg/L after aerial an application at 1.12 kg/hectare. (From Figure 7 of Bernhardt et al. 1978.) Note projection of the estimated concentration curve beyond 11 h.

(F) Acephate in Cabin Creek, Montana, with no stream buffer. Actual peak concentration was 0.471 mg/L after an aerial application at 1.12 kg/hectare. (From Table 12 of Flavell et al. 1977.) Note projection of the estimated concentration curve beyond 9.25 h.

(G) Urea in Coyote Creek, Oregon, with no stream buffer. Actual peak concentration was 1.39 mg/L after an aerial application of 224 kg N/hectare (as urea). (From Table 1 of Moore 1975b; and personal communication, D. G. Moore, U.S. Forest Service.) Note the time scale is not the same as in panels A–F.

(H) Urea in Trapper Creek, Washington, with 60-m stream buffer. Actual peak concentration was 0.7 mg/L after aerial application of 224 kg N/hectare (as urea). (From Figure 2 of Moore 1975a.) Note the time scale is not the same as in panels A–F.

managers must be aware of potential indirect effects of forest chemicals on patterns and processes of stream ecosystems.

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 have been investigated extensively (Swanson et al. 1982a; Triska et al. 1982); documented studies of indirect effects of forest chemicals on aquatic systems are rare. Indirect effects of herbicides on aquatic communities have been observed when, for example, aquatic plants were killed and subsequent shifts occurred in other components of stream ecosystems (Haven 1963; Smith and Isom 1967). Most such observations have followed direct applications of herbicide for aquatic weed control. In reviews of secondary effects of pesticides in aquatic systems, Hurlbert (1975) and Newbold (1975) considered mortality to aquatic plants to be the only indirect effect of herbicides. Concentrations of herbicides in surface waters after forest applications are much lower (<0.1 mg/L) than those needed to control aquatic weeds (>2 mg/L) (Norris and Moore 1971, 1976; National Research Council of Canada 1978; Norris 1978), so forest herbicides are unlikely to cause indirect effects due to the death of aquatic vegetation in streams, 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. Plant succession after a disturbance generally goes through three stages: an herbaceous stage, generally lasting less than 5 years; a shrub stage, roughly lasting from the 5th year through the 15th year; and a tree-dominated stage, which begins after about 10–15 years (Dyrness 1973; Franklin and Dyrness 1973; Swanson et al. 1982a). In western North America, tree communities in the early stages of succession are often dominated by deciduous trees such as alder, bigleaf maple, or vine maple. Large shrubs such as rhododendron, ceanothus, and salmonberry 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 habitats, timing of litterfall, and quality of organic matter produced. Herbicide applications in the northwestern USA may have other long-term ecological implications because several pioneer species such as red alder and ceanothus are nitrogen fixers, and terrestrial plant production in this region is generally nitrogen limited. Therefore, reduction of pioneer communities may alter the nitrogen dynamics in watersheds, but few relevant data from herbicide-treated areas are available (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 is known 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 after watersheds have been logged, so they are not the primary cause of increased streamflow; rather, they may extend the 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 the amount of solar radiation that reaches the stream channel, which can raise summer water temperatures under many conditions of flow, gradient, and geomorphology (Brown 1969).

When the vegetative structure of watersheds is altered, streambanks may lose stability and hillslopes may erode more. The degree to which the vegetation and the rooting systems are altered by logging determines the extent of sedimentation and channel modification, but the sedimentation rates in deforested watersheds are frequently more than double those in forested watersheds (Swanston and Swanson 1976). To the extent that herbicide applications retard vegetative recovery in a watershed, they may extend the period of increased sedimentation, but there are no published studies on this matter. The detrimental effects of sedimentation and channel degradation on the structure and function of stream ecosystems are documented elsewhere in this volume.

In sufficient concentrations, herbicides may directly affect aquatic primary producers (plants); indirect effects of herbicide applications may also alter primary producers in streams. If increases in solar radiation result from alteration of streamside vegetation, aquatic primary production may be stimulated (McIntire and Phinney 1965; Hansmann 1969; Gregory 1980). Such responses have been observed after control of riparian vegetation by herbicides in rangelands of the southwestern USA (Smith et al. 1975). Temperature increases may also elevate rates of gross primary production in streams (McIntire 1966). Conversely, sedimentation from terrestrial systems influenced by herbicides may cover benthic algal communities, scour algal cells from substrate surfaces, or otherwise reduce standing crops of primary producers (Cordone and Pennoyer 1960; Chapman 1963; Nuttall 1972).

