Diminishing Returns:

Salmon Decline and Pesticides

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Diminishing Returns: Salmon Decline and Pesticides

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February 1999

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Executive Summary

Pacific salmon are in serious trouble. The enormous runs of migratory salmon of the past have slowly diminished to the trickle of adult spawning salmon that presently inhabit western rivers. Although salmon recovery efforts are underway, scientists, policy makers, and interest groups have thus far given insufficient attention to the role that pesticide contamination of our watersheds may play in salmon decline. Accordingly, the purpose of *Diminishing Returns: Salmon Decline and Pesticides* is to review scientific literature on the effects of sublethal concentrations of pesticides on salmonids (see full report for documentation and references). The report places special emphasis on how pesticides can alter the biology of fishes in subtle ways that decrease their chances for reproduction and survival.

Dynamics of Pesticides in the Environment

Pesticides include a broad class of chemical and biological agents that are purposefully introduced into the environment to kill or damage organisms, including insecticides, herbicides, and fungicides. Once applied, pesticides move into streams and rivers throughout watersheds and may pose problems far from the site of application. Movement often occurs through the medium of water, thereby exposing all aquatic organisms during this transport. Where water quality monitoring has been done, a great variety of pesticides are typically found in salmon habitat. Federal and state agencies have established few criteria or standards for the protection of aquatic life from short-term (acute) and long-term (chronic) exposure to pesticides.

Pesticides do not necessarily disappear with time. They transform into other compounds that may be less toxic, of equal toxicity, or of greater toxicity than the original compound. The toxicity of these breakdown products is not well understood, and in general how they affect aquatic life has not been studied. All the while, fish and other aquatic organisms must continue to cope daily with pesticides (and their breakdown products), some of which are no longer used but remain in watersheds.

Although pesticides are diluted by transport in rivers and streams, a number of mechanisms concentrate the chemicals, often to toxic levels. In a process known as bioaccumulation, pesticides absorbed into plant and animal tissues may become concentrated and reach levels many times higher than those in surrounding water.

Fish Kills and Acute Toxicity of Pesticides to Salmon

Pesticides are capable of killing salmon and other aquatic life directly and within a short period of time. For example, in 1996 the herbicide acrolein was responsible for the death of approximately 92,000 steelhead, 114 juvenile coho salmon, 19 resident rainbow trout, and thousands of nongame fish in Bear Creek, a tributary of the Rogue River. Deaths of threatened and endangered species from accidental contamination of waterways are of grave concern. The loss of each individual in a sensitive population makes recovery efforts that much more difficult. Fortunately, these deaths are relatively infrequent.

Behavioral Effects of Pesticides at Sublethal Concentrations

In contrast to dramatic fish kills, the effects of sublethal concentrations of pesticides are more subtle and go largely unseen and unregulated. Sublethal concentrations of pesticides do not cause immediate death, but can interfere with the biology of the organism in other ways and can ultimately impact the survival of the species. Laboratory studies show that sublethal concentrations of pesticides can affect many aspects of salmon biology, including a number of behavioral effects:

- Long-term exposure to certain pesticides can increase stress in juvenile salmonids and thereby render them more susceptible to predation.

- Certain pesticides can alter swimming ability, which in turn can reduce the ability to feed, to avoid predators, to defend territories, and to maintain position in the river system.

- Many pesticides interrupt schooling behavior, a critical tactic for avoiding predation during salmon migration. Disruption of schooling behavior is thought by some researchers to be a classic method for examining sublethal effects of pesticides because the effect is so common.

- Several pesticides (and other pollutants) have been shown to cause fish to seek suboptimal water temperatures, thus subjecting them to increased dangers of disease and predation.

- Some herbicides have been shown to inhibit normal migration to the sea, resulting in severe disruption of the life cycle. There is a dearth of research looking at this effect for common insecticides.

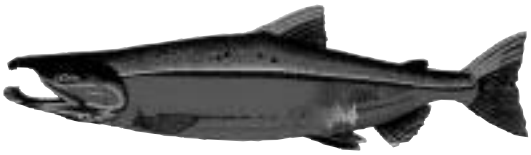
- Several studies suggest that certain pesticides can impair salmonid's ability to transition from freshwater to seawater. There is a need for further research in this area, placing particular emphasis on the critical period of transition that takes place in the estuary.

- Adult salmon adjust their migration patterns to avoid polluted areas, resulting in delayed spawning.

Compromised Immune Systems

In addition to changes in behavior, exposure to relatively low concentrations of

pesticides can disrupt the immune system of salmon. Evidence for these effects in salmonids is not as extensive as for disruption of behavior, but the data available suggest that pesticides can have serious negative impacts on the immune system. Such disruption results in the onset of disease and even death.



Endocrine Disruptors

Fish and other organisms are especially vulnerable to endocrine-disrupting effects during the early stages of development. Pesticides at low concentrations may act as mimics or blockers of sex hormones, causing abnormal sexual development, feminization of males, abnormal sex ratios, and unusual mating behavior. The unique plasticity of sex differentiation in fish suggests that these animals may be very susceptible to disruption of sexual characteristics by pollutants. Pesticides can also interfere with other hormonal processes, such as thyroid functioning and bone development.

Indirect Effects of Pesticides on Salmon

Pesticides can indirectly affect fish by interfering with their food supply or altering the aquatic habitat, even when the concentrations are too low to affect the fish directly. Such indirect effects greatly reduce the abundance of food organisms which in turn reduces the growth and probability of survival of the fish. In addition, removal of aquatic vegetation can decrease habitat suitability and increase the salmon's susceptibility to predation. These indirect effects are subtle, but evidence suggests that in complex ecosystems indirect effects can be even more important than direct effects.

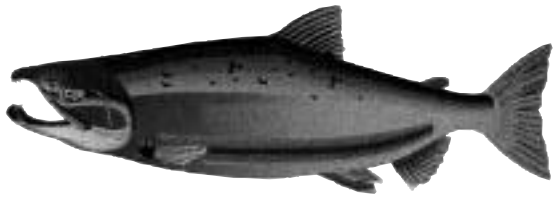
Recommendations

From the evidence available at present, there is a plausible basis for considering pesticides to be one of the causes of declining salmon populations in the Pacific Northwest. Based on this review, we offer several policy recommendations and identify areas for further research:

1. Address the impacts of pesticides on salmon when developing and implementing recovery plans for threatened and endangered species. To date, efforts to recover salmon have devoted insufficient attention to pesticides as a contributing factor in salmon decline. We must act now using available information to formulate management strategies that will minimize the potential danger from sublethal concentrations of pesticides.

2. Conduct ecoepidemiological studies in critical salmonid habitat. Most of the effects of pesticides referred to in this report have been determined in experimental laboratories. In the field, however, environmental conditions are not controlled, and many factors interact to confuse the determination of direct relationships. Ecoepidemiological investigations in the Great Lakes have established the relationship between chlorinated hydrocarbons and the decrease in lake trout populations. An ecoepidemiological approach for salmon in the Pacific Northwest would be particularly valuable because it is designed to attribute causality to events occurring in real-world situations.

3. Create comprehensive pesticide tracking systems in the Pacific Northwest. To better understand the relationship between pesticides and salmon decline, we must have accurate, site-specific data on the patterns of pesticide use in the watersheds of the Northwest. State and provincial governments need to collect data on which pesticides are used where, when, and in what amounts. Such data can then be combined with watershed-specific information on indicators of salmon health. (Currently, California is the only state with Pacific salmon habitat where such information is collected.) Pesticide use information will also enable efficient instream monitoring for pesticide contamination.



4. Establish instream monitoring programs in critical salmon habitats. A systematic monitoring program for pesticides and their breakdown products needs to be undertaken. Not all pesticides can be tested for in all locations, but current testing is woefully inadequate for understanding the role of pesticides in salmon decline. In conjunction with pesticide use data, these analyses can be targeted to the compounds of most concern. Such targeting can greatly improve the cost-effectiveness of monitoring.

5. Err on the side of caution when setting water quality standards for pesticides. There are few established criteria for the protection of aquatic life from pesticides. Moreover, evidence reviewed here shows that sublethal effects on salmonids have not been fully appreciated, that juvenile salmonids succumb more easily to toxins in the water, that laboratory studies do not reflect the natural life cycle of the fish, and that little is known about how pesticides affect aquatic ecosystems. These factors must be considered when setting standards, and a precautionary approach must be adopted.

6. Prevent pesticide contamination of salmonid habitat by reducing pesticide use. Once contaminated, water is difficult if not impossible to clean up. Therefore, pest management approaches that do not depend on pesticide use in agricultural and non-agricultural settings should be encouraged and further developed. There is ample evidence that ecologically sound and economically viable methods can be successfully implemented. The adoption of such alternatives can be encouraged through technical assistance, financial incentives and disincentives, demonstration programs, and information exchange opportunities.

7. Adopt state and provincial programs in the Pacific Northwest to phase out pesticides that persist and bioaccumulate in the environment. Numerous pesticides, including some that are no longer used and many that are currently used, are known to persist in the environment and to bioaccumulate in aquatic systems. Washington State's Department of Ecology is now considering a plan to end the release of such toxins, including certain pesticides, into the environment. To ensure salmon recovery, all state and provincial governments in the Pacific Northwest should adopt similar programs.

Introduction

Salmon are a keystone species for Northwest ecosystems. Entire foodchains base their existence on the proliferation of salmon and trout that surge from the sea into streams and rivers, work their way upstream to their natal spawning areas, lay their eggs, and die, providing a source of nutrients for the very organisms that later feed the salmon's progeny.

Salmon and trout make up the family Salmonidae, a group of fish characterized by an adipose fin located near the tail. These fish require clean, cold fresh water for much of their life history. Because such habitat is low in nutrients, salmon and many of the trout developed anadromy, a behavior pattern in which the juveniles migrate to the sea. In the sea, the juvenile fish use rich ocean productivity for feeding and growth, then return to the streams where they were born to lay their eggs.

Today, migratory salmon and trout are fighting for their survival. The enormous runs of migratory salmonids in the 1860s have slowly diminished to the trickle of adult spawning salmon that presently inhabit western rivers. The causes for the decline are many, some of which are well documented. Yet, in their recovery efforts for threatened populations, scientists, policy makers, and interest groups have tended to overlook the role of pesticides that flow daily through salmon habitat, potentially changing the biology of the fish in subtle ways that decrease their chances for survival and reproduction.

A great deal of work is being done to catalog and restore the physical aspects of Northwest streams (e.g., restoring riparian vegetation and gravel beds, planting buffer strips along streams). Insufficient attention, however, has been devoted to the use and presence of pesticides in the watersheds and the role this water quality degradation plays in salmon decline. Accordingly, the purpose of this report is to present information from scientific literature that points to the unseen danger posed by the existence of pesticides in salmonid habitats.

Rivers and streams have become great conduits through which pesticides, either intact or as breakdown products, flow to the sea. Salmon now live throughout their life cycle with these residues as part of their daily environment.

Pesticides include a broad class of chemical and biological agents that are purposefully applied to the environment to kill or damage organisms (National Research Council 1993). These agents are used in a wide range of occupations for a variety of purposes. Foresters use herbicides to keep broadleaf plants from competing with conifer seedlings, and insecticides to deal with numerous insect pests that damage forests. Farmers use insecticides to protect their crops and keep insects away from their livestock, and herbicides to remove unwanted weeds from fields and waterways. State and local agencies use herbicides to remove brush from roadsides. Fishermen use defouling compounds like tributyltin to keep organisms from settling on the hulls of their boats. The ordinary homeowner uses a wide variety of pesticides: herbicides on the lawn, insecticides on pets, and fungicides in house paint.

Oregon's ocean-going salmon

Chinook Salmon (ocean-rearing)

- Distribution includes coast and Columbia Basin mainstem rivers.
- Juveniles migrate to the ocean the first fall after they hatch, rearing briefly in estuaries.
- They rear over a broad ocean area, ranging from northern California to the Gulf of Alaska.
- Adults, typically 3 to 5 years old, return to fresh water in the spring, summer or fall.
- Spring and summer migrants prefer deep, cool pools where they hold several months before fall spawning.
- Adults spawn in large concentrations on mainstem gravel bars; may use both upper and lower mainstems.

Chinook (stream-rearing)

- In Oregon, they are only in upper Columbia Basin tributaries.
- Juveniles migrate to the ocean as 1-year-olds, in the spring.
- Little is known about the ocean distribution of Oregon's stream-rearing chinook.
- Adults return to fresh water in the spring, when 3 to 5 years old, and require deep, cool pools to hold for several months over the summer before fall spawning.
- They spawn in concentrations on gravel bars in upper tributaries.

Chum Salmon

- Shortest freshwater residence of all salmon. Adults stay only about a week prior to spawning; juveniles migrate to the ocean hours after hatching.
- Juveniles rear briefly in estuaries.
- Most Oregon chums migrate to the Gulf of Alaska for ocean rearing.
- Adults spawn at 3 to 5 years of age.
- Spawning occurs in lower mainstems, concentrated on large gravel bars.
- Adults are unable to pass even minor barriers.

Coastal Cutthroat

- Some coastal cutthroats migrate to the ocean. But others may migrate only to the estuary or river mainstems, or they may not migrate at all.
- Those that do go to the ocean migrate out in the spring, stay only a few months close to shore, then return in the fall.
- The ones that migrate may rear in fresh water for several years before going to the ocean.
- They spawn in the winter and early spring, using small pockets of gravel. They may spawn more than once. The spawning age of cutthroats seems to vary over their distribution area.
- Cutthroat prefer the smallest, highest tributaries in a basin.

Coho Salmon

- Juveniles rear throughout watersheds and tend to live in pools in the summer.
- Juveniles migrate to the ocean at 1 year, in the spring.
- Most Oregon coho rear just off our coast.
- Adults return to fresh water in the fall and spawn in late fall and winter.
- Adults tend to spawn in concentrations on gravel bars in upper watersheds.
- Most adults spawn when they are 3 years old.

Sockeye/Kokanee Salmon

- There is both an ocean-going form (called sockeye), and a resident form (called kokanee).
- Juveniles rear in a lake, spending 1 to 2 years in fresh water before migrating to the ocean in the spring.
- Columbia Basin sockeye migrate to the Gulf of Alaska for ocean rearing.
- Adults typically spend 2 years in the ocean.
- Loss of Oregon sockeye resulted from blocked access to lakes. Kokanee are thriving in some lakes.

Steelhead

- There are two subspecies of steelhead in Oregon. Each also has a resident form. Coastal steelhead are closely related to rainbow trout. Inland steelhead are closely related to redband trout.
- Most juveniles rear in fresh water for 1 or 2 years and migrate to the ocean in the spring.
- Most steelhead spend 2 years in the ocean. Their distribution is poorly known but appears to be further offshore than other salmon.
- Most inland steelhead return to fresh water in the summer while most (but not all) coastal steelhead return in the winter.
- Summer-run steelhead require cold, deep pools where they hold until spawning. All steelhead spawn in the winter and may spawn more than once.

ILLUSTRATION AND INFORMATION COURTESY: KATHRYN KOSTOW, OREGON DEPARTMENT OF FISH AND WILDLIFE

Once applied, pesticides can move away from the point where they were used. As a result, rivers and streams have become great conduits through which pesticides, either intact or as breakdown products, flow to the sea. Salmon now live throughout their life cycle with these residues as part of their daily environment.

This report reviews our existing scientific knowledge of the effects of pesticides on salmonids, placing a special emphasis on sublethal effects of pesticides. Sublethal effects are those that result from exposure to a pesticide in an amount that is not high enough to cause death, but can damage an organism in other ways, including physiological and behavioral changes that can ultimately impact the survival of the species. These effects can occur throughout the entire life history of salmonids, from hatching of eggs, entry of juveniles into the ocean, and return of adults for spawning. With the data currently available, it is possible to identify key areas that need immediate attention.

The present report emphasizes research performed on salmonids. Most of the studies included here, however, have been done with rainbow trout, which are found throughout the United States, are relatively easy to grow, and provide a reasonable standard for examining physiological and behavioral changes. Fewer studies were done with anadromous (seagoing) salmon. Where this research exists, the information is emphasized. Studies from other species of fish are introduced when certain points need to be made and information is not available from salmonid research.

The data suggest there is a plausible basis for considering pesticides as a causative factor in salmon population declines.

Dynamics of Pesticides in the Environment

Pesticides are Ubiquitous in Western Watersheds

In 1991, the U. S. Congress provided funds for the U. S. Geological Survey to conduct a National Water-Quality Assessment (NAWQA) on major river systems in the United States. The study units chosen encompass sources of drinking water for about 70 percent of the U. S. population. In the Northwest, study units included the Puget Sound and the central Columbia River plateau of Washington, the Willamette River Basin of Oregon, the Sacramento and San Joaquin River systems of California, and the Snake River Basin of Idaho. Table 1 presents the numbers of pesticides detected in streams and the numbers of pesticide detections that exceed criteria for aquatic life.

A major problem with the interpretation of the data presented in Table 1 is that concentrations of pesticides compatible with aquatic life are not well defined. The U.S. Environmental Protection Agency (EPA) has identified aquatic life criteria for the protection of aquatic organisms from short-term (acute) or long-term (chronic) exposure for very few pesticides. Of the 118 pesticides typically looked for in water quality studies, only 20 (17%) have been

Table 1. Pesticide detections in the western United States as measured by the U. S. Geological Survey NAWQA program.

State, region	# Pesticides Examined	# Pesticides Detected	# Exceeding Aquatic Life Criteria[1]	# For Which Aquatic Life Criteria Are Available
Oregon				
Willamette Basin[2]	86	36	4	22
Washington				
Puget Sound Basin[3]	NA	23	4	NA
Central Columbia River Plateau[4]	84	45	5	18
California				
San Joaquin Basin[5]	83	49	7	16
Idaho				
Snake River Basin[6]	80	36	2	17

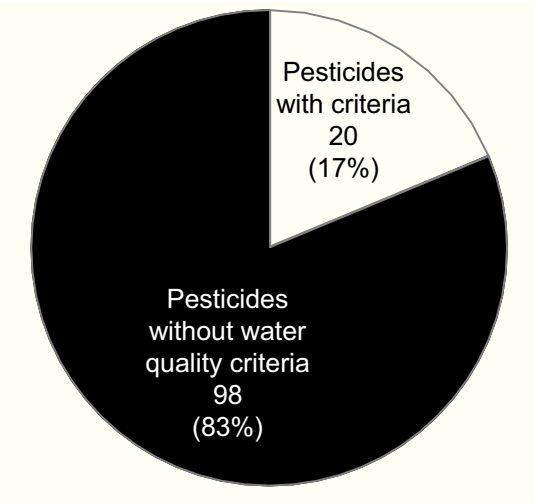
NA, not available.

