

Rotenone Characterization and Toxicity in Aquatic Systems

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Abstract

Rotenone is a compound that has been used by fisheries managers since the 1930's as a piscicide, or fish poison. It can have effects on various non-target species as well, like benthos and zooplankton. Rotenone is an isoflavonoid and is a naturally occurring rotenoid plant extract found in many species of the pea (Leguminosae) family in South America, Australia, and Southern Asia. It can be applied to lotic water bodies by drip stations or sprayers, and to lentic water bodies by powerboat propeller wash or aircraft. Its application concentration varies from 0.5 – 5mg/l, dependent on the fish species targeted. Rotenone is generally unstable and degrades rapidly in water with the presence of light, heat, turbidity, shallow depths, and low organics. Degradation of rotenone results in at least 20 degradation products, of which only one is toxic: 6 α , 12 α β -rotenolone. Rotenone acts to increase blood oxygen content. Fish death is most likely a result of rotenone inhibiting mitochondrial NADH-ubiquinone reductase, which makes oxygen unavailable for respiration. The high susceptibility of fish to rotenone stems from rotenone's efficient entry through the gills. Using potassium permanganate, chlorine, activated carbon, methylene blue, and fresh, oxygenated water can reverse the toxic action of rotenone.

Introduction

For many years, introduced fish species have created problems with native aquatic systems. The desirable native fish stocks often suffer, even to the point of extirpation, because of the unwanted species' generalist tendencies and competitiveness. Many methods for removing these unwanted species, from netting, to shocking, to outright drainage of a water body, have resulted in limited success. The use of fish poisons, on the other hand, is one method that has shown some promise in ridding important aquatic habitat of introduced species. Of the many poisons used, rotenone is by far the most popular (Bradbury 1986).

Since the first extensive application of rotenone for fish management in the 1930's, it became widely used nationwide by fisheries biologists. As seen all too often in the early to mid 20th century, chemicals were applied without much consideration into their potential damaging impacts and repercussions to other life (Clemens and Martin 1953). The first scientist to publish works concerning the potential adverse effects of rotenone was Dr. Carl Hubbs (1963), who is also given credit for the first applied treatment of the chemical to fish populations in the United States. Since that time, many studies have been published regarding rotenone's impact to other aquatic life, including macroinvertebrates (Mangum and Madrigal 1999; Bradbury 1986), zooplankton (Beal and Anderson 1993; Bradbury 1986), and phytoplankton (Bradbury 1986; Bonn and Holbert 1961). In addition to studies of the impacts of rotenone to aquatic systems, studies have been published concerning its effects on humans and other mammals (Bradbury 1986; Schnick 1974).

Rotenone has been used to meet many different objectives set forth by managers of aquatic systems. It has been used to improve migratory waterfowl habitat by removing unwanted carp that are responsible for uprooting much of the critical vegetation used by ducks and geese (Weier and Starr 1950). Rotenone has been used to sample fish populations in ponds (Brunson 1999), lakes (Krumholz 1950), streams (Boccardy and Cooper 1963), and rivers (Binns et al. 1967) for use as survey and management tools. Rotenone has even been used to rid municipal water supplies of turbidity and algae caused by bottom feeding fish (Bonn and Holbert 1961).

Objectives

The purpose of this report is to characterize the effects of rotenone use on aquatic life based on a review of the scientific literature. The report will focus on the sources, pathways, receptors, and controls of rotenone in aquatic systems. Specifically, the report will analyze rotenone sources, fate and transport in aquatic systems, toxicological endpoints in aquatic organisms, and controls that aid in mitigation of exposure.

Discussion

Sources

Rotenone, with an empirical formula of $C_{23}H_{22}O_6$ (figure 1), is an isoflavonoid compound with a molecular weight of 394.41 (Schnick 1974). It consists of 70.04% carbon, 5.62% hydrogen, and 24.34% oxygen. It melts at 165–166 °C. Rotenone is

very soluble in a number of organic solvents like alcohol and acetone, but is practically insoluble in water.