Primary production in streams may be stimulated by increased nutrient concentrations. Herbicide application has been followed by increased nitrogen inputs to streams (Sollins et al. 1981). Nitrogen concentrations in stream water increased after herbicide treatment of a watershed in New Hampshire (Likens et al. 1970); however, the herbicide application rate there was much greater than commonly used in forestry (Likens et al. 1970). Primary production in most northwestern U.S. streams is nitrogen limited (Thut and Haydu 1971; Gregory 1980), and increases in dissolved nitrogen released by herbicide treatments may stimulate stream productivity.

Aquatic invertebrates may be affected by physical changes that result from herbicide application. Any increase in sedimentation could scour or otherwise

degrade their habitats (Cummins and Lauff 1969; Burns 1972; Brusven and Prather 1974; Cederholm and Lestelle 1974). Temperature increases may stimulate the growth and production of aquatic insects if the increases are slight; if stream temperatures exceed a species' optimum, however, the effect will be 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 (Jensen 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 deciduous riparian vegetation to conifers reduces the quality of food for detritus-feeding invertebrates.

The production of grazing insects could increase if aquatic primary production is stimulated after herbicide treatment. Grazers in streams are often food limited (McIntire and Colby 1978); therefore, increases in their food supply enhances their production. This enhancement of grazing invertebrates 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 sometimes is greater than it is in forested sections (Aho 1976; Erman et al. 1977; Hall et al. 1978; Murphy 1979; Murphy et al. 1981; Hawkins et al. 1983). 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 the 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 them. Sedimentation may reduce egg and fry survival (Neave 1947; Phillips 1964; Koski 1966; Bjornn[17]) and the quality of rearing habitat (Everest and Chapman 1972; Bjornn et al. 1974). Salmonids also require cover; streamside vegetation provides a major portion of this feature (Lewis 1969; Hunt 1978). Reduction of streamside vegetation by forest herbicides would, therefore, adversely affect salmonid populations.

Thus, herbicides may indirectly affect stream ecosystems either positively or negatively. The degree of effect is a function of the extent, level, patterns, and timing of applications. Evaluations of potential effects of herbicides on stream ecosystems must take all these factors into account.

[17]Unpublished annual completion report, "Embryo survival and emergence studies," by T. C. Bjornn, Project F-49-R-6, Job 6, Salmon-Steelhead Investigations, Embryo Survival and Emergence Studies, Idaho Fish and Game Department, Boise, 1969.

Insecticides

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

Insecticides may directly kill stream invertebrates or induce catastrophic drift of invertebrates out of treatment areas. Early studies of the effects of DDT on aquatic organisms noted that invertebrate drift increased immediately after spraying, invertebrate densities were reduced, and the composition of invertebrate communities in streams was altered for up to 4 years (Filteau 1959; Ide 1967). Experimental applications of permethrin, a synthetic pyrethroid, along streams in Canadian forests resulted in decreased abundances of aquatic invertebrates for 3–16 months (Kreutzweiser 1982; Kingsbury 1983). The reductions were attributed to both catastrophic drift that lasted for 3–12 h and invertebrate mortality (piles of dead invertebrates were observed on the stream bottoms). Invertebrate abundances in the stream were depressed for up to 2 km downstream from the application areas. Similar responses were observed when the carbamate insecticide aminocarb was applied near an Ontario trout stream (Holmes and Kingsbury 1982). Such alterations of abundance and community structure of aquatic insects can, in turn, change the abundances and community dynamics of the predators that feed on them.