[1] Aquatic life criteria set by the National Academy of Sciences and National Academy of Engineering or by the Canadian Council of Resources and Environment Ministers.
[2] Data from Anderson et al. (1997).
[3] Data from Bortleson and Davis (1997).
[4] Data from Wagner et al. (1996).
[5] Data from Dubrovsky et al. (1998).
[6] Data from Clark et al. (1998).

assigned aquatic life criteria (Larson et al. 1997). Of the 96 herbicides, 55 insecticides, and 30 fungicides that currently have the highest agricultural use in the United States, EPA has established aquatic life criteria for only 6 insecticides (Larson et al. 1997). EPA has not established any aquatic life criteria for the herbicides and fungicides most commonly used today.

The National Academy of Sciences (NAS) and National Academy of Engineers (NAE) set aquatic life criteria for a number of commonly used pesticides in 1973, but these are outdated. Their derivation was based on acute toxicity data but did not take into account bioaccumulation, sublethal effects, or synergistic effects.

Figure 1
Number of pesticides with aquatic life criteria.



(Based on 118 pesticides typically looked for in water quality studies)

Source: Larson et al. (1997)

The EPA and NAS/NAE criteria are commonly used as indicators of the degree of water pollution, but, as we shall see, may be far higher than the levels at which damage to fish can occur.

From the NAWQA studies, one can see that pesticide use is prevalent in our watersheds, and the water in our river basins contains dozens of pesticides. Exactly what happens to the hundreds of tons of pesticides put on the land each year?

Pesticides Persist in the Environment and Degrade into Toxic Products

Most pesticides undergo chemical transformations after application (Day 1991). The transformations may result from either physical processes, such as oxidation or photolysis, or biological metabolism by microbes. The breakdown products may be less toxic, of equal toxicity, or more toxic than the original compound. In many cases, the breakdown products of pesticides are not completely understood and their toxicities to aquatic life have not been studied. In surveys of pesticides in streams, the parent compound is typically looked for. When it is not found, it may be concluded that the pesticide has been cleared from the system. However, the breakdown products may still be present and constitute a danger to the organisms living there.

For example, Roundup, or glyphosate, has been publicized as an environmentally friendly herbicide that breaks down shortly after application. However, experiments have shown that glyphosate may persist in the environment for as long as 3 years (Torstensson et al. 1989). Its metabolite, AMPA, may persist even longer (World Health Organization 1994). Glyphosate is

typical of many pesticides in that its breakdown is dependent upon the environmental conditions in which it is used and that the toxicity of its breakdown products is equal to or greater than the toxicity of glyphosate itself.

The rate at which a pesticide breaks down varies widely, depending upon the conditions of application. Degradation depends largely upon temperature. Pesticides such as glyphosate may oxidize in as little as 3 days in Texas or as long as a year in Canada. Conditions that favor microorganisms also tend to promote pesticide degradation (Barbash and Resek 1996), although in some cases, the presence of humic material in the soil stabilizes the pesticide (Chapman et al. 1981; Barbash and Resek 1996). Light and water are also important in the degradation of pesticides (Barbash and Resek 1996). Pesticides break down more quickly under bright sunshine than under cloud cover. Breakdown occurs more quickly under moist conditions than under dry conditions. Oxygen concentration is also important for metabolism of pesticides. For example, pesticides are resistant to breakdown in the anoxic, highly reducing muds of estuaries where there is an absence of oxygen (Barbash and Resek 1996).

Pesticides may remain in the environment much longer than expected or claimed, and the breakdown products may also be toxic to organisms.

In short, the timing of degradation of a pesticide is highly variable and dependent upon environmental conditions. Pesticides may remain in the environment much longer than expected or claimed, and the breakdown products may also be toxic to organisms. Fish and aquatic organisms must cope daily with a variety of pesticides or metabolites that may have been used years before. Many of the pesticides which were banned long ago still appear in water quality surveys. One aspect of pesticide degradation is quite clear. Pesticides and their metabolites do not magically disappear from the environment.

Pesticides Move throughout Watersheds

Water promotes the transport of pesticides from their site of application (see Figure 2). Streams are often the recipients of pesticide residues following rainfall. But the rapid downstream transport of chemicals by streams and rivers does not mean that pesticides have no effects on a variety of organisms living in the area of application. Pesticides in streams typically reach high levels for short periods of time after application, then decrease to very low or undetectable levels. During the brief period of high concentration, damage to

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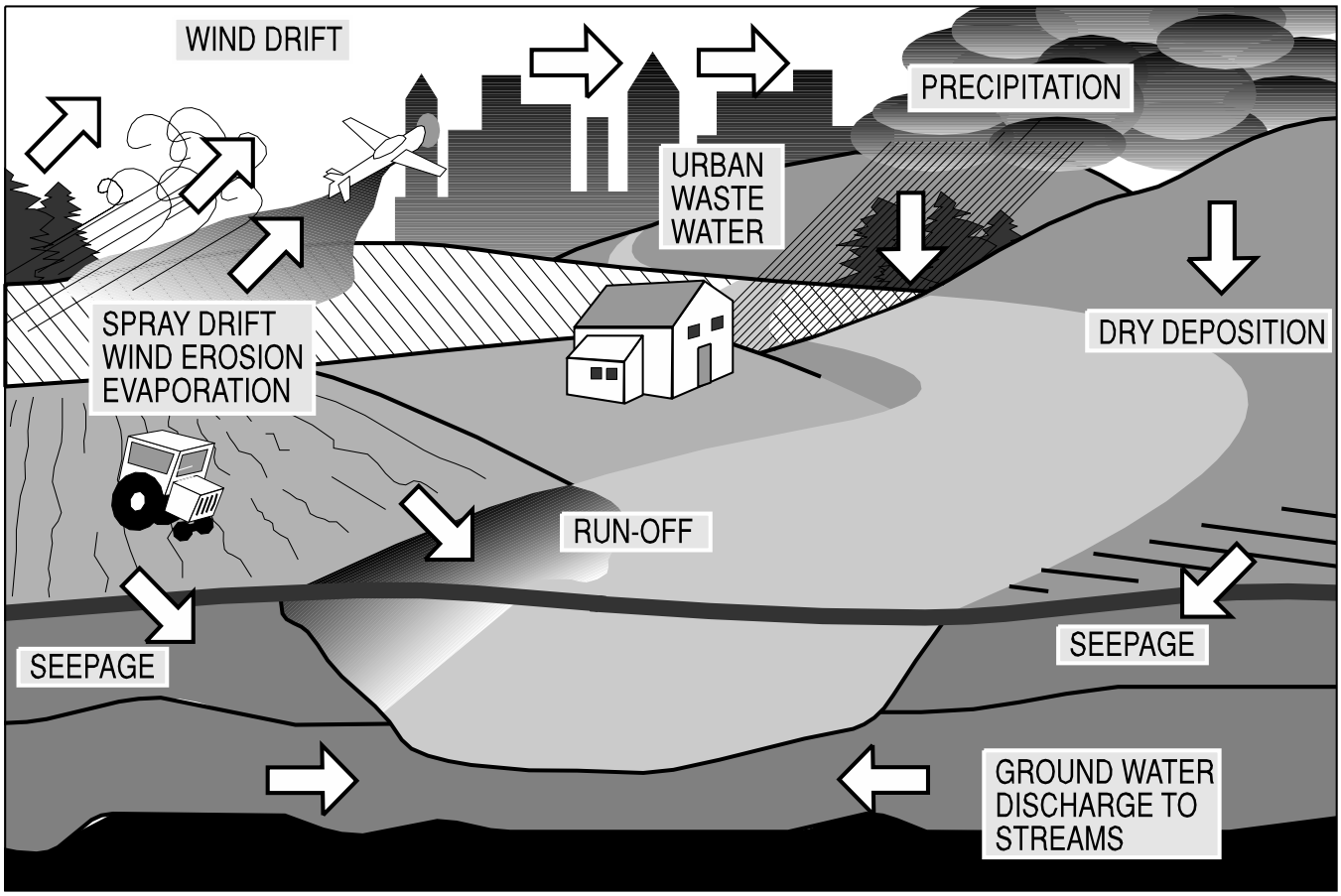
Pesticide residues move readily into streams and rivers following rainfall.

Figure 2. Routes through which pesticides move in the water cycle.



organisms in the stream may occur. Fish and wildlife at early stages of development are particularly vulnerable. Brief exposures to toxic materials at early stages in development may arrest future development of complete organ systems (Guillette et al. 1995). During early development, the sexual characteristics of the animal are formed, the immune system develops competence, and a variety of hormones regulate the formation of bone structure and organ systems. Disruption of any of these complex systems at critical times can lead to permanent impairment.

Pesticides in streams are rapidly diluted by riverflow so that soon after entry into a stream they can be detected only at very low levels or no longer be detected at all. While the concentrations in streams may be relatively harmless during rain and high water flow, under certain conditions pesticides are concentrated rather than diluted. Depending on the nature of the pesticide, they may attach to sediment particles, accumulate in the tissues of various organisms, or become buried in the sediment (Barbash and Resek 1996). Eventually, all probably reach the sea, where they become a problem of relatively unknown magnitude. If pesticides are applied in huge amounts each year for dozens of years, the dilution effect begins to disappear as the chemical concentrations increase in their particular final resting place. Thus, spraying of hillsides with a certain pesticide may not affect the animals in the immediate vicinity but may have disastrous effects on

other populations downstream from the site of application.

Most pesticides reach the water bound to soil particles (Barbash and Resek 1996). Erosion, which in the United States is estimated to transport four billion tons of soil a year into waterways, is a major contributor of pesticides to rivers and river beds. Thus, poor land-use practices not only contribute buildup of silt to the spawning areas in streams which results in suffocation of eggs. Erosion may also deliver pesticides to the rivers at a time when the fish, as embryos, are most sensitive to their deleterious effects.

Pesticides can be taken up from the water and accumulated in tissues of aquatic organisms, often becoming magnified thousands of times higher in the organism than in the surrounding water.

Pesticide Concentrations Can Become Magnified in Tissues

Under certain circumstances, pesticides can be taken up from the water and accumulated in tissues of aquatic organisms, often becoming magnified thousands of times higher in the organism than in the surrounding water. Usually biomagnification is a function of the fat solubility of the pesticide (Norris et al. 1991). Biomagnification is thus dependent upon the metabolic state of the animal, such that fatter animals tend to accumulate more lipid soluble pesticides. This reaches an extreme in the lipid-rich eggs and embryos.

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Erosion, which in the United States is estimated to transport four billion tons of soil a year into waterways, is a major contributor of pesticides to rivers and river beds.

Eggs of aquatic organisms usually have enough fats in the form of yolk to meet early energy requirements during development. Lipid soluble pesticides accumulate rapidly in these fat deposits and are not easily removed. There they may compete with hormones provided maternally for use by the embryos (Bern 1990). The presence of environmental contaminants in the yolk that act as endocrine mimics thus may have a powerful influence on embryonic development.

Not only are pesticides taken up directly from the environment, fishes can also absorb them from food organisms. A study of DDT accumulation in brook trout (Macek and Korn 1970) found that ten times more of the available DDT and its metabolites were absorbed from food than from the water. This can be magnified still further as the pesticide travels through food chains. Metcalf et al. (1971) demonstrated this biomagnification effect convincingly with radioactive DDT in a model ecosystem. C^{14}-DDT was applied to *Sorghum* as the source for the pesticide. The *Sorghum* was then

Table 2. Bioaccumulation of various pesticides, pesticide additives, and pesticide contaminants in fishes.

Pesticide	Category	Bioaccumulation Factor	Species	Reference
Carbaryl (Sevin)	Insecticide	30	golden ide	1
Chlordecone	Insecticide	1100-2200	fathead minnow	2
Chlorothalonil	Fungicide	840	rainbow trout	3
Chlorpyrifos (Dursban)	Insecticide	1374	rainbow trout	4
Cypermethrin	Insecticide	700-1000	rainbow trout	5
Diazinon	Insecticide	60	carp	2
Dichlobenil (Casoron)	Herbicide	40	golden ide	1
Fenvalerate	Insecticide	40-200	salmon	6
Flucythrinate	Insecticide	3000-5000	fathead minnow	6
Hexachlorobenzene	Contaminant of chlorothalonil, picloram, and pentachlorophenol	5500	rainbow trout	7
Nonyl phenol	Surfactant	1300	stickleback	8
Pentachlorophenol	Insecticide	251-5370	rainbow trout	2
Permethrin	Insecticide	1700-3300	fathead minnow	6
		73	Atlantic salmon	9
Pentachlorophenol	Fungicide	100	rainbow trout	10
2,3,7,8-TCCD	Contaminant of Dacthal, and 2,4-D	28,000	rainbow trout	11

1. Freitag et al. 1985.
2. Howard 1991.
3. World Health Organization 1996.
4. Racka 1993.
5. Hill 1985.
6. Smith and Stratton 1986.
7. Veith et al. 1979.
8. Ahei et al. 1993.
9. McLeese et al. 1980.
10. Hattula et al. 1981.
11. Mehrle et al. 1987.

introduced into a system with several components that provided a food web. *Sorghum* was eaten by a salt marsh caterpillar. Excreta from the caterpillar was consumed by diatoms, which were subsequently eaten by nine species of plankton. The plankton was eaten by mosquito larvae which were subsequently eaten by *Gambusia,* the mosquito fish. After one month, 54% of the radioactivity was found in the fish, of which most was DDE, a breakdown product of DDT. The concentration of DDT was 84,000 times greater in the fish than in the water, while the concentration of DDE was 110,000 times greater in the fish than in the water. Biomagnification of concentrations of DDT and its metabolites has been well established (Woodwell et al. 1967; Risebrough et al. 1967), which was a major factor leading to its removal from the pesticide market in the United States.

Modern pesticides are usually more water soluble and do not accumulate in high concentrations in fat deposits. However, they do show bioaccumulation (Table 2). While these accumulations are not as dramatic as those of DDT, they show that the concentration of pesticide in the water may not be relevant in determining whether levels of pesticide will cause biological effects. These accumulations are usually

measured in whole animals. Localized concentrations of pesticides at the cellular level may be extremely high, but this is an area that has not been widely explored.

Bioaccumulation and biomagnification properties of pesticides in tissues represents a problem in our assessment of their toxicity. Without complete analysis of the pesticides and their breakdown products in aquatic organisms in a natural state, we are unable to determine the exact concentrations to which they are subjected. If aquatic life has been exposed for long periods of time, the concentrations within the tissues may be much higher than that of the surrounding water. In addition, exposure to many types of pesticides may lead to interactions between them that increase their toxicity. Little work has been done on the interactions between different pesticides, but available evidence suggests that these interactions can increase toxic effects (Koenig 1977; Cook et al. 1997).

Without complete analysis of the pesticides and their breakdown products in aquatic organisms in a natural state, we are unable to determine the exact concentrations to which they are subjected. If aquatic life has been exposed for long periods of time, the concentrations within the tissues may be much higher than that of the surrounding water.

Fish Kills and Acute Toxicity of Pesticides

Pesticides are capable of killing salmonids and other aquatic organisms quickly. These short-term, acute toxicities to fish have been studied extensively for most chemicals used in forestry, agriculture, manufacturing, and the home. Much of this information is provided by the chemical manufacturer in order to meet requirements by the Environmental Protection Agency and various state agencies.

Most of these acute toxicity studies report lethal amounts as LC_{50}s, the concentrations of chemicals that kill 50% of the test animals within 48 or 96 hours, or LD_{50}s, the doses of chemicals in milligrams per kilogram body weight of the test animal which kill 50% of the animals within 48 or 96 hours. Organisms used for these tests range from algae to mice and rats. Rainbow trout are a common subject, as are bluegills, fathead minnows, mosquitofish (*Gambusia*), and zebrafish (*Brachydanio rerio*). A number of LC_{50}s for various fish are available in the literature (e.g., Norris et al. 1991; Anderson et al. 1997) or on the internet (e.g.,http://ace.orst.edu/info/extoxnet/pips/ghindex.html). In the interest of space, they will not be reproduced here.

The herbicide acrolein killed approximately 92,000 steelhead, 114 juvenile coho, 19 resident rainbow trout, and thousands of nongame fish in the Rogue River Basin.

Acute toxicity studies are usually performed on subadult or adult fish. Few of these studies have used eggs or fry, even though studies from the Great Lakes have shown that exposure during this stage of life can lead to profound results. Cook et al. (1997) found that the toxicities of congeners of TCDD injected into eggs of lake trout were additive and effective at much lower dosages than with juvenile fish. In general, embryonic stages are the most sensitive to environmental pollutants (Guillette et al. 1995). This is an area of research in western salmonids which has been overlooked in the past and needs immediate consideration.

When pesticides in water supplies exceed their lethal concentrations, the results are immediate. Large numbers of fish are killed, and these are reported to a variety of federal and state agencies. The spill is cleaned up when possible and the responsible parties are fined. An example of this regulatory action comes from southern Oregon. The Rogue River has received a number of inadvertant spills of pesticides, particularly acrolein, an herbicide used for removal of aquatic vegetation. This herbicide is very toxic to fish. Lorz et al. (1979) reported a 1977 release of treated irrigation water containing Magnicide H, a gaseous form of acrolein, into the Rogue River within 24 hours of treatment instead of the recommended holding time of 6 days. A 10 mile section of the river was affected. ODFW officials estimated that 238,000 fish were killed, including 42,000 salmonids with an estimated value of $284,000.

