

Lecture 19: Chemical inputs and effects on fishes

I. Introduction

A. Objectives

1. Brief overview of sources, pathways of chemical inputs
2. Summarize pathways, effects of chemicals inside fishes
3. Summarize key aspects of fish toxicology
4. Summarize major sources, impacts of anthropogenic chemicals
5. Illustrate how relations between chemistry and fishes are used in management contexts

Chemical = (usually) inorganic substances that can affect biological reactions, survival, growth, reproduction

II. Sources and pathways

Chemical inputs come from many sources, natural and anthropogenic

Chemicals enter fish-supporting waters via precipitation, surface runoff, groundwater

Aquatic ecosystems function as landscape sinks

A. Naturally-occurring toxins

1. Plant products

- juglone, found in walnut husks, is toxic to other trees and to aquatic organisms
- in low-ANC, surface waters may be tea-colored from tannic/humic acids dissolved from plant material
- $C_{23}H_{22}O_6$ – white crystalline substance from certain plant roots

2. Leachates from rock and soil

- acids, bases, metals can leach from soil, especially when newly exposed
- underlying rock-type strongly affects chemistry of groundwater, surface water
- USEPA ecoregions represent geographic variation in physicochemical conditions in surface waters

3. Byproducts of metabolism

- fish must deal with the toxic byproducts of metabolism that they themselves produce (eg ammonia)
- density-dependent control?

B. Natural toxins increased by human actions

1. Increased rate at which naturally-occurring toxins enter aquatic systems

- metals (eg mercury, zinc, copper) from manufacturing and mining
- acids (eg, sulfuric and nitric) from fossil-fuel combustion

2. Case study on mercury – most common, fastest growing fish toxin in US

3. US health advisories on eating fish – loss of a valued ecosystem service

- a. extent: 1/7 of river miles, 1/4 of lake acres, 3/4 of estuary acres
- b. species most likely tainted: large, old piscivores, benthivores

C. Synthetic chemicals

- fish often lack evolutionary response to exposure
- pesticides (eg, DDT, malathion)
- by-products of industry, manufacturing (eg PCBs, organics, petrochemicals)

III. Chemical entry into, exit from individual fish

A. General absorption pathways: water or food

- water pathways: chemicals dissolved or suspended in water → gills or gut

1. Diffusion across cell membranes

- a. water and many solutes can pass through semi-permeable lipid cell membranes
 - passively via osmosis
 - actively via active transport

2. Fat-soluble substances can dissolve directly into lipid membrane

B. Storage and transport

1. Chemical may be stored in liquid or fatty deposits or transported through the body
 - fate depends on body chemistry, size or solubility of the chemical
2. Fat-soluble organics (eg, PCB, DDT, kepone) can accumulate in fat deposits (eg, salmon, carp, catfish)
 - during low feeding or gonadal development, fat-stored toxics can be metabolized
 - potential problem for migratory, semelparous fishes
3. Metals often accumulate in kidneys; many toxics accumulates in liver
4. Bioaccumulation = slow accumulation, storage → in-body concentrations 10-50X a lethal acute dose
 - a. bioaccumulation at multiple levels of food chain → “biomagnification”
 - PCBs in Lake Michigan increased 50,000,000X between phytoplankton and salmon
 - although only trace amounts occur in water, the fish we (and wildlife) eat can be toxic

C. Cellular mechanisms

1. Receptor = part of the cell that enables the action of the toxin
 - a toxin is “toxic” only if it binds with its receptor
 - eg, malathion binds with cholinesterase at neuron junctions
 - eg, cyanide interferes with oxidative phosphorylation

D. Excretion

Fish’s best defense is to get rid of toxins

1. Fish can directly excrete many chemicals through lipid membranes in the gills
 - fish kidneys not important for excreting metabolic waste
2. Fish also (to limited extent) “biotransform” toxins by coupling them to carbohydrate or amino acid
 - typically detoxifies toxin → water-soluble, easier to excrete

IV. Factors affecting toxicity and toxin detection

- variation among life stages, individuals

A. Taxonomy

- wide variation in sensitivity to a given toxin among taxa
- wide variation in ranks of toxicity of a given set of toxins among taxa (no most-sensitive fish)

B. State of the individual

1. Size or age
 - a. smaller/younger individuals have higher metabolism, surface:volume ratio
 - can be exposed to more toxicants over a shorter time period
 - very difficult to document/track effects of toxins in wild
 - b. smaller fish have less plasma to dilute toxins
 - c. larger/older individuals have had more time to accumulate toxins
 - may eat higher on the food chain
2. Reproductive condition
 - toxic effects usually greater for reproductively active fish, especially females
3. Environmental history
 - prior exposure (frequency, duration, magnitude) to toxins
 - short-term, long-term responses to exposure

- a. acclimation = short-term physiological adjustment to environment
 - allows fish to tolerate greater range of conditions
 - eg, fish acclimated at pH=6 for a while can live longer at pH=4 than can fish acclimated at pH=8
 - longer acclimation period may increase fish's ability to tolerate more toxin
- b. genetic makeup
 - individuals in a population vary in genotype, including sensitivity to toxins (tolerance can evolve)
 - exposure to toxins can occur too quickly, severely for evolution (requires gene-based survival)
 - evolving resistance to a toxin may have evolutionary costs for other adaptations (always tradeoffs)

C. Chemical interactions

1. Additive effects
 - cumulative effect of multiple toxins = effect of multiple doses of single toxin
2. Antagonistic effects
 - conditions that decrease effect of one toxin increase effect of another
3. Synergistic effects
 - toxins intensify each others' effects

V. Effects of chemicals on fish

A. Acute exposures

1. Short-term, high-concentration exposure
 - often highly publicized, visible fish kills
2. Tragic example on Clinch River
 - rupture of dike at AEP waste-settling pond near Carbo, VA,
 - released >100 million gallons of water (pH = 12-13) into Dumps Creek near confluence with the Clinch
 - severe kill of aquatic life for 126 km
 - affected 55-60 species of fish, including 5-6 extirpations
 - most other species have slowly recovered

B. Chronic exposures

1. Long-term, low-concentration exposure (often sub-lethal)
 - may pose greater threat to fish populations than occasional fish kills
2. Effects on reproduction, growth, physiology
 - a. fat-soluble chemicals can be transferred to eggs
 - lake trout fry suffer heavy mortality from DDT as yolk absorption completes
 - b. kepone and mirex reduced collagen metabolism → scoliosis, vertebral damage in fathead minnows
 - c. impaired healing
 - acetyl-cholinesterase inhibitors (malathion, parathion, sevin) slowed fin regeneration in killifish
 - d. 1 ppm detergent severely impaired taste receptors in bullhead catfishes
3. Effects on behavior
 - a. convict cichlids exposed to low (5-5.5) pH alter breeding behaviors
 - females less interested in brooding
 - males increase brooding behavior, aggression toward other males
 - b. mummichogs exposed to mercury, cadmium, other metals
 - more sluggish, less capable of capturing guppy prey
 - grew slower than mummichogs from control areas
 - larvae more disoriented, collided with each other more frequently