Benthic algal communities 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 increased 2- to 20-fold (Barnley and Prentice 1958; Hynes 1961; Binns 1967; Chutter 1970). Similar responses have been observed in streams when watersheds were treated with DDT to control forest insects (Adams et al. 1949; Morgan and Kremer 1952; Webb and MacDonald 1958; Filteau 1959; Ide 1967). 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 population levels (Ide 1967),

and their full recovery may be further delayed by competition with established short-lived species. Predators tend to have longer life cycles than other types of invertebrates, so full recovery of invertebrate communities may require 5–10 years. Nevertheless, invertebrate predators sometimes increase after application of forest insecticides. For example, populations of dobsonfly larvae (*Nigronia* sp.) increased in streams flowing through Connecticut watersheds that were treated for spruce budworm (Hitchcock 1965). Other populations of predacious insects, such as plecopterans, decreased during this period.

Insecticide use can not only kill aquatic insects and increase the rate of insect drift (Crouter and Vernon 1959; Ide 1967; Kreutzweiser 1982), it is likely to greatly increase the number of terrestrial insects that fall on stream surfaces (Warner and Fenderson 1962; Kreutzweiser 1982). These insects are ingested by drift-feeding fish such as trout and salmon and may induce a secondary toxic effect on the fish. If the toxic effect is slight (or nonexistent), the sudden increase in food may cause a brief acceleration of predator growth. Such an enhancement of food supply is brief at best, however; a more frequent response is an overall reduction in invertebrate prey and a decline in predator growth. For example, the diets of brook trout and slimy sculpins reflected changes in both abundance and community structure of aquatic insects after a synthetic pyrethroid was applied to a forest (Kruetzweiser and Kingsbury 1982). As insect communities recovered, food consumption by fish returned to previous quantities and composition; after 16 months, condition factors of fish in treated and untreated areas were similar. Growth rates of 1- and 2-year-old Atlantic salmon parr decreased immediately after deposition of the same synthetic pyrethroid in another stream, but increased in late summer to the extent that fish in treated and untreated areas achieved the same size by summer's end (Kingsbury 1983). Over the long term, decreased populations of aquatic insects will most likely result in decreased growth and production of fish populations. Recovery of fish populations is determined, therefore, by recovery of invertebrate communities.

Microbial pathogens are being considered increasingly for control of forest insect pests because of their specificity for target organisms and low toxicity to other organisms. Polyhedral viruses have been used in forests to control insect pests and appear to be safer than chemical insecticides (Pimentel 1980), but there have been few, if any, studies of indirect effects of viruses in aquatic ecosystems. Specific strains of the bacterium *Bacillus thuringiensis* (*B.t.*) have been used for the control of Lepidoptera and Diptera in forest environments. Aquatic Lepidoptera are relatively rare, but caddisflies, a major component of most stream ecosystems, are closely related and might be more susceptible than most other aquatic insects. Aquatic Diptera are exceedingly common; the most common dipteran pests for which *B.t.* is applied are mosquitoes and blackflies, both aquatic insects. Application of *B.t.* for control of aquatic insects alters the aquatic community structure and so influences other aquatic organisms, and assessment of the need for such control projects must consider these potential effects. Although the potential exists for effects on nontarget organisms, little evidence has been found for such responses (Buckner et al. 1974; Ali 1981; Burges 1982). The high degree of specificity of *B.t.* for target organisms makes it unlikely that indirect effects will be substantial.

Fertilizers

Some forests in the northwestern USA are fertilized for several decades after logging. Urea, the most common fertilizer, is quickly converted to ammonium or nitrate in the soil, so nitrogen can enter streams in all three forms. Most of the urea that enters streams does so within the first 48 h after application (Moore 1975a, 1975b). After that, nitrogen enters streams primarily as nitrate. Fertilization generally increases the nitrogen content of stream water by 50 mg/L or less, and these nitrogen pulses last for about 1 year (Fredriksen et al. 1975; Moore 1975a, 1975b).

As previously described, streams in the northwestern USA are commonly nitrogen limited, and primary production in such streams may be enhanced by fertilization (Thut and Haydu 1971; Stockner and Shortreed 1976; Gregory 1980). Nutrient stimulation of primary production occurs only with sufficient light intensity (Gregory 1980), but trees in most fertilized watersheds are less than 40 years old, so unless old-growth buffer strips had been left along streams, shading should not inhibit stream productivity.