On May 9, 1996, another large fish kill occurred in a four-mile stretch of Bear

Creek, a tributary of the Rogue River, Oregon. A head gate was found open on an irrigation canal which had been treated with acrolein. The acrolein was used to remove aquatic vegetation that grew in the canal and interfered with its operation. Approximately 92,000 steelhead, 114 juvenile coho salmon, 19 resident rainbow trout, and thousands of nongame fish were killed within a short period of time after exposure. Talent Irrigation District was fined $356,000 for the loss of steelhead by Oregon Department of Fish and Wildlife. They were also fined $50,000 by the Oregon Department of Environmental Quality and an additional fine of $407 by the Oregon Department of Agriculture for allowing the pesticide to enter the stream (Evenson 1998).

Deaths of threatened and endangered species from inadvertant contamination of waterways are of grave concern because the loss of each individual in a sensitive population makes recovery efforts that much more difficult.

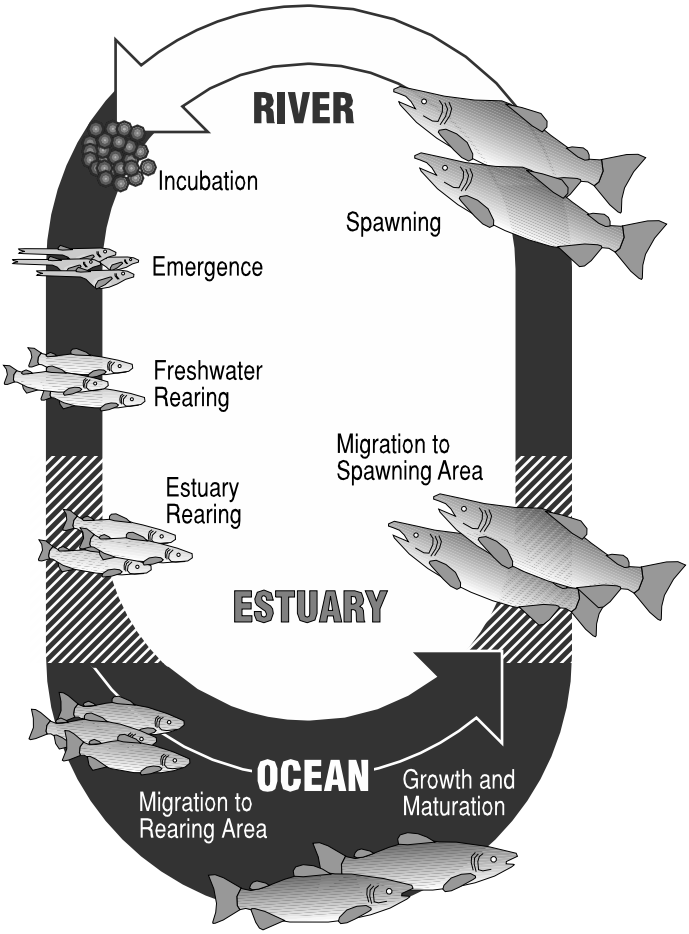
Deaths of threatened and endangered species from inadvertant contamination of waterways are of grave concern because the loss of each individual in a sensitive population makes recovery efforts that much more difficult. Fortunately, these events are relatively infrequent. By contrast, pesticide contamination at sublethal levels are probably an even greater danger to salmonid populations because the contamination is poorly regulated, the mortalities go unseen, and the consequences are unknown.

Behavioral Effects of Pesticides at Sublethal Concentrations

Behavior of salmonids is specifically directed at the continuance of the species. Each aspect of behavior has been highly selected over the generations to contribute to survival. (See Figure 3.) Interruptions of these behavior patterns will therefore reduce survival and diminish populations.

Pesticides at sublethal concentrations have been shown to disrupt many of the behavior patterns of both juvenile and adult salmonids and probably have deleterious impacts on their survival. Examples of these disruptions are presented below, although their impact on survival of fish populations is unknown at this time.

Figure 3
Salmon life cycle.



Salmon and many trout have developed anadromy, a behavior pattern in which juveniles migrate to the sea. In the sea, juvenile fish feed and grow before returning to the stream where they were born to lay their eggs.

Pesticides Impair Swimming Performance

The ability to feed, to avoid predators, to defend territories, and to maintain position in the river system are all dependent upon the swimming ability of the fish. A decrease in swimming performance reduces the ability of the fish to survive and compete with others. Swimming performance is commonly measured in swim tubes where the fish is forced to swim against a gradually increasing current. The current velocity at which the fish loses its position is referred to as critical swimming speed. The time of swimming at a particular velocity before it loses its position is referred to as swimming stamina. Both are used as indications of swimming performance.

Exposure to sublethal concentrations of pesticides often causes a loss in swimming performance. The fungicide TCMTB, which is used to prevent fungal staining of logs, causes deleterious effects on the swimming ability of salmonids. Chinook salmon and rainbow trout juveniles exposed to 5-20 ppb TCMTB for 48 hours, then removed to clean water for 12 hours, showed a dose-depen-

dent reduction in critical swimming speeds (Nikl and Farrell 1993). A further study with coho salmon at the same concentrations indicated that the reduction in critical swimming speed was dependent on both the concentration of TCMTB and the time of exposure to the chemical (Nikl and Farrell 1993). Damage to gill structure also increased with concentration and time of exposure. The researchers speculated that gill damage prevented respiratory exchange and that the decrease in oxygen availability was at least partially responsible for the decrease in swimming speed.

Colquhoun et al. (1984) tested the swimming stamina of brown trout exposed to the insecticide naled for 24 hours at a concentration of 84 ppb. The stamina of the exposed fish was reduced 57% compared to controls. Paul and Simonin (1996) felt that this exposure time was unrealistic when compared to the actual exposure times from aerial application of pesticides over streams. They held brook trout in water containing 23 and 46 ppb naled for 6 hours and found no change in swimming stamina. However, they found that both an insecticide formulation containing resmethrin and Scourge, a synergised formulation with resmethrin and piperonyl butoxide, did affect swimming stamina. Brook trout that were held in solutions of 3.2 ppb synergised and non-synergised resmethrin for 6 hours had significantly reduced stamina. Little et al. (1990) examined behavior of rainbow trout exposed for 96 hours to sublethal concentrations of five agricultural chemicals: carbaryl, chlordane, 2,4-D amine, methyl parathion, and pentachlorophenol. All chemicals inhibited spontaneous swimming activity and swimming stamina.

Rainbow trout exposed to tributyltin oxide, an anti-fouling agent used in boat paint, lost their ability to orient themselves in a current (Chliamovitch and Kuhn 1977). Rainbow trout exposed to Aqua-Kleen, 2,4-D butoxyethanol ester, also lost orientation to currents with increasing concentrations (Dodson and Mayfield 1979a). At near lethal concentrations, the orientation became variable. The most evident behavior change was a lethargy that increased with greater concentrations of the herbicide.

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Studies show that low concentrations of certain pesticides impair the swimming performance of rainbow trout.

Half of juvenile Atlantic salmon subjected to 1 ppm of the insecticide fenitrothion for 15-16 hours lost the ability to maintain territories for a period of six days after exposure (Symons 1973). Exposure of rainbow trout to 46 ppb of Vision, a glyphosate herbicide, for one month caused increased aggressive behavior (Morgan and Kiceniuk 1992). Rainbow trout exposed to the synthetic pyrethroids, fenvalerate and permethrin, tended to swim at the water surface, coughed repeatedly (attempting to clear the gill surface) and increased gill movements, suggesting respiratory distress (Holcombe et al. 1982). Those exposed to the organophosphate pesticides, Dursban (chlorpyrifos) and disulfoton, became darker and showed indications of muscle spasms and skeletal deformations. Both pesticides are cholinesterase inhibitors and the reactions were attributed to blocked

neural transmission. Trout exposed to the chlorinated hydrocarbon, Kelthane (dicofol), became dark, lethargic, and showed signs of respiratory distress, suggesting both respiratory and neurotransmission problems (Holcombe et al. 1982).

These experiments show that pesticides at very low concentrations can interfere with swimming performance in salmonids and that these effects can last for some time after exposure to the chemicals.

These experiments show that pesticides at very low concentrations can interfere with swimming performance in salmonids and that these effects can last for some time after exposure to the chemicals. The evidence comes from laboratory experiments and would be very difficult to replicate in the field. If similar effects occurred in field situations, however, interference with swimming performance would result in increased death from predation.

Pesticides Can Increase Predation on Juvenile Salmonids

The end result of any decrease in swimming ability will be an increase in predation upon the juvenile salmonids. If a young salmonid is slowed in its escape responses by ion imbalances, metabolic problems, or disease, it becomes prey for a number of hungry predators. Its demise is not immediately evident without sophisticated predator tests. Several studies have demonstrated that pesticides can indeed increase predation.

For example, Kruzinski and Birtwell (1994) found that juvenile coho salmon exposed to sublethal concentrations of the fungicide TCMTB showed fright and escape responses similar to controls in a simulated estuarine environment. After five days, however, TCMTB-treated fish had been preferentially consumed by introduced rockfish 5.5 times more frequently than controls. In another study, after a 24-hr exposure to 1.0 ppm fenitrothion, Atlantic salmon parr were more vulnerable to predation by brook trout than controls (Hatfield and Anderson 1972). Exposure to DDT had no effect on the predation. Little et al. (1990) found that carbaryl and pentachlorophenol increased vulnerability of rainbow trout to predation.

Predator avoidance tests with coho (Olla et al. 1992) and chinook salmon (Olla et al. 1995) indicated that stresses causing increased plasma cortisol levels resulted in susceptibility to predation, but that predator avoidance in coho salmon returned within 90 minutes (Olla et al. 1992) or 4 hours (Olla et al. 1995). In chinook salmon, predator avoidance was recovered in 24 hours after exposure to stress. While acute stresses seem to render the juveniles susceptible to predation, the effects of chronic stresses from pesticide exposure are not well known. Exposure of juvenile coho salmon to sublethal levels of two triclopyr herbicides for four hours did not result in significant changes in secondary stress effects, such as respiration, plasma glucose and lactate, and hematocrit (Janz et al. 1991). No plasma cortisol levels were measured, but these usually correlate well with plasma glucose concentrations. However, exposure to sublethal concentrations

of the butoxyethanol ester of 2,4-D caused enlargement of the interrenal gland, an indication that cortisol production was increased (McBride et al. 1981). If chronic exposure to pesticides does cause increase stress responses in salmonids, the studies of Olla et al. (1992, 1995) suggest that predation will be much greater in exposed fish.

Temperature Selection is Changed by Pesticides

Most organisms respond to the presence of pathogens through temperature selection. Cold-blooded animals such as lizards or fish typically seek warmer environments to increase metabolic repair when subjected to toxicants or infections. In salmonids, studies showed that the temperature selected depended on the concentration of the pesticides to which the fish were exposed. Juvenile Atlantic salmon exposed to low doses of DDT selected lower temperatures than controls (Ogilvie and Anderson 1965; Peterson 1973). At higher doses of DDT, exposed fish selected higher temperatures than controls. This effect was accentuated in fish acclimated to warm temperatures (17^0 C) compared to fish acclimated to cool temperatures (8^0 C). Fish acclimated to 17^0 C and exposed to 10 ppb or higher of DDT became hyperactive when introduced into cold water. Brook trout exposed to isomers of DDT and derivatives showed similar changes in temperature selection (Miller and Ogilvie 1975; Gardner 1973). The alteration in temperature selection by subyearling Atlantic salmon exposed to sublethal concentrations of DDT persisted for at least a month after the fish were transferred to clean water (Ogilvie and Miller 1976). Exposure to the organochlorine insecticide aldrin at a concentration of 100-150 ppb also caused Atlantic salmon juveniles to select lower water temperatures (Peterson 1973).

These results suggest that salmonids exposed to some pesticides select temperatures according to the concentration of pesticide. At low concentrations, fish try to lower their body temperature to minimize the effects on physiological processes. As the concentration increases, they respond by seeking warmer water to stimulate detoxification. These effects can last for a considerable time after the pesticide is removed, causing the fish to seek abnormal water temperatures and thus subjecting them to increased dangers of disease and predation.

Disruption of schooling behavior by many pesticides suggests that these compounds may increase predation upon juvenile salmonids and lead to population losses that would be very difficult to detect by conventional fisheries techniques.

Schooling Behavior is Reduced by Pesticides

Schooling behavior is a tactic to reduce predation. Predators are presented with a large mass of fish from which it is difficult to focus on a single individual (Cushing and Harden-Jones 1968). Seaward migration of juvenile salmonids is accompanied by schooling responses, probably to reduce losses to the population during the migration.

Many pesticides disrupt schooling behavior. Drummond et al. (1986) found

that loss of schooling behavior in fathead minnows was the most sensitive indicator of stress for 133 of 139 organic chemicals tested, including a number of pesticides. Exposure to sublethal concentrations of the fungicide TCMTB interferes with schooling behavior in chinook salmon (Kruzinski and Birtwell 1994). Exposure to methoxyclor caused a progressive increase in inter-fish distance in brook trout (Kruzinski 1972). Holcombe et al. (1982) found that schooling behavior was disrupted in fathead minnows exposed to sublethal concentrations of Kelthane, Dursban, disulfoton, fenvalerate, and permethrin.

Disruption of schooling behavior by many pesticides suggests that these compounds may increase predation upon juvenile salmonids and lead to population losses that would be very difficult to detect by conventional fisheries techniques. The prevalence of this effect by organic chemicals (Drummond et al. 1986) suggests that this sublethal effect may be more widespread than we might suspect.

Pesticides Can Interfere With Seaward Migration

Juvenile salmon rearing in streams reach a stage where their color changes from brown to silver. This is the stage of parr-smolt transformation where a variety of morphological, physiological, and behavioral changes occur. In addition to the silvery color, the smolts lose their territorial behavior, begin to school, and to migrate downstream to the sea. The mechanisms of this process have been widely debated and have been the subject of many local and international workshops.

The effects of pesticides on seaward migration have not been extensively studied because of the difficulty of measurement of migration tendency. Lorz et al. (1979) studied the effects of three herbicides on seaward migration in coho salmon. Diquat at concentrations from 0.5 ppm to 3.0 ppm inhibited seaward migration in a dose-dependent manner. Dinoseb inhibited migration only at the high (but sublethal) concentrations of 40 and 60 ppm. Tordon 101 (a mixture of picloram and 2,4-D) caused a slight inhibition of migration at the highest levels studied (1.2 and 1.8 ppm), but the results were not significantly different from controls. Although the herbicides were not lethal to the fish at the concentrations used, the levels were very high (ppm) compared to other concentrations of pesticides (ppb) described in this section. This may be due to the low toxicity of most herbicides, compared to other classes of pesticides. More work on seaward migration should be done with other pesticides, especially the organochlorine and organophosphate insecticides commonly found in western streams.

INSTITUTE FOR FISHERIES RESOURCES

Estuaries are a critical transition zone where salmon adapt to saltwater.

Pesticides Interfere with Seawater Adaptation

The end result of seaward migration is the entry of juvenile salmon into an estuary

and the tranformation of the fish's osmoregulatory mechanisms from a fresh water to a saltwater environment. These osmoregulatory processes permit the fish to maintain required water and ion concentrations in various organ systems. The stresses involved with this transition can be surmised from the large mortality (usually about 90% or greater for hatchery smolts) which results from seawater entry (Pearcy 1992). Death can result from several causes. The juvenile fish may simply not make the transition to seawater and die from osmoregulatory problems. The juvenile fish may be slowed by the osmotic stresses and be eaten by birds or predatory fish. The immune system of the fish may be compromised by the osmotic stresses, permitting the onset of disease and subsequent predation by piscivorous birds or fish.

Because the transition from fresh water to seawater is a critical period in the survival of the juvenile salmon, one would expect pesticides to have some of their greatest effects at this stage.

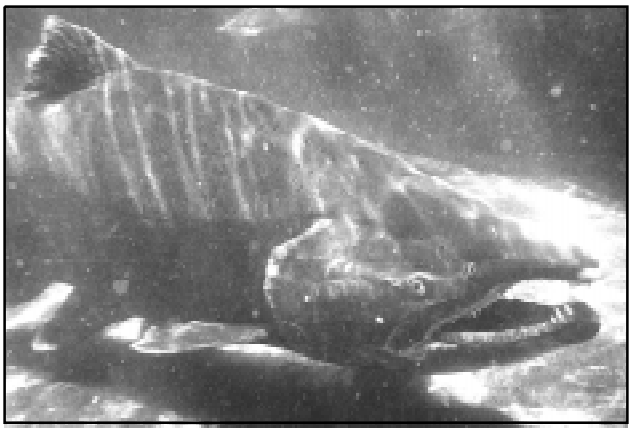
Most salmonids spend a period of transition in the estuary where they seek the salinities they prefer. Many utilize the less saline estuarine surface water which stratifies over deeper more saline water (Iwata et al. 1982; Birtwell and Kruzinski 1989).

Few studies have looked at the salinity preference of fish exposed to sublethal levels of pesticides. One would expect that the stress from pesticide exposure might cause the fish to select salinities comparable to that of their blood to relieve the extra burden of osmotic stress. Coho salmon in the stratified salinity tank set up by Kruzinski and Birtwell (1994) sought the freshwater wedge if they were previously exposed to TCMTB. Mosquitofish, however, sought a higher salinity if exposed to DDT (Hansen 1972). No change in salinity preference was induced by malathion (Hansen 1972).

In a study on the effects of herbicides on juvenile coho salmon, three of the thirteen herbicides studied, Amitrole-T, diquat, and paraquat, caused dose-dependent mortality in seawater tests (Lorz et al. 1979). An interesting phenomenon occurred in fish exposed to Tordon 101 (a mixture of 2,4-D and picloram). In December, fish were tested at eight different concentrations. The two lowest concentrations, 0.25 and 0.5 ppm, caused increased mortalities, while at higher concentrations up to 20 ppm all fish survived. When this was repeated in March, fish exposed to 20 ppm Tordon 101 all died in seawater. The researchers could not explain the unexpected mortalities at the low concentrations but suggested that pre-smolted coho salmon in December were less sensitive to environmental pesticides than those in smolted condition in March (Lorz et al. 1979). Mitchell et al. (1987) found that a 10-day exposure to 2.78 ppm of the herbicide Roundup (a glyphosate formulation) did not affect seawater adaptation or growth.