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 Tesch 1961; Albrecht 1968; LeCren 1969; Mills 1969; Hall et al. 1978; Murphy et al. 1981; Hawkins et al. 1983) and attributed to greater primary production. Fertilization could, therefore, indirectly enhance 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 northwestern USA for the suppression and control of forest fires. Fires often start on ridgetops, away from streams. As fires develop, they may sweep across streams and rivers, so direct entry of fire retardants into streams is possible.

Application of fire retardants usually increases the concentrations of ammonia in stream waters (see footnote 2). These concentrations may range from 0.01 to 100 mg N/L. As already discussed, such nitrogen increases can stimulate primary and secondary production in streams. Increased production of aquatic biota could be precluded if toxic effects occurred. Potential indirect effects of retardants on the mortality of invertebrates or fish are the same as those previously described for insecticides—if concentrations in streams are sufficiently high. In an experimental release of a fire retardant containing diammonium phosphate, no significant positive or negative 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 and the release time of nitrogen to the stream is longer. Most fire-retardant drops occur in watersheds well drained by streams, but if retardants are used in or around basins with oligotrophic lakes, bogs, or swamps, their aquatic effects may be prolonged and exaggerated.

If fire retardants are not used and fires are allowed to burn, this too has implications for aquatic environments. Fire is a natural reset mechanism in

northwestern forests and a fundamental driver of terrestrial plant succession. Human logging practices have duplicated many of the results of fire by converting much forest land to a pioneer stage of succession. These effects were reviewed by Norris et al. (see footnote 2) and Swanston (1991, this volume). Briefly, potential effects of fire on salmonid habitat may include 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. If the hazards of fire retardants are to be assessed accurately, indirect effects of fire retardants must be weighed against those that would result if fire were not controlled.

General Perspectives on Indirect Effects of Forest Chemicals

Forest chemicals have great potential for indirectly altering aquatic communities and salmonid 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 on fish habitats must be considered in relation to previous or simultaneous effects of other forestry and land-use practices.

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 features of aquatic systems are influenced by the alteration of terrestrial succession: allochthonous organic inputs; tree and shrub canopy over streams; stream chemistry; and sedimentation rates. These factors are major fundamental determinants of the structure and function of stream ecosystems and are affected by logging with or without the use of herbicides. The basic effects of herbicides are to extend the early stages of watershed recovery, to minimize intermediate stages, and to accelerate development of coniferous stages. Potential indirect effects of herbicides on aquatic ecosystems must be viewed within this successional framework.

Fertilizers 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. In coniferous forests, fertilizers are usually applied after the conifer canopy has closed to avoid stimulating growth of competing species and to allow greater utilization by conifers. Fertilization at 5-year intervals could gradually increase nitrogen concentrations in forest streams at base flow.

Fire retardants, unlike other forest chemicals, are generally applied while watersheds are being acutely modified. Fire has many effects on salmonid habitats, as reviewed by Swanston (1991). The indirect effects of fire retardant are generally limited to stimulation of primary production, and even that effect is greatly influenced by the extent to which the fire itself reduces the vegetation canopy over streams.

Insecticides are applied more frequently than other forest chemicals to watersheds that are least influenced by human activities. Even so, the effects of insecticides on aquatic systems must be viewed in relation to the effects of not using them and of allowing insect damage to forests. Insect-related effects are much less severe than the effects associated with logging or fire, but still must be incorporated into decision-making processes.

In assessing potential indirect effects of forest chemicals on salmonid habitats, land managers must consider the influence of protective measures (particularly buffer strips) on aquatic systems. 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 must be transferred through such zones and become greatly diminished in the process. In clearcut watersheds where buffer strips of uncut vegetation are left, the additional use of spray buffer strips would be even more effective in reducing indirect effects of forest chemicals on aquatic communities. If buffer strips of uncut vegetation and no-spray zones are used in watershed management, many of the indirect effects of forest chemicals on stream ecosystems described in this chapter would not occur.