Because the transition from fresh water to seawater is a critical period in the survival of the juvenile salmon, one would expect pesticides to have some of their greatest effects at this stage. However, studies showing relationships between pesticides and osmoregulatory problems leading to mortality in salmonids are few. Most of the studies that have been done have focused on marine species or euryhaline species which do not undergo the

radical transformation of osmoregulation experienced by salmonids and other anadromous species. Studies on the effects of pesticides, especially organochlorine and organophosphate pesticides, on salmonids are needed in this area.



OREGON SEA GRANT

An adult chinook salmon that is ready to spawn.

Pollutants Affect Adult Returns

The result of interference with life history mechanisms of the juveniles should be the decrease in numbers of returning adults. Because the sublethal effects of pesticides on fish survival have not been fully appreciated, there are few studies that have correlated the return of adult salmon with the extent of pesticide use in the river basin. In New Brunswick, however, researchers have found that decreased returns of Atlantic salmon have been correlated with the use of pesticides in the area. Specific concern has focused on the compounds known as nonyl phenols, which are used not only as wetting agents in pesticides, but also in the production of paper, textiles, and petroleum products. Nonyl phenols are thought to act both by endocrine disruption and by interference with the normal adaptation of salmon smolts to seawater. While the evidence is convincing that the salmon runs have been affected by spraying, the experimental evidence has not yet been published in the scientific literature. A paper describing these effects has been submitted for publication (Fairchild 1999).

Migration patterns and timing of adult salmon may be altered by pollutants as they re-enter fresh water during their spawning runs. Elson et al. (1972) showed that radiotagged Atlantic salmon entering the unpolluted Tabusintac estuary in New Brunswick moved quickly up the estuary and into the river system. In contrast, salmon entering the Miramichi estuary, a heavily industrialized estuary, spent time swimming back and forth in an attempt to avoid heavily polluted water. They were never found in a channel on the northwest side of Beaubears Island, where a marsh at the lower end of the island served as a reservoir for effluent from a wood-preservative plant. This effluent seeped into the river at high tides. Salmon clung to the cleanest side of the river as they swam upstream past the industrial area. Clearly, the fish were slowed in their spawning migration as they searched for the cleanest parts of the river.

Respiration of adult coho salmon was examined as they attempted to pass a polluted estuary within the city limits of Seattle, Washington (Smith et al. 1972). They found that respiration decreased and blood lactate increased during swimming in the polluted area. They suggested that the salmon either avoided the areas of pollution, resulting in delayed passage times, or swam through the polluted areas, resulting in an oxygen debt and decreased energy reserves. Decreased energy reserves could result in smaller eggs and insufficient energy for passage into upstream spawning areas.

In summary, these studies on the disruption of behavior of salmonids by pesticides suggest that the presence of pesticides can have profound effects on the survival of the juveniles. Every aspect of their biology can be affected. The studies reported here consider mainly the freshwater portion of the salmonid life history. Exposure to pesticides continues in the estuaries and in the sea. For example, a major food source for juvenile salmon in the estuary is the amphipod *Corophium,* which spends the day buried in sediments which may be the final repository for many of the pesticides applied far upstream. It is likely that the amphipods accumulate pesticides and transfer them to the salmonids when eaten. Few studies have documented the behavior of juvenile salmonids once they reach the estuary. Even fewer have examined the effect of pollutants and pesticides on their behavior and survival.

Compromised Immune Systems

Fish in polluted waters are subject to more frequent and more severe outbreaks of disease because many synthetic chemicals tend to suppress the immune system (Moller 1985; Repetto and Baliga 1996). Immune systems protect fish from serious diseases caused by external bacteria, parasites, and viruses. The complexities of these systems are regulated and coordinated by a variety of compounds that act similar to hormones but that do not quite fit into the classical definition (Bern 1990). Most texts on molecular endocrinology include interleukins, cytokines, epithelial growth factors, and other compounds that influence the immune system as hormones (Bolander, 1994). These immunohormones can be compromised in various ways by environmental pollutants (Anderson 1996; Repetto and Baliga 1996).

Fish in polluted waters are subject to more frequent and more severe outbreaks of disease because many synthetic chemicals tend to suppress the immune system.

Although considerable work has been done on the immune system of fish, our understanding is still far from that we have gained about the intricacies of the human and rat immune mechanisms. As in mammals, fish have both cellular and humoral (antibody) responses to foreign agents, and exert both the B cell and T cell responses to these stimuli. It is generally agreed that size rather than age of fish determines the immunological maturity (Tatner 1996). In rainbow trout, a gradual increase in immune competence occurs during early growth, with non-specific immunity developing first, followed by cell-mediated and then humoral immunity. Full humoral immunity, the ability to produce circulating antibodies, develops at about 1 g in size.

A number of natural and anthropogenic factors suppress the function of the immune system. Stresses from habitat changes, poor water quality, lack of food, and pollutants all result in immunosupression and an increase in disease (Pickering 1993).

Information on immunosuppression in salmonids due to pesticides is scanty, although other fish have received more

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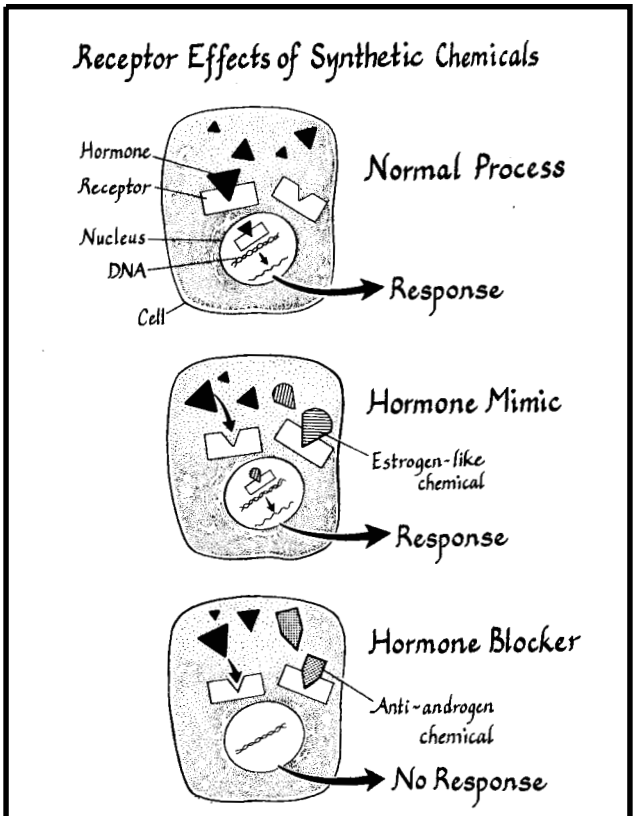
attention (Anderson 1996; Iwama and Nakanishi 1996). The insecticide endrin reduced the activities of phagocytes and antibody producing cells in rainbow trout (Bennett and Wolke 1987). Organotin compounds caused immune suppression in rainbow trout (O'Halloran et al. 1998). Although tributyltin is considered more toxic to fish than its breakdown product, dibutyltin, the latter was the more potent immunotoxin. Phenol and the PCB Arochlor 1254 were found to reduce antibody producing cells in rainbow trout (Anderson et al. 1984) and coho salmon (Cleland et al. 1989). TCDD caused mitogenic suppression in rainbow trout (Spitsbergen et al. 1986) and increased susceptibility to infectious hematopoetic necrosis virus (Spitsbergen et al. 1988).

The timing of exposure to pollutants may be an important factor for the health of the immune system in anadromous salmonids. Maule et al. (1987) found that immune suppression occurred to a greater extent during smolting of coho salmon. This may be due to the higher levels of the stress hormone, cortisol, found during smolting in salmonids (Specker and Schreck 1982; Thorpe et al. 1987). Cortisol is known to suppress the immune system of salmonids (Maule et al. 1987; Pickering and Pottinger 1989; Maule and Schreck 1990). It is likely that the effects of exposure to immunosuppressant chemicals during the period of seaward migration may be compounded by the high levels of plasma cortisol in the fish at that time. Many pesticides cause some degree of immunosuppression in mammalian systems (Repetto and Baliga 1996). It seems likely that similar effects may be found in the fish immune systems. Before the necessary research provides answers, it seems prudent to err on the side of caution and assume that similar immunosuppression by pesticides can be found in salmonids.

Endocrine Disruptors

In the late 70s, researchers Mike Howell and Ann Black found that their seine hauls from a small coastal stream in the panhandle of Florida contained mosquitofish with a strange characteristic. They were all males (Howell et al. 1980). On further inspection, another oddity was noted. Many of the apparently male fish had a gravid spot on their gonopodium, which normally indicates a female with internally developing young. Further analysis in the laboratory indicated that many of the apparent males were in fact masculinized females. A few months later, a second population of masculinized females was discovered in the Fenholloway River of coastal Florida (Bortone and Davis 1994), a stream approximately 300 kilometers east of the first stream. This collection included not only mosquito fish but also the least killifish and the sailfin molly. In both streams, the masculinized females were found downstream of paper mills. Some component of the effluent from paper mills caused the females to acquire masculine traits.



Endocrine-disrupting chemicals can interfere with normal functioning of the body's hormone system in a variety of ways. By mimicking or blocking hormones, the synthetic chemicals disrupt normal cellular communication, altering or preventing the appropriate biological response. Some endocrine disruptors can also interfere with chemical messengers involved with the immune system, hormone synthesis, neurotransmitters, and growth processes.

K. BORN

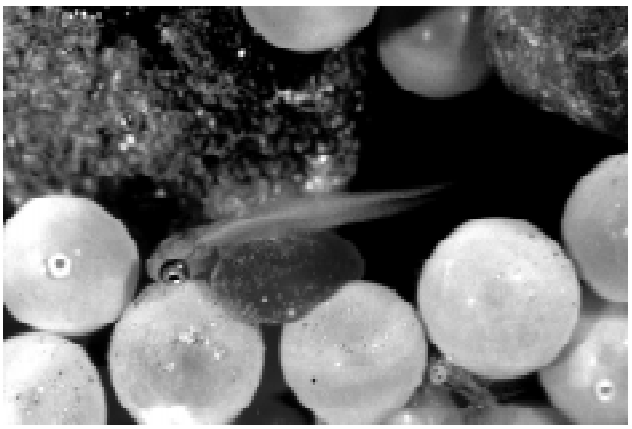
This research was one of many studies conducted over the past twenty years that indicated that certain synthetic chemicals, including many pesticides, can alter the sexual endocrine (or hormone) system of wildlife (Colborn and Clement 1992). The effects of these pseudo-hormones can vary considerably. In the above example, female fish became masculinized. Other pollutants act as artificial female sex hormones or estrogens. In still others, sexual expression is not affected. Instead, the chemicals interfere with bone formation during early development and result in stunted or malformed adults. As scientists gain greater insights into the complex workings of endocrine systems, they have come to appreciate the delicate balance of hormones needed to direct the early development of an embryo and the profound effects that endocrine mimics may produce.

Endocrine systems in an organism control production of hormones, which in turn control many of its functions. An endocrine system is a complex network of positive effectors and feedback loops that

carry messages from one part of the body to another to keep the body functioning properly. Initial input to the system comes from the environment in the form of temperature, light, or other sensory stimuli. These stimuli are recognized and integrated in the brain. Through a number of pathways, brain centers control the synthesis of primary hormones in the pituitary, a small bud of nerves and tissues at the base of the brain. In the reproductive cycle, the pituitary produces gonadotropins which act on the gonads to produce steroid hormones: androgens predominating for males and estrogens predominating for females. Steroids and other hormones travel through the blood to seek specific receptors in the cytoplasm of responsive cells. Each sex contains both male and female receptors in its cells so that the relative concentration of androgens and estrogens are the determining factor for sexual differentiation. When the steroid binds to the receptor protein, the steroid-receptor complex finds its way to the nucleus where it binds to DNA response elements to stimulate or regulate the production of steroid specific proteins.

Certain pesticides and pollutants can mimic the effects of estrogens, causing feminization of male animals.

In such a complex system, pesticides and their derivatives can interact at many levels in the brain-pituitary-target organ axis with a variety of effects. These effects can manifest themselves through behavioral, reproductive, immunological, or physiological changes (Bern 1990). Our knowledge of the hormonal relationships to many of these functions is not well enough defined at present to exactly determine what effects pollutants may have on these functions. However, our knowledge of the female endocrine system and the effects of pesticides on its function has grown rapidly in recent years, and numerous studies have defined some of the unwanted effects.



OREGON DEPT. OF FISH AND WILDLIFE

During early development, fish are especially vulnerable to pesticides that can mimic natural hormones.

Pesticides Can Mimic Estrogens

The sex of fishes can be determined relatively late in development. For example, applications of testosterone during the egg and fry stages of salmonids can convert the young fish completely to males or cause them to become sterile, depending on the concentration. Applications of estradiol, the female sex hormone, can cause production of 100% female salmon (Hunter and Donaldson 1983). In chinook salmon, Piferrer et al. (1994) suggested that aromatase, an enzyme that converts androgens to estrogens, acts as a regulatory switch for the process of sex differentiation. They showed that a treatment of young salmon with an aromatase inhibitor for only

two hours could transform genetic females into functional males. These transexual males had testes indistinguishable from normal males, underwent all stages of sperm maturation, and were later used to fertilize females to produce all female offspring. Concentrations of androgens and estrogens circulating through the blood provide a stimulus for induction of levels of aromatase activity (Callard and Callard 1987). With a system of sex differentiation in fishes that is labile (i.e., likely to change), the potential for major impacts from estrogen mimics may be quite large.

Certain pesticides and pollutants can mimic the effects of estrogens, causing feminization of male animals. Actions of pesticides that result in feminization in fish are thought to occur in three ways:

1) Estrogen mimics compete with estradiol, the female sex hormone, for its receptor protein. The complex that is formed enters the nucleus, binds the DNA response elements, and stimulates the responses expected from normal female sex hormones. This has little effect in females but stimulates inappropriate responses in males, which have only very low concentrations of circulating estrogens. The result is feminization of the males.

2) Antiandrogenic compounds compete with androgens, or male sex hormones, for the androgen receptor protein. Once bound, the complex enters the nucleus but is not able to bind to DNA response elements. Consequently, no androgenic response is stimulated. In the absence of an androgenic response, the fish assumes that it is a female and the result is feminization.

3) Compounds may interfere with neurotransmitters or signal transduction

OREGON DEPT. OF FISH AND WILDLIFE

Pesticide exposure at the fry stage can cause feminization of male fish or even sterility.

mechanisms in the brain. The interference results in an upset of the regulatory mechanisms that distinguish males from females and sends the wrong messages to the pituitary. The result may be the release of gonadotropins that stimulate feminization of the juvenile fish.

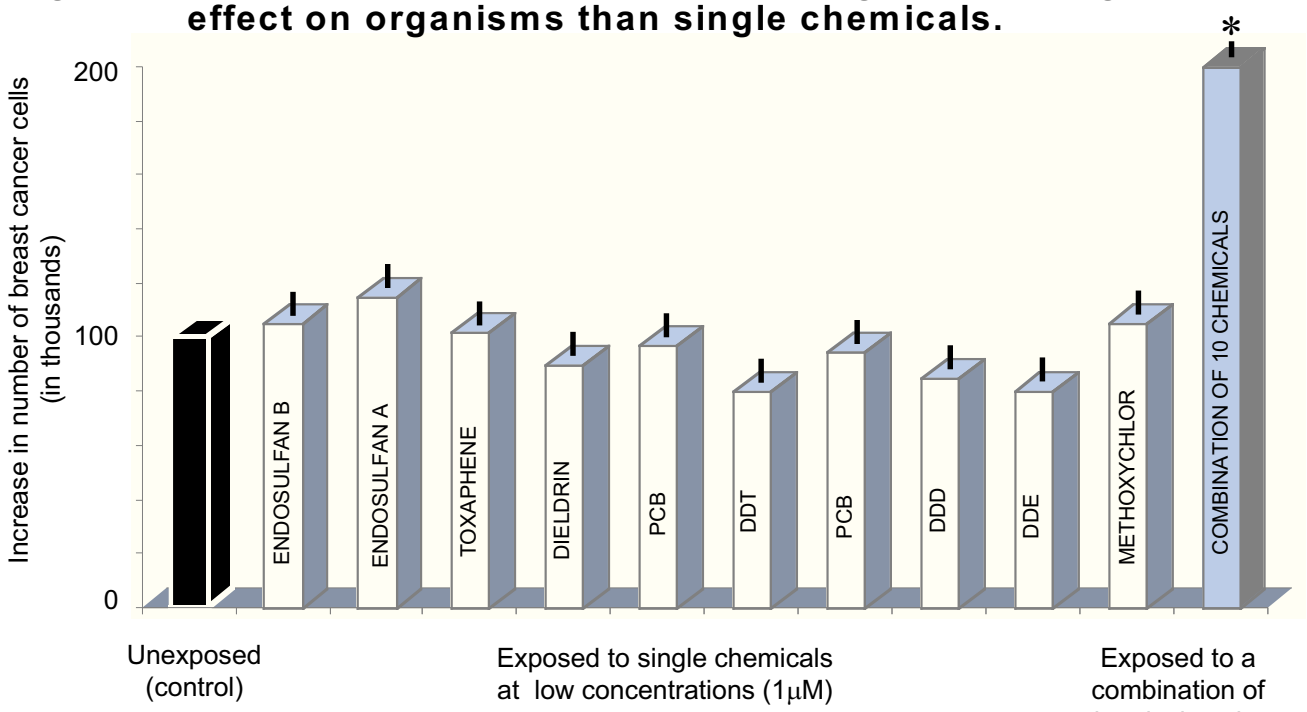
The best known examples of feminization by competition of pesticides for estrogen receptors have occurred with alligators in the Southeast (Guillette et al. 1995), sea gulls (Fry and Toone 1981) and Caspian terns (Fox 1992). Increasing numbers of chemicals are found to have estrogenic activity in fish (Jobling and Sumpter 1993; Jobling et al. 1995). Unknown chemical contaminants from sewage effluents have been shown to produce estrogenic responses in male carp (Folmar et al. 1996). DDT and its long-lived breakdown product, o,p'-DDE, have been shown to bind estrogen receptors to provide weakly estrogenic responses, whereas p,p'-DDE does not bind estrogen receptors except at high concentrations (Donohoe and Curtis 1996). p,p'-DDE

has a higher affinity for androgen receptors (Kelce et al. 1995), suggesting that the binding may be nonspecific for a number of steroid receptors. Lindane, chlordecone (Donohoe and Curtis 1996; Thomas and Khan 1997), alkylphenols (Jobling et al. 1995; White et al. 1994), endosulfan, dieldrin, and toxaphene (McLachlan 1993; Soto et al. 1994) have all been shown to provide estrogenic responses in fish and other organisms through binding to estrogen receptors.

Environmental compounds in combination may act together with the estrogen receptors to provide responses far greater than that of single compounds.

Most of these pesticides bind only weakly to the estrogen receptor. For example, the binding of the insecticides endosulfan, dieldrin, toxaphene, and chlordane to the estrogen receptor was very weak: less than 1/10,000 that of the natural binding compound, estradiol (McLachlan,1993; Soto et al. 1994). For this reason, these compounds were thought for a long time to produce few environmental effects. However, environmental compounds in combination may act together

Figure 4. Combinations of chemicals have a greater estrogenic effect on organisms than single chemicals.



Notes: Lines above the bars represent standard errors. * indicates that the increase in number of cells is significantly greater than unexposed cells. Proliferation of breast cancer cells is a standard test for estrogenic effects.

Source: Soto, A. M., K. L. Chung, and C. Sonnenschein. 1994. The pesticides endosulfan, toxaphene, and dieldrin have estrogenic effects on human estrogen-sensitive cells. *Environ. Health Perspect.* 102:380-383.

with the estrogen receptors to provide responses far greater than that of single compounds (see Figure 4).

Feminization of males may also be brought about by antiandrogenic compounds. Vinclozolin, a fungicide used widely for grapes, ornamental plants, snap peas, and turfgrass (Kelce et 1994), causes feminization of males. The fungicide binds to androgen receptors and enters the nucleus, but blocks normal androgenic responses. The vinclozolin-androgen receptor complex cannot recognize the appropriate DNA response elements to stimulate production of essential male proteins (Wong et al. 1995). DDE, the persistent break-down product of DDT, also binds to androgen receptors with similar results (Kelce et al. 1995). Antiandrogenic responses in fish are not well understood at present. Part of this difficulty is the problem of demonstrating androgen binding activity in fish tissues (Monosson et al 1997). The relationship of the active androgen in fish, 11-ketotestosterone, to receptors for testosterone in the nucleus is not clear (Pasmanik and Callard 1985; Callard and Callard 1987), although a receptor for 11-ketotestosterone in the ovaries of coho salmon has been reported (Fitzpatrick et al. 1994).

The peculiar lability of sex differentiation in fishes suggests that endocrine-disrupting chemicals in the environment may profoundly alter the sex ratios and breeding capabilities of our native fish.

The third category of hormonal disruption which may result in feminization by environmental pollutants arises from the interference with regulatory functions in the brain and pituitary. This interference can affect neurotransmitter functions, sensory systems, control of serum hormone levels and therefore hormonal feedback. We know little about the role of pesticides in this area, although there is ample information that shows that brain function is a necessary part of providing the correct hormones for sexual differentiation. Serotonin has been shown to cause release of gonadotropins from the pituitary in croakers (Khan and Thomas 1992) and goldfish (Somoza et al. 1988) so compounds that interfere with serotonin levels may affect the release of gonadotropins. Arochlor 1254 has been shown to cause decreases in serotonin and dopamine levels in the brains of fish (Kahn and Thomas 1996), due either to increased degradation or decreased synthesis of the neurotransmitters. Many insecticides are cholinesterase inhibitors, that is, they interfere with the normal breakdown of acetylcholine, a neurotransmitter, by blocking the enzyme cholinesterase. This results in a longer lifespan for acetylcholine and a disruption of normal neural function.

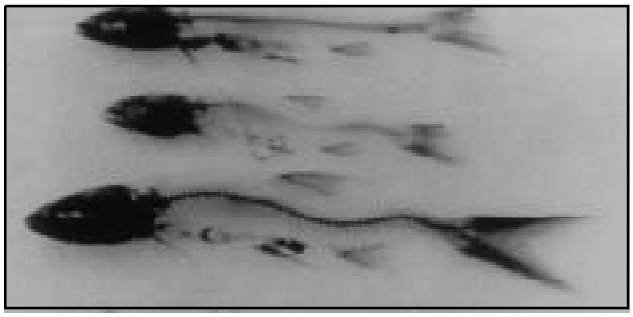
CORVALLIS ENVIRONMENTAL CENTER

The peculiar lability of sex differentiation in fishes (Chan and Yeung 1983) suggests that endocrine-disrupting chemicals in the environment may profoundly alter the sex ratios and breeding capabilities of our native fish. This field is still young and effects on fish population are largely unknown. With the development of more sophisticated methods for detection of pseudo-estrogens and pseudo-androgens in the environment, we should have a better understanding of the effects of chemicals that find their way into our watersheds.

Pesticides May Have Other Endocrine Effects

Although disruption of sex steroid function has received the most attention, it is not the only type of endocrine disruption that may result from exposure to environmental chemicals. Brucker-Davis (1998) listed over 90 synthetic chemicals that affected function of the thyroid gland. Thyroid hormones are thought to influence seasonal adaptations, reproduction, migration, and growth of fish. Hypothyroidism resulting in the formation of goiters was shown in coho and chinook salmon rearing in the Great Lakes and was related to environmental contaminants in the lakes (Moccia et al. 1977; Moccia et al. 1981). Thyroid impairment has been shown in the catfish *Clarias batrachus* after exposure to carbaryl (Sinha et al. 1991) and malathion (Sinha et al. 1992). Both malathion and carbaryl suppressed levels of thyroxine in the kidney, but increased thyroxine in the pharyngeal thyroid. Peripheral conversion of thyroxine to the active hormone, triiodothyronine, was suppressed with both compounds.

Skeletal deformities have long been recognized as an effect of environmental pollution and have been suggested as an excellent indicator of the extent of pollution (Bengtsson 1979). A comparison of reports of skeletal damage from old and new sources has given rise to the suspicion that water pollution may be having a larger impact on fish populations that we expect.



OREGON DEPT. OF ENVIRONMENTAL QUALITY

Squawfish from the Newberg pool in the Willamette River have a high incidence of skeletal deformities.

Bone formation and structure relies upon the mobilization and handling of calcium ions, which are controlled by a variety of hormones, including growth hormone, calcitonin, parathyroid hormone, and stanniocalcin (Davis 1997). These are in turn controlled by the brain-pituitary axis described earlier and subject to similar feedback mechanisms to regulate their synthesis and concentration. Unfortunately, calcium regulation mechanisms have not received the study that reproductive hormones have and consequently we know little about specific mechanisms underlying skeletal defects. Evidence that links environmental pesticides to skeletal abnormalities, however, is widespread.

In the early 1970s, fish biologists studying members of the drum family in James River, Virginia, found that a large number of specimens had shortened vertebral columns compared to similar species in the nearby York River. With the help of toxicologists, they found that a manufacturing

facility for chlordecone, a widely-used insecticide, had been releasing large amounts of the compound into the James River. The stunted fish contained high chlordecone levels (Davis 1997). Exposure to chlordecone interfered with calcium deposition into bone and produced scoliosis and compression of the vertebrae (Couch et al. 1977). After 1976, when manufacture of chlordecone had stopped, the frequency of vertebral abnormalities decreased.

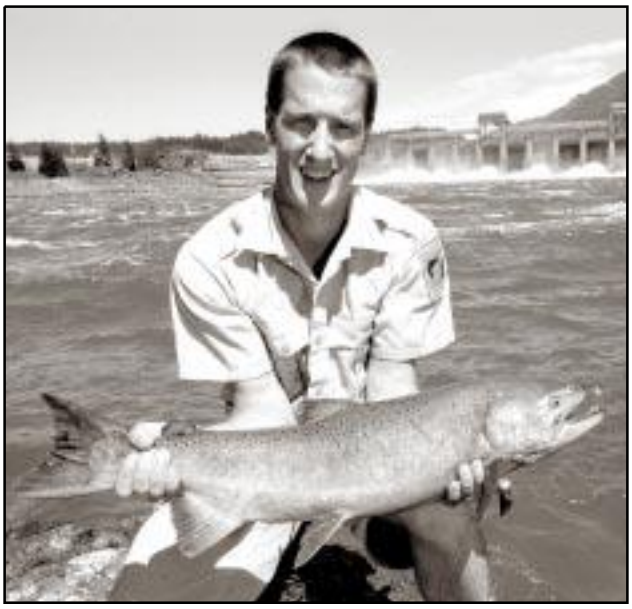
Wells and Cowan (1982) reported dysplasia in Atlantic salmon parr after a simulated spill of trifluralin, a pre-emergent herbicide, in the Eden River of Scotland. The fish were exposed to the herbicide for less than 1 day. Parr developed scoliotic vertebral abnormalities that caused them to appear stumpy. Laboratory tests showed that exposure to trifluralin resulted in hyperosteosis, a proliferation of the bone tissue (Couch et al. 1979). This was accompanied by fusion of vertebrae, persistence of osteoblasts, abnormal vertebral processes, and, strangely, the presence of notochord tissue in the vertebral canal. Clearly, the mechanisms of bone formation had been severely disrupted.

Trifluralin, a pesticide found in the Willamette River, has been shown to cause bone abnormalities in Atlantic salmon.

A number of other environmental pollutants, including chlorinated hydrocarbons (toxaphene, DDT), organophosphate pesticides (malathion, parathion, demeton), crude oil, and heavy metals (Bengtsson 1979), have been implicated in bone abnormalities in fishes. In most of these studies, researchers feel that the damage is done in the egg or early fry stages, possibly by effects on neuromuscular interactions during early development.

Recent studies on squawfish in the Willamette River have shown elevated numbers of abnormalities in bone formation, especially in the Newberg Pool (RM 26 to RM 60) (Altman et al. 1997). In the Newberg Pool, skeletal deformities ranged from 22.6 to 52.0 percent. No specific cause for the deformities has been identified, but a variety of factors including high temperatures, low dissolved oxygen, pesticides, PCBs, and effluents from pulp mills, paper mills, and ore smelters have been suggested. Analyses by the U. S. Geological Survey showed the presence of heavy metals (Altman et al. 1997), organochlorine pesticides, organophosphate pesticides, and even trifluralin in the Willamette River and its tributaries (Anderson et al. 1997). Although no skeletal abnormalities were found in salmonids in the watershed, the widespread occurrence of pesticides in Willamette River and the presence of skeletal abnormalities in squawfish should stimulate research on the potential endocrine-disrupting effects of the most common contaminants, such as atrazine, simazine, metolachor, and diuron. At the least, such abnormalities in the fish populations should provide a warning for the presence of endocrine disruptors in our watersheds.

Indirect Effects of Pesticides on Salmon



LYNN KETCHUM, OREGON STATE UNIVERSITY

Pesticides can indirectly affect fish by interfering with their food supply or altering the aquatic habitat, even when the concentrations are too low to affect the fish directly. Such indirect effects may greatly reduce the abundance of food organisms which in turn reduces the growth and probable survival of the fish.

Excellent examples of indirect effects of pesticides on salmonids were documented in the attempts to control spruce budworm in the forests of northeastern U. S. and Canada (Muirhead-Thomson 1987). In the mid 1970s, permethrin, a synthetic pyrethroid, was introduced to kill the spruce budworm. The insecticide was lethal to insects and moderately toxic to fish. Experimental sprayings of permethrin at 13 different streams between 1976 and 1980 did not result in moralities in native fish (Kingsbury and Kreutzweiser 1987). Blocking seines set at the bottom of the sprayed areas were never found to contain dead fish. Minnows and juvenile Atlantic salmon held in live cages in the sprayed area showed no mortality attributed to the spraying regimes.

Pesticides can indirectly affect fish by interfering with their food supply or altering the aquatic habitat, even when the concentrations are too low to affect the fish directly.

The same could not be said for the aquatic larvae in the streams, however (Kreutzweiser and Kingsbury 1987). Huge numbers of poisoned nymphs were found drifting downstream after spraying. Comparisons with pre-spray drift samples showed that the numbers of dead nymphs increased as much as 6000 times after spraying. This massive mortality was followed by a feeding frenzy among the salmonids. Atlantic salmon increased the number of nymphs in their stomachs by 2-4 fold. Brook trout fed even more voraciously, increasing their stomach contents by as much as 10-fold.

The poisoned insects seem to have no apparent direct effects on the salmonids, which fed as long as the drifting larvae were available. With time, however, the diets of the salmonids, especially the brook trout, changed considerably. With few of the normal food components of stonefly and mayfly nymphs and caddis larvae left in the streams, the trout changed to a diet of primarily *Dipteran* (fly) larvae and terrestrial insects.

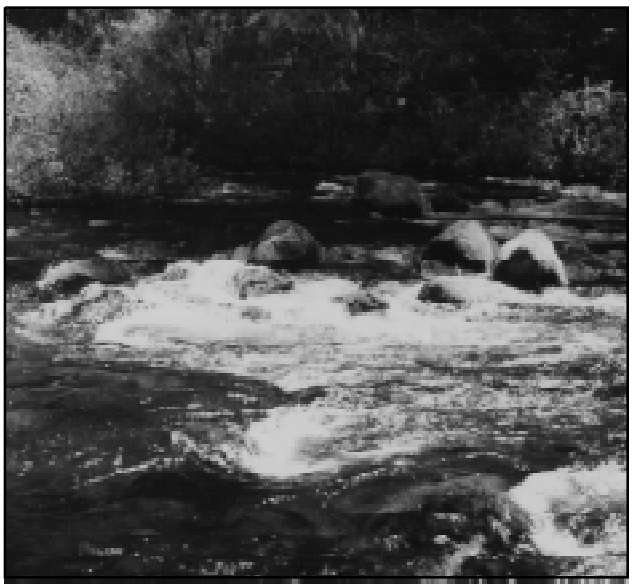
Densities of Atlantic salmon and brook trout were followed for a year after spraying. One month after spraying, the density of Atlantic salmon rearing in the area of highest application had decreased substantially. Brook trout populations did not show as great a decrease. It was felt that the reduced density of the Atlantic salmon was due to migration from the area in response to a reduced food supply.

Insect larvae and crustaceans are very sensitive to many types of pesticides, reducing the availability of these organisms to fish as prey (Macek et al. 1976; Brown 1978; Muirhead-Thomson 1987). Experimental spraying of ponds with carbaryl to simulate overspraying for spruce budworm control resulted in massive mortality of aquatic invertebrates (Muirhead-Thomson 1987). Some species of amphipods were not found for three years after the spray event (Gibbs et al. 1984). Walker (1964) found no fish mortality after application of 2-6 ppm of atrazine, a common herbicide, to ponds but insect larvae were killed immediately.

Pesticides may also indirectly affect prey species. Aquatic herbicides kill algae and diatoms on which aquatic insect larvae feed, thereby reducing the number of aquatic insects and decreasing growth and survival of fish feeding on the insects (Brown 1978).

In experimental treatments of replicate ponds with atrazine, deNoyelles et al. (1989) found that phytoplankton and aquatic plants were reduced by as much as 95% at the higher concentrations. Zooplankton did not seem to be directly affected. However, several months later, zooplankton populations also were reduced (deNoyelles et al. 1982). In the same study on atrazine, Dewey (1986) reported a decrease in the number of nonpredatory insects, while the predatory insect population was unaffected. She suggested that the decrease in nonpredatory insects was due to a reduction of algae and plants used as food and a reduction of water plants used as habitat for the insects. Further study of the fish populations indicated that the reproduction of bluegills was greatly reduced, probably from a lack of food. The stomach contents of the bluegills in the treated ponds were significantly lower than those in the control ponds (Kettle et al. 1987). Populations of young bluegills were further reduced by the lack of aquatic vegetation in which to hide. Without the aquatic vegetation, the young were preyed upon by the adult bluegills (Kettle et al. 1987).

At present, we have no information on whether deleterious effects of pesticides to algae (Stadnyk et al. 1971; Brown 1978; Bester 1995) or diatoms (Menzel et al. 1970; Brown 1978) may have trickle-down effects on salmonid populations, although it seems likely that they would be subject to population dynamic forces similar to those demon-



NEVA HASSANEIN

The integrity of aquatic ecosystems is essential for healthy salmon populations.

strated with bluegills. Interference with growth of salmonids, however, has deleterious effects on smolting (Ewing et al. 1980), immune function (Tatner 1996), predation (Parker 1971), seaward migration (Wedemeyer et al. 1980), and seawater adaptation (Folmar and Dickhoff 1980; Mahnken et al. 1982).

Increased predation on small fish may occur when water plants are killed by herbicides in the aquatic environment.

In addition to interference with their food supply, pesticides may also affect the habitat of the fish. Increased predation on small fish may occur when water plants are killed by herbicides in the aquatic environment (Lorz et al. 1979; Kettle et al. 1987). Adverse water temperatures may result from the removal of riparian vegetation by herbicides (Norris et al. 1991). Fish may avoid contaminated areas with a consequent reduction in availability and suitability of feeding and spawning areas (Beitinger 1990; Birge et al. 1993). Brown (1978) found that application of the herbicide paraquat caused death and decomposition of aquatic plants which in turn lowered the oxygen content of the water enough to kill trout.

These results and others of similar nature (Brown 1978; Muirhead-Thomson 1987) indicate that disruption of the food chains in aquatic ecosystems can have relatively long-lasting effects on fish populations. In complex ecosystems, indirect effects can be more important than direct effects (Lampert et al. 1989) and non-target organisms may be better indicators of ecosystem health than the organisms of interest. Management of pesticide applications for protection of aquatic habitats is obviously much more complex than promoting the use of LC_{50}s for setting application limits.

Conclusions

Salmonids require coordinated responses to environmental stimuli and internal physiological reactions for healthy development, territorial defense, smolting, seaward migration, and adaptation to seawater. Interruptions of this interplay may cause inappropriate physiological responses that have deleterious effects on survival of the salmon. As we have shown here, sublethal concentrations of pesticides can cause such interruptions and therefore result in harmful changes in physiology and behavior. The wide array of effects that have been demonstrated is cause for concern (Table 3).