Indirect effects of forest chemicals on salmonids and aquatic ecosystems must be evaluated on appropriate temporal and spatial scales. Most biological processes in streams exhibit strong seasonal patterns, and the responses of aquatic organisms are closely related to the timing of application of a forest chemical. For example, summer is a period of low streamflow and winter is a period of high streamflow in many streams of the northwestern USA. Application of fertilizer to a watershed has a potentially greater effect on aquatic primary production in summer, when discharge is low and solar radiation is high, than in winter, when discharge is high and solar radiation is low. Location within a basin also influences the ecological responses to chemicals. The abundance and distribution of aquatic organisms change from headwaters downstream to large rivers (Vannote et al. 1980). Streams are connected within a drainage, and application of chemicals at one point may influence downstream communities. The terrestrial adults of many aquatic insects disperse upstream to lay their eggs, and effects of forest chemicals at one point in a drainage may influence insect recruitment to upstream reaches. Salmonids may spawn in one area of a basin, but the fry may rear in either upstream or downstream reaches and tributaries. The complex patterns of biological processes through time and the distribution of communities throughout a basin must be considered when the potential indirect effects of forest chemicals on salmonids and other aquatic organisms are evaluated.

Forest chemicals are major tools in forest management. Risks of chemical use must be evaluated, however. Direct toxic effects of chemicals on aquatic organisms are major concerns, and 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 directly 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.

Although few studies of indirect effects of forest chemicals on salmonid habitats are available to land managers, the perspectives presented in this chapter 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 decision maker can be of reaching correct conclusions about it. This truism prompts scientists to prepare lengthy lists of research needs, many items of which are repetitions of earlier lists. All research needs are not equally important. We have attempted to identify gaps in knowledge that cause the greatest uncertainty in the information presented earlier in this chapter. We believe that these specific gaps are discrete and small enough to be filled by a single scientist with supporting staff. No one area will require major long-term grants or funding programs, although in aggregate, the solutions of these problems will require substantial effort. We present the list of research needs in the order the subjects appeared in the chapter.

Behavior of Chemicals in the Environment

- *Quantify the influence of buffer strips on concentrations of forest chemicals in streams.* Research and practice have demonstrated that buffer strips reduce the entry of chemicals into forest streams, but the degrees of protection provided by strips of different widths have 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 patterns of entry of atrazine, fosamine ammonium, glyphosate, triclopyr, and hexazinone 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 applications, were done when phenoxy herbicides were the predominant forest chemicals. Consequently, few data are available on other forest chemicals. The lack of data is particularly acute for the chemicals listed above.

- *Determine more precisely the fates 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 this chapter are mostly from laboratory studies or from intentional applications of chemicals 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 the systems used by salmonids.

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 exposure duration for forest chemicals with respect to the more sensitive aquatic species.* Most toxicity tests hold the concentration of chemical constant for a specified period (such as 24, 48, or 96 h) and evaluate organism response soon afterward. In the field, aquatic organisms typically are exposed 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. Calibrations need to be established that will permit use of the extensive constant-exposure toxicity data base for evaluations of field exposures. In this chapter, we used an integral of the time–concentration curve, but this approach has not been fully validated.

• *Determine if the results of classical 96-h exposure tests with forest chemicals are adequate predictors of the long-term well-being of aquatic organisms.* Nearly all the toxicity testing on aquatic organisms has incorporated short-term exposures and only short-term observations of effects. Research is needed to determine if long-term latent effects result from short-term exposures. 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 exposures 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.* Indirect and sublethal effects may result from very low chemical concentrations; both laboratory and (especially) field research on these subjects are needed before safe use of forest chemicals can be assured. Indirect effects on aquatic ecosystems 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, appropriately located, and properly timed to permit the observation of changes in aquatic communities.

Conclusions

The use of forest chemicals can result in both direct and indirect effects on salmonids and their habitats. 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 of the probability that use of a specific forest chemical will directly affect salmonids. The larger the margin of safety, the less likely direct effects will occur. Margins of safety less than 1.0 indicate direct effects are likely to occur. We calculated margins of safety for fish, based on the maximum acute and short-term chronic exposures likely to occur in operational uses of these chemicals (Table 7.17). These margins of safety will be 5–10 times greater when streams do not occur in areas to be treated, when buffer strips are used along streams, and when 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 densities and community organization 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 important to the well-being of salmonid fishes. These changes have not been as thoroughly studied as the direct effects, but may be the most likely to occur.