The magnitude of the problem from pesticides is difficult to assess because of the lack of monitoring programs for the use and distribution of pesticides. Yet, where water quality monitoring has been done, pesticides are usually detected. A recent report commissioned by the Oregon Legislature (Botkin et al. 1995:104) concluded that fertilizers and pesticides were detri-

Table 3. Sublethal effects of selected pesticides found in the Willamette River Basin Study by USGS.[1]

	Herbicides		Insecticides		
	Atrazine	Simazine	Malathion	Chlorpyrifos	Carbaryl
Negative Effects on:					
Food Supply	+[2]		+[3]	+[4]	+[5]
Growth	+[2]		+[6]	+[7]	+[8]
Reproductive success	+[2]			+[9]	+[10]
Bone Abnormality			+[11]	+[12]	+[13]
Endocrine Disruptor			+[14]		+[13,14]
Immune System			+[15]		
Behavior		+[16]	+[17,18]	+[12,17]	+[12]
Schooling					+[19]
Predator Avoidance	+[20]		+[18]		

Plus indicates a detectable response from sublethal concentrations of pesticide. Blanks indicate that studies were not found analyzing these effects. Superscripts are references.

1. Anderson et al. 1997.
2. Macek et al. 1976.
3. Naqvi and Hawkins 1989.
4. Washino et al. 1972.
5. Burdick et al. 1960.
6. Hermanutz 1978.
7. Brazner and Kline. 1990.
8. Arunachalam and Palanichamy 1982.
9. Jarvinen et al. 1983.
10. Carlson 1971.
11. Weis, P. and J. S. Weis. 1976.
12. Holcombe et al. 1982.
13. Weis, J. S. and P. Weis. 1976.
14. Bruckner-Davis 1998.
15. Plumb and Areechon. 1990.
16. Dodson and Mayfield. 1979b.
17. Hansen 1969.
18. Hansen 1972.
19. Weis, P. and J. S. Weis. 1974.
20. Lorz et al. 1979.

mental to salmon: "With such large applications of pesticides and fertilizers, some portion is sure to make its way into nearby streams, especially since most agricultural land lies in close proximity to streams and rivers. *Since little monitoring is done to detect the presence of pesticides and fertilizers in streams, the levels of exposure by fish to chemicals are largely unknown* (authors' emphasis)."

Certain facts about pesticides in the environment are known and reviewed briefly here:

• Pesticides are typically found in great variety in salmonid habitats.

• Pesticides break down into products that may be less toxic, of equal toxicity, or of greater toxicity than the original compounds.

• Fish and other aquatic organisms must continue to cope daily with a variety of pesticides or breakdown products which were used years ago but remain in aquatic environments.

• Pesticides move in streams and rivers throughout the watersheds and may pose problems far from the site of application.

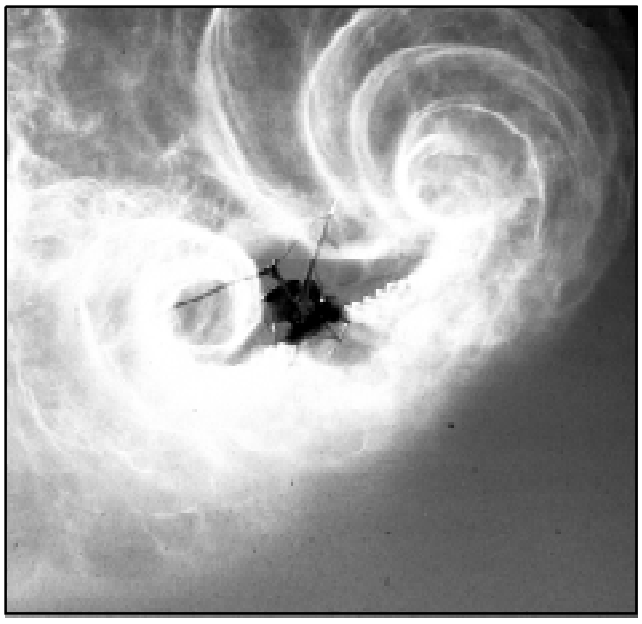
• In a process known as bioaccumulation, pesticides absorbed into plant and animal tissues may become concentrated and reach levels many times higher than the concentrations in the surrounding water.

The levels of pesticides encountered in streams and rivers have occasionally reached lethal concentrations for salmonids, as evidenced by the major fish kills that have occurred in the Rogue River Basin and elsewhere. Loss of each individual in a sensitive population thwarts our efforts to recover wild salmonids.

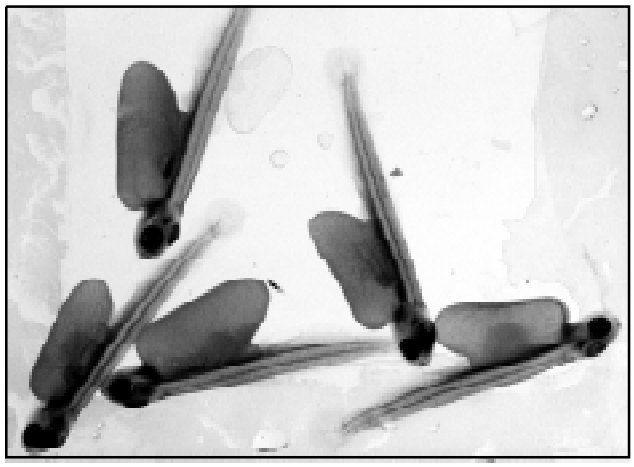
In contrast to these dramatic fish kills, sublethal concentrations of pesticides are more subtle and their effects are largely unseen. From laboratory experimentation, researchers have found that sublethal concentrations of pesticides can affect many aspects of salmonid biology, including swimming performance, predator avoidance, temperature selection, schooling behavior, seaward migration, immunity to disease, reproduction, and food supply. Specifically, the literature reviewed here shows that:

• A variety of pesticides impair swimming performance which can reduce such critical behaviors as the ability to feed, to avoid predators, to defend territories, and to maintain position in the river system.

• Chronic exposure to certain pesticides can increase stress in juvenile salmonids and thereby render them more susceptible to predation.



OREGON DEPT. OF FISH AND WILDLIFE



OREGON DEPT. OF FISH AND WILDLIFE

Pesticides can affect salmon biology at many stages in their development.

• Several pesticides have been shown to cause fish to seek abnormal water temperatures, thus subjecting them to increased dangers of disease and predation.

• Many pesticides interrupt normal schooling behavior of salmon, a critical tactic for avoiding predation during migration.

• Several herbicides have been shown to inhibit normal seawater migration patterns; however, there is a dearth of research looking at this effect for common insecticides.

• Several studies suggest that certain pesticides can impair salmonids' ability to transition from fresh water to sea water; however there is a need for further research in this area, placing particular emphasis on the critical period of transition that takes place in the estuary.

• As with other pollutants, pesticides may interrupt the spawning migrations of adult salmon by interfering with the timing of migration and by draining energy reserves.

• Pesticides can suppress the normal functioning of the immune system, resulting in a higher incidence of disease.

• Certain pesticides can act as hormone mimics or blockers, causing abnormal sexual development, feminization of males, abnormal sex ratios, and abnormal mating behavior.

• Pesticides can interfere with other hormonal processes, such as normal thyroid functioning and bone development.

• Fishes and other organisms are especially vulnerable to endocrine-disrupting effects during the embryonic and early development stages of life.

• Pesticides can indirectly affect salmonids by altering the aquatic habitat and food supply for salmonids.

The Ecoepidemiological Approach

An extensive literature on the effects of synthetic chemicals on survival, physiology and reproduction of fishes is available (Murty 1986). The literature is disparate, however, and largely fails to attribute causality to events occurring in particular aquatic ecosystems. A new approach to appreciate the dangers pesticides pose for salmonids may be necessary. One method that has proved useful to establish the relationship between chlorinated hydrocarbon concentrations and the decrease in lake trout populations in the Great Lakes is the application of principles borrowed from epidemiology (Mac and Edsall 1991).

Epidemiology is the medical science used to determine the causes of disease in a particular population when many confounding factors are present simultaneously. The subdiscipline used for examining environmental health hazards to wildlife populations is called ecoepidemiology (Morgenstern and Thomas 1993). In epidemiology, researchers form a central hypothesis about the cause of an outbreak of a disease. The validity of the central hypothesis is then tested by five criteria, as illustrated by Mac and Edsall's (1991) study of the Great Lakes' trout.

The hypothesis in Mac and Edsall's study was that large concentrations of chlorinated hydrocarbons were responsible for the reduced reproductive success in populations of lake trout. The first criterion is that the problem must be temporally related to the appearance of the causative agent. In the Great Lakes, during the period 1975 to 1988, survival of trout eggs improved significantly as concentrations of PCBs, DDT, and oxychlordane declined. The second criterion is that there must be a strong correlation between exposure to the hypothetical agent and the disease. In a study of egg and fry survival at different locations, the extent of egg and fry loss was directly proportional to the concentration of PCBs in the water and in female fish at each location. Thirdly, the causative agent must be shown to induce the disease. Tests done in various laboratories established that PCBs could decrease egg and fry survival. Fourth, the observed cause-effect relationship must be shown in repeated studies. Comparison of different species at the same time, the same species at different times, and the same species at different locations established that reproductive impairment correlated with PCB exposure. Finally, there must be a biological plausibility of the relationship between the causative agent and the symtoms. Laboratory studies of tissue histology, exposure to other chlorinated hydrocarbons, and studies of enzyme induction were all consistent with the hypothesis that PCBs and chlorinated hydrocarbons were impairing egg and fry survival.

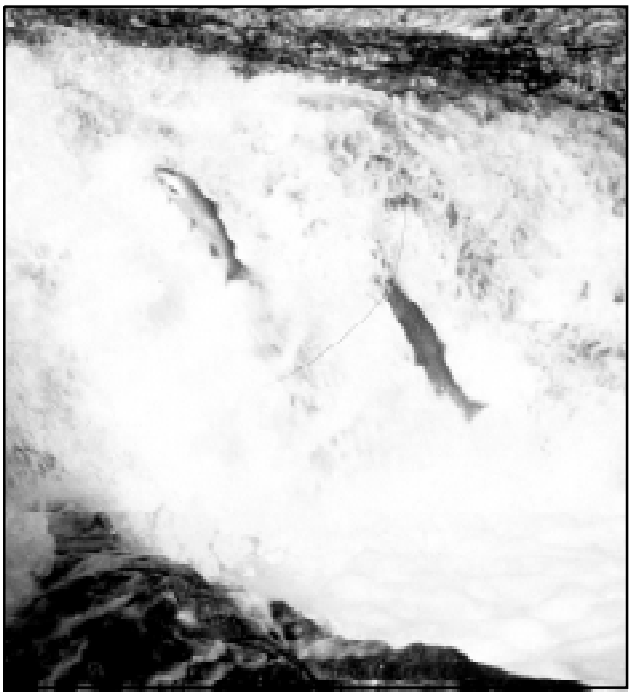
The authors concluded that their hypothesis was strongly supported, although they point out that "much of this evidence is circumstantial with little definitive proof of causality, which is why the epidemiological approach is used" (Mac and Edsall 1991). In other words, the strength of the ecoepidemiological approach is that it involves trying to understand relationships between environmental contaminants and population survival in real-world settings where a host of factors come into play. Restrictions on the use and dumping of organochlorine compounds into the Great Lakes resulted in partial restoration of the lake trout populations.

Recommendations

From the evidence available at present, it can be concluded that there is a plausible basis for considering pesticides as one causative factor in the decrease of salmon populations. Based on this review, we offer several policy recommendations and identify areas for further research:

1. Address the impacts of pesticides on salmon when developing and implementing recovery plans for threatened and endangered species. As federal, state, provinicial, and local agencies work to recover salmon in the Pacific Northwest, all factors contributing to the decline should be addressed. To date, pesticides have been overlooked as a factor deserving attention in our recovery efforts. Although there is a need for more information on the effects of pesticide use in salmon habitats, our salmon runs may be extinct by the time lengthy and laborious studies using strict scientific methods are complete. Thus, we must act now using available information to formulate management strategies that will minimize the potential danger from sublethal concentrations of pesticides.

2. Conduct ecoepidemiological studies in critical salmonid habitat. Most of the effects of pesticides referred to in this report have been determined in experimental laboratories. In the field, however, environmental conditions are not controlled, and many factors interact to confuse the determination of direct relationships. An ecoepidemiological approach (see inset opposite page) would be particularly valuable because it is designed to attribute causality to events occurring in real-world situations. This approach requires data which are not currently being collected. In particular, ecoepidemiological studies at the watershed level require specific pesticide use information in order to establish correlations between salmon decline and pesticide use.



INSTITUTE FOR FISHERIES RESOURCES

We must act now to protect salmon from pesticides.

3. Create comprehensive pesticide tracking systems in the Pacific Northwest. To better understand the relationship between pesticides and salmon decline, we must have accurate, site-specific data on the patterns of pesticide use in the watersheds of the Northwest. State and provincial governments need to collect data on which pesticides are used where, when, and in what amounts. Such data can then be combined with watershed-specific information on a host of physiological and behavioral indicators of salmon health. (Currently, California is the only state with salmon habitat where such information is collected.) Pesticide use information will also enable efficient instream monitoring for pesticide contamination.

4. Establish instream monitoring programs in critical salmon habitats. A systematic monitoring program for pesticides and their breakdown products needs to be undertaken. Clearly, not all pesticides can be tested for in all locations, but current testing is woefully inadequate for understanding the role of pesticides in salmon decline. In conjunction with pesticide use information, these analyses can be targeted to the compounds of most concern. Such targeting can greatly improve the cost-effectiveness of monitoring.

5. **Err on the side of caution when setting water quality standards for pesticides.** As discussed in this report, there are few established criteria for the protection of aquatic life from pesticides. Moreover, evidence reviewed here shows that sublethal effects on salmonids have not been fully appreciated, that juvenile salmonids succumb more easily to toxins in the water, that laboratory studies do not reflect the natural life cycle of the fish, and that little is known about how pesticides affect aquatic ecosystems. These factors must be considered when setting standards, and a precautionary approach must be adopted. Much can be gained by emphasizing how to eliminate the introduction of these toxic substances into the watersheds that comprise critical salmonid habitat.

6. Prevent pesticide contamination of salmonid habitat by reducing pesticide use. Once contaminated, water is difficult if not impossible to clean up. Therefore, pest management approaches that do not depend on pesticide use in agricultural and non-agricultural settings should be encouraged and further developed. There is ample evidence that ecologically sound and economically viable methods can be successfully implemented. The adoption of such alternatives can be encouraged through technical assistance, financial incentives and disincentives, demonstration programs, and information exchange opportunities.

7. Adopt state and provincial programs in the Pacific Northwest to phase out pesticides that persist and bioaccumulate in the environment. Numerous pesticides, including some that are no longer used and many that are currently used, are known to persist in the environment and to bioaccumulate in aquatic systems. Washington State's Department of Ecology is now considering a plan to end the release of such toxins, including certain pesticides, into the environment. To ensure salmon recovery, all state and provincial governments in the Pacific Northwest should adopt similar programs.

References

Ahei, M., J. McEvoy, and W. Giger 1993. Bioaccumulation of the lipophilic metabolites of nonionic surfactants in freshwater organisms. *Environ. Poll.* 79:243-248.

Altman, B., C. M. Henson, and I. R. Waite. 1997. Summary of information on aquatic biota and their habitats in the Willamette Basin, Oregon, through 1995. U. S. Geological Survey Water Resources Investigations Report 97-4023, Portland, OR. pp. 174.

Anderson, C. W., T. M. Wood, and J. L. Morace. 1997. Distribution of dissolved pesticides and other water quality constituents in small streams, and their relation to land use, in the Willamette River Basin, Oregon, 1996. Water Resources Investigations Report 97-4268, U. S. Geological Survey, Portland, OR.

Anderson, D. P., O. W. Dixon, and F. W. van Ginkel. 1984. Suppression of bath immunization in rainbow trout by contaminant bath pretreatments. pp. 289-293. IN: M. Kende, J. Gainer, and M. Chirigos, eds. *Chemical Regulation of Immunity of Veterinary Medicine.* Alan R. Liss, New York.

Anderson, D. P. 1996. Environmental factors in fish health: Immunological aspects. pp. 289-310. IN: Iwama, G. and T. Nakanishi, eds. *The Fish Immune System. Organism, Pathogen, and Environment.* Academic Press, New York.

Arunachalam, S. and S. Palanichamy. 1982. Sublethal effects of carbaryl on surfacing behavior and food utilization in the air-breathing fish, *Macropodus cupanus*. *Physiol. Behavior* 29:23-27.

Barbash, J. E. and E. A. Resek. 1996. *Pesticides in Ground Water. Distribution, Trends, and Governing Factors.* Ann Arbor Press, Inc., Chelsea, MI.

Beitenger, T. L. 1990. Behavioral reactions for the assessment of stress in fishes. *J. Great Lakes Res.* 16:495-528.

Bengtsson, B. E. 1979. Biological variables, especially skeletal deformities in fish, for monitoring marine pollution. *Phil. Trans. Roy. Soc. London* B 286:457-464.

Bennett, R. O. and R. E. Wolke. 1987. The effect of sublethal endrin exposrue on rainbow trout (*Salmo gairdneri* Richardson). I. Evaluation of serum cortisol concentrations and immune responsiveness. *J. Fish Biol.* 31:375-385.

Bern, H. A. 1990. The new endocrinology: its scope and impact. *Amer. Zool.* 30:877-885.

Bester, K., H. Huhnerfuss, U. Brockmann, and H. J. Rick. 1995. Biological effects of triazine herbicide contamination on marine phytoplankton. *Arch. Environ. Contam. Toxicol.* 29:277-283.

Birge, W. J., R. D. Hoyt, J. A. Black, M. D. Kercher, and W. A. Robison. 1993. Effects of chemical stresses on behavior of larval and juvenile fishes and amphibians. pp. 55-65. IN: Fuiman, L. A., ed. *Water Quality and the Early Life Stages of Fishes.* American Fisheries Society Symposium 14. American Fisheries Society, Bethesda, MD.

Birtwell, I. K. and G. M. Kruzinski. 1989. In situ and laboratory studies on the behavior and survival of Pacific salmon (genus *Oncorhynchus*). *Hydrobiologia* 188/189:543-560.

Bolander, F. 1994. *Molecular Endocrinology*, 2nd Edition. Academic Press, New York.

Bortleson, G. C. and D. A. Davis. 1997. Pesticides in selected small streams in the Puget Sound Basin, 1987-1995. U. S. Geological Survey Fact Sheet 067-97, U. S. Geological Survey, Tacoma, WA. 4 pp.

Bortone, S. A. and W. P. Davis. 1994. Fish intersexuality as indicator of environmental stress. *Bioscience* 44:165-172.

Botkin, D., K. Cummins, T. Dunne, H. Regier, M. Sobel, L. Talbot, and L. Simpson. 1995. Status and future of salmon of western Oregon and northern California. Findings and Options. Rept. #8. The Center for the Study of the Environment, Santa Barbara, CA.

Brazner, J. C. and E. R. Kline. 1990. Effects of chlorpyrifos on the diet and growth of larval fathead minnows, *Pimephales promelas,* in littoral enclosures. *Can. J. Fish. Aquat. Sci.* 47:1157-1165.

Brown, A. W. A. 1978. *Ecology of Pesticides.* John Wiley and Sons, New York

Brucker-Davis, F. 1998. Effects of environmental synthetic chemicals on thyroid function. *Thyroid* 8:827-856.

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

Callard, I. P. and G. V. Callard. 1987. Sex steroid receptors and non-receptor binding proteins. pp. 355-384. IN: D. O. Norris and R. E. Jones, eds. *Hormones and reproduction in fishes, amphibians, and reptiles.* Plenum Press, New York.

Carlson, A. R. 1971. Effects of long-term exposure to carbaryl (Sevin) on the minnow (*Pimephales promelas*). *J. Fish. Res. Bd. Can*. 29:583-587.

Chan, S. T. H. and W. S. B. Yeung. 1983. Sex control

and sex reversal in fish under natural conditions. pp. 171-222. IN: W. S. Hoar, D. J. Randall, and E. M. Donaldson, eds. *Fish Physiology.* Volume IX. Reproduction. Part B. Behavior and Fertility Control. Academic Press, New York.

Chapman, R. A., C. M. Tu, C. R. Harris, and C. Cole. 1981. Persistence of five pyrethroid insecticides in sterile and natural, mineral, and organic soils. *Bull. Environ. Contam. Toxicol.* 26:513-516.

Chliamovich, Y. P. and C. Kuhn. 1977. Behavioral, haematological and histological studies on acute toxicity of bis(tri-n-butyltin)oxide on *Salmo gairdneri* Richardson and *Tilapia rendalli* Boulenger. *J. Fish Biol.* 10:575-582.

Clark, G. M., T. R. Maret, M. G. Rupert, M. A. Maupin, W. H. Low, and D. S. Ott. 1998. Water quality in the upper Snake River Basin, Idaho and Wyoming, 1992-95. U. S. Geological Survey Circular 1160, U. S. Geological Survey, Boise, ID.

Cleland, G. B., P. J. McElroy, and R. A. Sonstegard. 1989. Immunomodulation in C56B1/6 mice following consumption of halogenated aromatic hydrocarbon-containing coho salmon (*Oncorhynchus kisutch*) from Lake Ontario, Canada. *J. Toxicol. Environ. Health* 27:477-486.

Colborn, T. and C. Clement, eds. 1992. *Chemically-Induced Alterations in Sexual and Functional Development: The Human/Wildlife Connection.* Princeton Scientific Publishing Co., Princeton, NJ.

Colquhoun, J. R., H. A. Simonin, H. J. Dean, and J. Symula. 1984. Swimming performance of juvenile brown trout exposed to acutely sublethal concentrations of naled. *New York Fish and Game J.* 31:133-137.

Cook, P. M., E. W. Zabel, and R. E. Peterson. (1997) The TCDD toxicity equivalence approach for characterizing risks for early life stage mortality in trout. pp. 9-27. IN: Rolland, R.M., Gilbertson, M., and R.E. Peterson, eds. *Chemically induced alterations in functional development and reproduction of fishes.* Setac Press, Pensacola, FL

Couch, J., J. Winstead, and L. Goodman. 1977. Kepone-induced scoliosis and its histological consequences in fish. *Science* 197:585-586.

Couch, J. A., J. T. Winstead, D. J. Hansen, and L. R. Goodman. 1979. Vertebral dysplasia in young fish exposed to the herbicide trifluralin. *J. Fish Dis.* 2:35-42.

Cushing, D. H. and F. R. Harden-Jones. 1968. Why do fish school? *Nature* (London) 218:918-920.

Davis, W. P. 1997. Evidence for developmental and skeletal responses as potential signals of endocrine-disrupting compounds in fishes. pp. 61-72. IN: Rolland, R.M., Gilbertson, M., and R.E. Peterson, eds. *Chemically induced alterations in functional development and reproduction of fishes.* SETAC Press, Pensacola, FL

Day, K. E. 1991. Pesticide transformation products in surface waters. Effects on aquatic biota. pp. 217-241. IN: Somasundarum, L. and J. R. Coats, eds. *Pesticide Transformation Products. Fate and Significance in the Environment.* American Chemical Society, Washington, D. C.

deNoyelles, F., W. D. Kettle, C. H. Fromm, M. F. Moffett, and S. L. Dewey. 1989. Use of experimental ponds to assess the effects of a pesticide on the aquatic environment. pp. 41-56. IN: J.R. Voshell, Jr., ed. Using Mesocosms to Assess the Aquatic Ecological Risk of Pesticides: Theory and Practice. Entomological Society of America, Misc. Public. 75.

deNoyelles, F., W. D. Kettle, and D. E. Sinn. 1982. The responses of plankton communities in experimental ponds to atrazine, the most heavily used pesticide in the United States. *Ecology* 63:1285-1293.

Dewey, S. L. 1986. Effects of the herbicide atrazine on aquatic insect community structure and emergence. *Ecology* 67:148-162.

Dodson, J. J. and C. I. Mayfield. 1979a. The dynamics and behavioral toxicology of Aqua-Kleen (2,4-D butoxyethanol ester) as revealed by the modification of rheotropism in rainbow trout. *Trans. Am. Fish. Soc.* 108:632-640.

Dodson, J. J. and C. I. Mayfield. 1979b. Modification of the rheotropic response of rainbow trout (Salmo gairdneri) by sublethal doses of the aquatic herbicides diquat and simazine. *Environ. Pollut.* 18:147-161.

Donohoe, R. M. and L. R. Curtis. 1996. Estrogenic activity of chlordecone, o,p'-DDT, and o,p'-DDE in juvenile rainbow trout: induction of vitellogenesis and interaction with hepatic estrogen binding sites. *Aquat. Toxicol.* 36:31-52.

Drummond, R. A., C. L. Russum, D. L. Geiger, and D. L. De Foe. 1986. Behavioral and morphological changes in fathead minnow (*Pimephales promelas*) as diagnostic endpoints for screening chemicals according to mode of action. pp. 415-435. IN: Poston, T. M., and R. Purdy, eds. *Aquatic Toxicology and Environmental Fate.* Vol. 9. ASTM STP 921. American Society for Testing and Materials, Philadelphia, PA.

Dubrovsky, N. M., C. R. Kratzer, L. R. Brown, J. M. Gronberg and K. R. Burow. 1998. Water quality in the San Joaquin-Tulare Basins, California, 1992-95. U. S. Geological Survey Circular 1159, U. S. Geological Survey, Sacramento, CA.

Elson, P. F., L. N. Lauzier, and V. Zitko. 1972. A preliminary study of salmon movements in a

polluted estuary. pp. 325-330. IN: M. Ruivo, ed. *Marine Pollution and Sea Life, Fishing News* (Books) Ltd, London.

Evenson, M. 1998. Personal communication, District biologist, Central Point, Oregon Department of Fish and Wildlife. October 22.

Ewing, R. D., H. J. Pribble, S. L. Johnson, C. A. Fustish, J. Diamond, and J. A. Lichatowich. 1980. Influence of size, growth rate, and photoperiod on cyclic changes in gill (Na+K)-ATPase activity in chinook salmon (*Oncorhynchus tshawytscha*). *Can. J. Fish. Aquat. Sci.* 37:600-605.

Fairchild, W. 1999. Personal Communication, Department of Fisheries and Oceans, Moncton, New Brunswick. January 26.

Fitzpatrick, M. S., W. L. Gale, and C. B. Schreck. 1994. Binding characteristics of an androgen receptor in the ovaries of coho salmon, *Oncorhynchus kisutch*. *Gen. Comp. Endocrinol.* 95:399-408.

Folmar, L. C., N. D. Denslow, V. Rao, M. Chow, D. A. Crain, J. Enblom, J. Marcino, and L. J. Guillette, Jr. 1996. Vitellogenin induction and reduced serum testosterone concentrations in feral male carp (*Cyprinus carpio*) captured near a major metropolitan sewage treatment plant. *Environ. Health Perspect.* 104:1096-1101.

Folmar, L. C. and W. W. Dickhoff. 1980. The parr-smolt transformation (smoltification) and seawater adaptation in salmonids. A review of selected literature. *Aquaculture* 21:1-37.

Fox, G. A. 1992. Epidemiological and pathobiological evidence of contaminant-induced alterations in sexual development in free-living wildlife. pp. 147-158. IN: T. Colborn and C. Clement, eds. *Chemically-Induced Alterations in Sexual and Functional Development: The Wildlife/Human Connection. Princeton Scientific Publishing Co., Inc., Princeton, New Jersey.*

Freitag, D. et al. 1985. Environmental hazard profile of organic chemicals. *Chemosphere* 14:1589-1616.

Fry, D. M. and C. K. Toone. 1981. DDT-induced feminization of gull embryos. *Science* 213:922-924.

Gardner, D. R. 1973. The effect of some DDT and methoxychlor analogs on temperature selection and lethality in brook trout fingerlings. *Pesticide Biochem. Physiol.* 2:437-446.

Gibbs, K. E., T. M. Mingo, and D. L. Courtemanch. 1984. Persistence of carbaryl (Sevin-4-Oil) in woodland ponds and its effects on pond macroinvertebrates following forest spraying. *Can. Entomol.* 116:203-213.

Guillette, L. J., Jr., D. A. Crain, A. A. Rooney, and D. B. Pickford. 1995. Organization versus activation: the role of endocrine-disrupting contaminants (EDCs) during embryonic development in wildlife. *Environ. Health Perspect*. Supp. 7 103:157-164.

Hansen, D. J. 1969. Avoidance of pesticides by untrained sheepshead minnow. *Trans. Am. Fish. Soc*. 98:426-429.

Hansen, D. J. 1972. DDT and malathion: effect on salinity selection by mosquitofish. *Trans. Am. Fish. Soc.* 82:346-354.

Hatfield, C. T. and P. H. Anderson. 1972. Effects of two insecticides on the vulnerability of Atlantic salmon (*Salmo salar*) parr to brook trout (*Salvelinus fontinalis*) predation. *J. Fish. Res. Bd. Can.* 29:27-35.

Hattula, M. L., V. M. Wasenius, H. Reunanen, and A. V. Arstila. 1981. Acute toxicity of some chlorinated phenols, catechols, and cresols to trout. *Bull. Environ. Contam. Toxicol.* 26:295-301.

Hermanutz, R. O. 1978. Endrin and malathion toxicity to flagfish (*Jordanella floridae*). *Arch. Environ. Contam. Toxicol*. 7:159-165.

Hill, I. R. 1985. Effects on non-target organisms in terrestrial and aquatic environments. IN: J. P. Leahey, ed. *The Pyrethroid Insecticides.* Taylor and Francis, London.

Holcombe, G. W., G. L. Phipps and D. K. Tanner. 1982. The acute toxicity of kelthane, Dursban, disulfoton, pydrin, and permethrin to fathead minnows *Pimephales promelas* and rainbow trout *Salmo gairdneri*. *Environ. Poll.* (Ser. A) 29:167-178.

Howard, P. H. 1991. *Handbook of environmental fate and exposure data for organic chemicals.* III. Pesticides. Lewis Publishers, Inc., Chelsea, MI

Howell, W. M., D. A. Black, and S. A. Bortone. 1980. Abnormal expression of secondary sex characters in a population of mosquitofish, *Gambusia affinis holbrooki*: evidence for environmentally induced masculinization. *Copeia* 1980:676-681.

Hunter, G. A. and E. M. Donaldson. 1983. Hormonal sex control and its application to fish culture. pp. 223-303. IN: W. S. Hoar, D. J. Randall, and E. M. Donaldson, eds. *Fish Physiology.* Volume IX. Reproduction. Part B. Behavior and Fertility Control. Academic Press, New York.

Iwama, G. and T. Nakanishi, eds. 1996. *The Fish Immune System. Organism, Pathogen, and Environment.* Academic Press, New York.

Iwata, M., T. Hirano, and S. Hasegawa. 1982. Behavior and plasma sodium regulation of chum salmon fry during transition into seawater. *Aquaculture* 28:133-142.

Janz, D. M., A. P. Farrell, J. D. Morgan, and G. A. Vigers. 1991. Acute physiological stress responses of juvenile coho salmon (*Oncorhynchus kisutch*) to sublethal concentrations of Garlon 4, Garlon 3A, and Vision herbicides. *Environ. Toxicol. Chem.* 10:81-90.

Jarvinen, A. W., B. R. Nordling, and M. E. Henry. 1983. Chronic toxicity of Dursban (chlorpyrifos) to the fathead minnow (*Pimephales promelas*) and the resultant acetylcholinesterase inhibition. *Ecotoxicol. Environ. Safety* 7:423-434.

Jobling, S., T. Reynolds, R. White, M. G. Porter, and J. P. Sumpter. 1995. A variety of environmentally persistent chemicals, including some phthalate plasticizers are weakly estrogenic. *Environ. Health Perspect.* 103:582-588.

Jobling, S. and J. P. Sumpter. 1993. Detergent components in sewage effluent are weakly estrogenic to fish: an *in vitro* study using rainbow trout (*Oncorhynchus mykiss*) hepatocytes. *Aquat. Toxicol.* 27:361-372.

Kelce, W. R., E. Monosson, M. P. Gamsik, S. C. Laws, and L. E. Gray. 1994. Environmental hormone disruptors: evidence that vinclozolin developmental toxicity is mediated by antiandrogenic metabolites. *Toxicol. Appl. Pharmacol.* 126:276-285.

Kelce, W. R., C. R. Stone, S. C. Laws, L. E. Gray, J. A. Kemppainen, and E. M. Wilson. 1995. Persistent DDT metabolite p,p'-DDE is a potent androgen receptor antagonist. *Nature* 375:581-585.

Kettle, W. D., F. deNoyelles, Jr., B. D. Heacock, and A. M. Kadoum. 1987. Diet and reproductive success of bluegill recovered from experimental ponds treated with atrazine. *Bull. Environ. Contam. Toxicol.* 38:47-52.

Khan, I. A. and P. Thomas. 1992. Stimulatory effects of serotonin on maturational gonadotropin release in the Atlantic croaker, *Micropogonias undulatus*. *Gen. Comp. Endocrinol.* 88:388-396.

Khan, I. A. and P. Thomas. 1996. Disruption of neuroendocrine function in Atlantic croaker exposed to Arochlor 1254. *Mar. Environ. Res.* 42:145-149.

Kingsbury, P. D. and D. P. Kreutzweiser. 1987. Permethrin treatments in Canadian forests. Part 1: Impact on stream fish. *Pesticide Sci.* 19:35-48.

Koenig, C. C. 1977. The effects of DDT and Mirex alone and in combination on the reproduction of a salt marsh Cyprinodont fish, *Adinia xenica.* pp. 357-376. IN: F. J. Vernberg, A. Calabrese, F. P. Thurberg, and W. B. Vernberg, eds. *Physiological Responses of Marine Biota to Pollutants*. Academic Press, New York.

Kreutzweiser, D. P. and P. D. Kingsbury. 1987. Permethrin treatments in Canadian forests. Part 2: Impact on stream invertebrates. *Pesticide Sci.* 19:49-60.

Kruzynski, G. M. 1972. Some effects of dietary methoxychlor on brook trout (*Salvelinus fontinalis*). M. Sc. thesis, Sir George Williams (Concordia) University, Montreal, Quebec. Reported in Kruzinski and Birtwell (1994) *Can. J. Fish. Aquat. Sci.* 51:1780-1790

Kruzynski, G. M. and I. K. Birtwell. 1994. A predation bioassay to quantify the ecological significance of sublethal responses of juvenile chinook salmon (*Oncorhynchus tshawytscha*) to the antisapstain fungicide TCMTB. *Can. J. Fish. Aquat. Sci.* 51:1780-1790.

Lampert, W., W. Fleckner, E. Pott, U. Schober, and K. U. Storkel. 1989. Herbicide effects on planktonic systems of different complexity. *Hydrobiol.* 188/189:415-424.

Larson, S. J., P. D. Capel, and M. S. Majewski. 1997. *Pesticides in Surface Waters. Distribution, Trends, and Governing Factors.* Ann Arbor Press, Chelsea, MI.

Little, E. E., R. D. Archeski, B. A. Flerov, and V. I. Kozlovskaya. 1990. Behavioral indicators of sublethal toxicity in rainbow trout. *Arch. Environ. Contam. Toxicol.* 19:380-385.

Lorz, H. W., S. W. Glenn, R. H. Williams, C. M. Kunkel, L. A. Norris, and B. R. Loper. 1979. Effects of selected herbicides on smolting of coho salmon. U. S. Environmental Protection Agency, Environmental Research Laboratory, EPA 600/3-79-071. Corvallis, OR.

Mac, M. J. and C. C. Edsall. 1991. Environmental contaminants and the reproductive success of lake trout in the Great Lakes: An epidemiological approach. *J. Toxicol. Environ. Health* 33:375-394.

Macek, K. J., K. S. Buxton, S. Sauter, S. Gnilka, and J. W. Dean. 1976. Chronic toxicity of atrazine to selected aquatic invertebrates and fishes. USEPA Ecological Res. Serv. EPA-600/3-76-047, Duluth, MN. 50 pp.

Macek, K. J. and S. Korn. 1970. Significance of the food chain in DDT accumulation by fish. *J. Fish. Res. Bd. Can*. 27:1496-1498.

Mahnken, C., E. Prentice, W. Waknitz, G. Monan, C. Sims, and J. Williams. 1982. The application of recent smoltification research to public hatchery releases: An assessment of size/time requirements for Columbia River hatchery coho salmon (*Oncorhynchus kisutch*). *Aquaculture* 28:251-268.

Maule, A. G. and C. B. Schreck. 1990. Changes in numbers of leukocytes in immune organs of juvenile coho salmon (*Oncorhynchus kisutch*) after acute stress or cortisol treatment. *J. Aquat. Anim. Health* 2:298-

304.

Maule, A. G., C. B. Schreck, and S. L. Kaattari. 1987. Changes in the immune system of coho salmon (*Oncorhychus kisutch*) during the parr-smolt transformation and after implantation of cortisol. *Can. J. Fish. Aquat. Sci.* 44:161-166.

McBride, J. R., H. M. Dye, and E. M. Donaldson. 1981. Stress response of juvenile sockeye salmon (*Oncorhynchus nerka*) to the butoxyethanol ester of 2,4-dichlorophenoxyacetic acid. *Bull. Environ. Contam.* 27:877-884.

McLachlan, J. A. 1993. Functional toxicology: a new approach to detect biologically active xenobiotics. *Environ. Health Perspect.* 101:386-387.

McLeese, D. W., C. D. Metcalf, and V. Zitko. 1980. Lethality of permethrin, cypermethrin and fenvalerate to salmon, lobster and shrimp. *Bull. Environ. Contam. Toxicol.* 25:950-955.

Mehrle, P. M. et al. 1987. Toxicity and bioconcentration of 2,3,7,8-tetrachloro-dibenzo-p-dioxin and 2,3,7,8-tetrachloro-dibenzofuran in rainbow trout. *Environ. Toxicol. Chem.* 27:47-62.

Menzel, D. W., J. Anderson, and A. Randtke. 1970. Marine phytoplankton vary in their response to chlorinated hydrocarbons. *Science* 167:1724-1726.

Metcalf, R. L., G. K. Sangha, and I. P. Kapoor. 1971. Model ecosystem for the evaluation of pesticide biodegradability and ecological magnification. *Environ. Sci. Tech.* 5:709-716.

Miller, D. L. and D. M. Ogilvie. 1975. Temperature selection in brook trout (*Salvelinus fontinalis)* following exposure to DDT, PCB, or phenol. *Bull. Environ. Contam. Toxicol.* 14:545-551.

Mitchell, D. G., P. M. Chapman, and T. J. Long. 1987. Seawater challenge testing of coho salmon smolts following exposure to Roundup herbicide. *Environ. Toxicol. Chem.* 6:875-878.

Moccia, R. D., J. F. Leatherland, and R. A. Sonstegard. 1977. Increasing frequency of thyroid goiters in coho salmon (*Oncorhynchus kisutch*) in the Great Lakes. *Science* 198:425-426.

Moccia, R. D., J. F. Leatherland, and R. A. Sonstegard. 1981. Quantitative interlake comparison of thyroid pathology in Great Lakes coho (*Oncorhynchus kisutch*) and chinook (*Oncorhynchus tshawytscha*) salmon. *Cancer Res.* 41:2200-2210.

Moller, H. 1985. A critical review on the role of pollution as a cause of fish diseases. pp. 169-182. IN: A. E. Ellis, ed. *Fish and Shellfish Pathology.* Academic Press, New York.

Monosson, E., W. R. Kelce, M. Mac, and L. E. Gray. 1997. Environmental antiandrogens: potential effects on fish reproduction and development. pp. 53-60. IN: Rolland, R. M., M. Gilbertson, and R. E. Peterson, eds. *Chemically Induced Alterations in Functional Development and Reproduction of Fishes.* SETAC Press, Pensacola, FL

Morgan, M. J., and J. W. Kiceniuk. 1992. Response of rainbow trout to a two month exposure to Vision, a glyphosate herbicide. *Bull. Environ. Contam. Toxicol.* 48:722-780.

Morgenstern, H. and D. Thomas. 1993. Principles of study design in environmental epidemiology. *Environ. Health Perspect.* 101 (Suppl. 4):23-38.

Muirhead-Thomson, R. C. 1987. *Pesticide Impact on Stream Fauna with Special Reference to Macroinvertebrates.* Cambridge University Press, Cambridge, UK.

Murty, A. S. 1986. *Toxicity of Pesticides to Fish.* Volumes 1 and 2. CRC Press, Inc., Boca Raton, FL.

Naqvi, S. M. and R. H. Hawkins. 1989. Responses and LC50 values for selected microcrustaceans exposed to Spartan, malathion, Sonar, Weedtrine-D, and Oust pesticides. *Bull. Environ. Contam. Toxicol.* 43:386-393.

National Research Council. 1993. Soil and Water Quality: An Agenda for Agriculture. Washington, DC: National Academy Press.

Nikl, D. L. and A. P. Farrell. 1993. Reduced swimming performance and gill structural changes in juvenile salmonids exposed to 2-(thiocyanomethylthio) benzothiazole. *Aquat. Toxicol.* 27:245-264.

Norris, L. A., H. W. Lorz, and S. V. Gregory. 1991. Forest Chemicals. pp. 207-296. IN: W. R. Meehan, ed. Influences of Forest and Rangeland Management on Salmonid Fishes and Their Habitats. *Amer. Fish. Soc. Sp. Publ*. 19, Bethesda, MD.

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

Ogilvie, D. M. and D. L. Miller. 1976. Duration of a DDT-induced shift in the selected temperature of Atlantic salmon (*Salmo salar*). *Bull. Environ. Contam. Toxicol.* 16:96-99.

O'Halloran, K., J. T. Ahokas, and P. F. A. Wright. 1998. Response of fish immune cells to *in vitro* organotin exposures. *Aquat. Toxicol.* 40:141-156.

Olla, B. L., M. W. Davis, and C. B. Schreck. 1992. Comparison of predator avoidance capabilities with corticosteroid levels induced by stress in juvenile coho salmon. *Trans. Am. Fish. Soc.* 121:544-547.

Olla, B. L., M. W. Davis, and C. B. Schreck. 1995. Stress-induced impairment of predator evasion and non-predator mortality in Pacific salmon. *Aquacult. Res*. 26:393-398.

Parker, R. R. 1971. Size selective predation among juvenile salmonid fishes in a British Columbia inlet. *J. Fish. Res. Bd. Can.* 28:1503-1510.

Pasmanik, M and G. Callard. 1985. Identification and characterization of an androgen receptor in the brain of goldfish (*Carassius auratus*). Am. Zool. 25:115A.

Paul, E. A. and H. A. Simonin. 1996. Effects of naled, synergised and non-synergised resmethrin on the swimming performance of young trout. *Bull. Environ. Contam. Toxicol.* 57:495-502.

Pearcy, W. G. 1992. *Ocean Ecology of North Pacific Salmonids*. Washington Sea Grant Program. Seattle: University of Washington Press.

Peterson, R. H. 1973. Temperature selection of Atlantic salmon (*Salmo salar*) and brook trout (*Salvelinus fontinalis*) as influenced by various chlorinated hydrocarbons. *J. Fish. Res. Bd. Can.* 30:1091-1096.

Pickering, A. D. 1993. Endocrine-induced pathology in stressed salmonid fish. *Fish. Res*. 17:35-50.

Pickering, A. D. and T. G. Pottinger. 1989. Stress responses and disease resistance in salmonid fish: Effects of chronic elevation of plasma cortisol. *Fish Physiol. Biochem.* 7:253-258.

Piferrer, F., S. Zanuy, M. Carrillo, I. I. Solar, R. H. Devlin, and E. M. Donaldson. 1994. Brief treatment with an aromatase inhibitor during sex differentiation causes chromosomally female salmon to develop as normal, functional males. *J. Exp. Zool.* 270:255-262.

Plumb, J. A. and N. Areechon. 1990. Effect of malathion on humoral immune response of channel catfish. *Develop. Comp. Immunol*. 14:355-358.

Racka, K. D. 1993. Environmental fate of chlorpyrifos. *Rev. Environ. Contam. Toxicol.* 13:1-151.

Repetto, R. and S. S. Baliga. 1996. *Pesticides and the immune system: The public health risks.* World Resources Institute, Washington, D. C.

Risebrough, R. W., D. B. Menzel, D. J. Marsten, Jr., and H. S. Olcott. 1967. DDT residues in Pacific sea birds: A persistent insecticide in marine food chains. *Nature* 216:589-591.

Rolland, R. M., M. Gilbertson, and R. E. Peterson, eds. 1997. *Chemically Induced Alterations in Functional Development and Reproduction of Fishes.* SETAC Press, Pensacola, FL.

Sinha, N., B. Lal, and T. P. Singh. 1991. Carbaryl-induced thyroid disfunction in the freshwater catfish, *Clarias batrachus*. *Ecotoxicol. Environ. Safety* 21:240-247.

Sinha, N., B. Lal, and T. P. Singh. 1992. Thyroid physiology impairment by malathion in the freshwater catfish, *Clarias batrachus*. *Ecotoxicol. Environ. Safety* 24:17-25.

Smith, L. S., R. D. Cardwell, A. J. Mearns, T. W. Newcomb, and K. W. Watters. 1972. Physiological changes experienced by Pacific salmon migrating through a polluted urban estuary. pp. 322-325. IN: M. Ruivo, ed. *Marine Pollution and Sea Life,* Fishing News (Books) Ltd, London.

Smith, T. M. and G. W. Stratton. 1986. Effects of synthetic pyrethroid insecticides on nontarget organisms. *Residue Reviews* 97:93-120.

Somoza, G. M., K. L. Yu, and R. E. Peter. 1988. Serotonin stimulates gonadotropin release in female and male goldfish, *Carassius auratus* L. *Gen. Comp. Endocrinol.* 72:374-382.

Soto, A. M., K. L. Chung, and C. Sonnenschein. 1994. The pesticides endosulfan, toxaphene, and dieldrin have estrogenic effects on human estrogen-sensitive cells. *Environ. Health Perspect.* 102:380-383.

Specker, J. L. and C. B. Schreck. 1982. Changes in plasma corticosteroids during smoltification of coho salmon, *Oncorhynchus kisutch*. *Gen. Comp. Endocrinol.* 46:53-59.

Spitzbergen, J. M., K. A. Schat, J. Kleeman, and R. E. Peterson. 1986. Interaction of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) with immune responses of rainbow trout. *Vet. Immunol. Immunopath.* 12:263-280.

Spitzbergen, J. M., K. A. Schat, J. Kleeman, and R. E. Peterson. 1988. Effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) or Arochlor 1254 on the resistance of rainbow trout, *Salmo gairdneri* Richardson, to infectious hematopoetic necrosis virus. *J. Fish Dis.* 11:73-83.

Stadnyk, L., R. S. Campbell, and B. T. Johnson. 1971. Pesticide effect on growth and ^{14}C assimilation in a freshwater algae. *Bull. Environ. Contam. Toxicol.* 6:1-8.

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

Tatner, M. F. 1996. Natural changes in the immune system of fish. IN: Iwama, G. and T. Nakanishi, eds. *The Fish Immune System. Organism, Pathogen, and Environment.* Academic Press, New York.

Thomas, P. and I. A. Khan. 1997. Mechanisms of

chemical interference with reproductive endocrine function in sciaenid fishes. pp 29-51. IN: Rolland, R. M., M. Gilbertson, and R. E. Peterson, eds. *Chemically Induced Alterations in Functional Development and Reproduction of Fishes.* SETAC Press, Pensacola, FL.

Thorpe, J. E., M. G. McConway, M. S. Miles, and J. S. Muir. 1987. Diel and seasonal changes in resting plasma cortisol levels in juvenile Atlantic salmon, *Salmo salar* L. *Gen. Comp. Endocrinol*. 65:19-22.

Torstensson, N. T. L., L. N. Lundgren, and J. Stenstrom. 1989. Influence of climate and edaphic factors on persistence of glyphosate and 2,4-D in forest soils. *Ecotoxicol. Environ. Safety* 18:230-239.

Veith, G. D., D. L. Defoe, and B. V. Bergstedt. 1979. Measuring and estimating the bioconcentration factor of chemicals in fish. *J. Fish. Res. Bd. Can.* 36:1040-1049.

Wagner, R. J., J. C. Ebbert, and L. M. Roberts. 1996. Are agricultural pesticides in surface waters of the central Columbia plateau? U. S. Geological Survey Fact Sheet 241-95, U. S. Geological Survey, Tacoma, WA.

Walker, C. R. 1964. Simazine and other s-triazine compounds as aquatic herbicides in fish habitats. *Weeds* 12:134-139.

Washino, R. K., W. Ahmed, J. D. Linn, and K. G. Whitesell. 1972. Effects of low volume Dursban sprays upon aquatic non-target organisms. *Mosq. News* 32:531-537.

Wedemeyer, G. A., R. L. Saunders, and W. C. Clarke. 1980. Environmental factors affecting smoltification and early marine survival of anadromous salmonids. *Mar. Fish. Rev.* 42:1-15.

Weis, J. S. and P. Weis. 1976. Optical malformation induced by insecticides in embryos of the Atlantic silverside *Menidia menidia*. *Fish. Bull.* 74:208-214.

Weis, P. and J. S. Weis. 1974. Sevin and schooling behavior of *Menidia menidia* in the presence of the insecticide Sevin (carbaryl). *Mar. Biol.* 28:261.

Weis, P. and J. S. Weis. 1976. Abnormal locomotion associated with skeletal malformations in the sheepshead minnow, *Cyprinodon variegatus*, exposed to malathion. Environ. Res. 12:196-200.

Wells, D. E. and A. A. Cowan. 1982. Vertebral dysplasia in salmonids caused by the herbicide trifluralin. *Environ. Pollut.* 29:249-260.

Wentz, D. A., B. A. Bonn, K. D. Carpenter, S. R. Hinkle, M. L. Janet, F. A. Rinella, M. A. Ulrich, I. R. Waite, A. Laenen, and K. E. Bencala. 1998. Water quality in the Willamette Basin, Oregon, 1991-1995. U. S. Geological Survey Circular 1161, Denver, CO.

White, R., S. Jobling, S. A. Hoare, J. P. Sumpter, and M. G. Parker. 1994. Environmentally persistent alkylphenolic compounds are estrogenic. *Endocrinol.* 135:171-181.

Williamson, A. K., M. D. Munn, S. J. Ryker, R. J. Wagner, J. C. Ebbert, and A. M. Vanderpool. 1998. Water quality in the central Columbia plateau, Washington and Idaho, 1992-1995. U. S. Geological Survey Circular 1144, U. S. Geological Survey, Tacoma, WA. 35 pp.

Wong, C. I., W. R. Kelce, and E. M. Wilson. 1995. Androgen receptor antagonist verses agonist activities of the fungicide vinclozolin relative to hydroxyflutamide. *J. Biol. Chem.* 270:19998-20003.

Woodwell, G. M., C. F. Wurster, Jr., and P. A. Isaacson. 1967. DDT residues in an East Coast estuary: A case of biological concentration of a persistent insecticide. *Science* 156:821-824.

World Health Organization. 1994. *Glyphosate*. Environmental Health Criteria #159. United Nations Environment Programme, Geneva, Switzerland.

World Health Organization. International Programme on Chemical Safety 1996. *Chlorothanlonil.* Environmental Health Criteria 183. Geneva, Switzerland. p. 53.

Glossary, Abbreviations and Scientific Names

2,4-D — 2,4-dichlorophenoxyacetic acid. A herbicide that mimics natural plant hormones.

DDT — dichlorodiphenyltrichloroethane. An insecticide widely used in the past but now illegal in the United States.

DDE — dichlorodiphenylethane. A long-lived breakdown product of DDT.

Eulachon — *Thaleichthys pacificus*

Euryhaline — the ability to tolerate wide ranges of salinity. Euryhaline fish are often found in estuaries where the salinities can range from fresh water to seawater.

Gonopodium — front rays of the anal fin of livebearing fish (Family Poeciliidae) are elongated to form an organ that assists in internal fertilization.

IHN virus — infectious hematopoetic necrosis virus, a major cause of mortalities in Columbia basin salmonids

Lamprey, Pacific — *Lampetra tridentata*

Minnow, fathead — *Pimophales promelas*

Mosquitofish — *Gambusia* sp.

Parr — a juvenile salmonid characterized by vertical bars, or parr marks, along its sides. Fish at this stage are brownish in color and hold territories in the stream.

Ppb — parts per billion; a concentration of pesticide equal to one gram of the compound dissolved in a billion grams of water. Equivalent to micrograms per liter (µg/L).

Ppm — parts per million; a concentration of pesticide equal to one gram of the compound dissolved in a million grams of water. Equivalent to milligrams per liter (mg/L).

Pesticide — anthropogenic chemicals used for control of target organisms. These include insecticides, herbicides, fungicides, and other biocides.

Redd — a depression dug in the streambed gravel in which salmonids lay their eggs.

Salmon, Atlantic — *Salmo salar*

Salmon, chinook — *Oncorhynchus tshawytscha*

Salmon, coho — *Oncorhynchus kisutch*

Salmon, sockeye — *Oncorhynchus nerka*

Shad, American — *Alosa sapidissima*

Smolt — a juvenile salmonid characterized by a uniform silvery color. Fish at this stage undergo a number of physiological changes, lose their ability to hold territories, tend to school, and begin migration toward the sea.

Steelhead — *Oncorhynchus mykiss*, a migratory form of rainbow trout

Surfactant — a chemical added to formulations of pesticides to reduce surface tension and cause wetting by the pesticide solution.

TCDD — 2,3,7,8-tetrachlorodibenzo-dioxin

TCMTB — 2-(thiocyanomethythio)-benzothiazole, an antisapstain fungicide used on timber to be exported.

Trout, brook — *Salvelinus fontinalis*

Trout, brown — *Salmo trutta*

Trout, cutthroat — *Oncorhynchus clarki*

Trout, lake — *Salvelinus naymaycush*

Trout, rainbow — *Oncorhynchus mykiss*

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