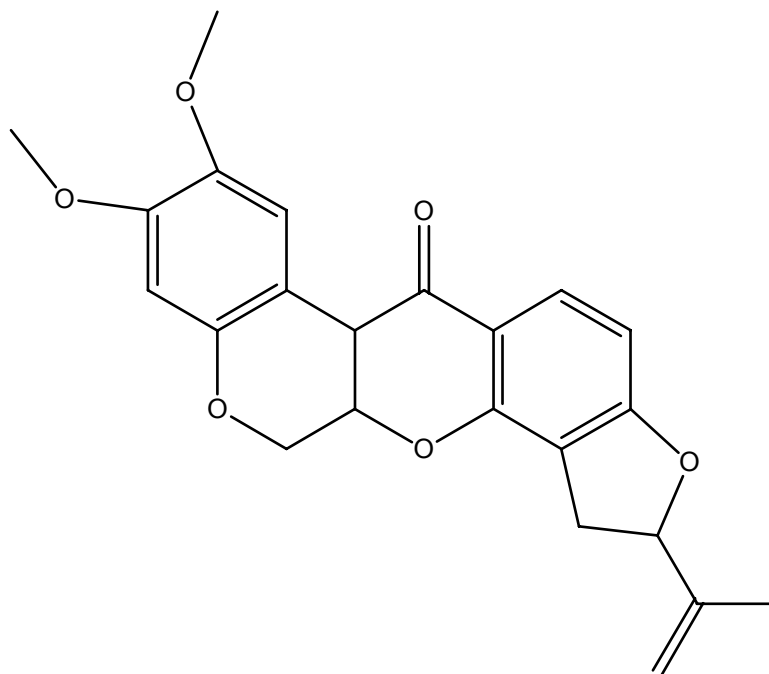


Figure 1. The chemical structure of rotenone

Rotenone is a naturally occurring rotenoid plant extract found in many species of the pea (Leguminosae) family (Brunson 1999). Most of these plants are native to South America and Australia, as well as to many countries in Southern Asia. Commercially available rotenone as a piscicide comes in three basic forms: 5% emulsifiable concentrate, 5% wettable powder, and 2.5% synergized emulsifiable concentrate (Schnick 1974). Pure crystalline rotenone for laboratory purposes can be extracted from plant resins with solvents like chloroform and benzene (Bradbury 1986).

Rotenone was first used in powder form to manage fish populations (Schnick 1974). This powdered form was irritating to the nose and throat and relatively inefficient

to disperse. A liquid emulsifiable concentrate that was easier to apply and mix, had better penetration, and was more effective in colder water temperatures (Krumholz 1950) soon replaced it. Then it was found that adding a synergist, like Sesamex (Bradbury 1986), to the liquid emulsion increased its toxicity and made it more cost effective. One setback of this synergized emulsion is that it can be foul smelling due to the solvents and carriers used (Schnick 1974). It wasn't until the 1950's that formulations were guaranteed to contain viable amounts of rotenone. Before this, it was difficult to get consistent results of many rotenone formulations.

Pathways

Rotenone can be applied to surface water in a number of ways. For lotic water bodies it is typical to use drip stations or sprayers (Brunson 1999). This will allow the moving water to carry the rotenone downstream. In lentic water bodies, rotenone can be pumped through a hose into the propeller wash of a powerboat. This allows for the systematic distribution of a known concentration to be spread through the entire lake, reservoir, or pond. In rare cases, where water bodies are remotely located and difficult to access, aircraft can be used to spray liquid rotenone (Schnick 1974). The most important consideration in applying rotenone is to keep the toxin evenly distributed, to prevent pockets of safe areas where the target organism might escape a lethal dose (Brunson 1999).

Rotenone is most commonly applied in 0.5 - 1 mg/l concentrations (Schnick 1974). Some fish are more resistant to rotenone than others and will require a larger

concentration, sometimes up to 5 mg/l. Resistant fish include bullheads, goldfish, and bowfin. Under normal conditions, rotenone treatments will kill fish within 24-36 hours.

Rotenone is generally unstable and degrades rapidly in water. It has been shown to degrade as fast as within 2 weeks of application (Schnick 1974) but can also persist for periods up to 5 months (Smith 1941, Leonard 1939). The length of degradation time is dependent on many factors including light, temperature, turbidity, depth, presence of organic debris, and dose (Bradbury 1986). Despite all the factors that go into rotenone degradation, Schnick (1974) claims that waters should still detoxify within 5 weeks of treatment.

Rotenone is photochemically unstable and will readily breakdown in the presence of light. Light will oxidatively decompose rotenone into nontoxic dihydrorotenone and water (Schnick 1974). This degradation process will occur at a quicker rate in the presence of higher water temperatures. Temperature appears to affect the breakdown of rotenone the most. Dawson et al. (1991) concluded that higher temperature waters would readily degrade rotenone faster than lower temperature waters. It is for this reason that many fish managers choose to apply rotenone in the warmer summer months.

Turbidity and organic debris in water will act to slow down the decay of rotenone. Its been shown that rotenone will adsorb to the sediment and organic particles and persist for longer periods of time (Dawson et al. 1991). High turbidity also corresponds to lower light penetration into the water, which will allow rotenone to be degraded at a slower rate.

Depth of water also plays a role in the breakdown of rotenone. Rotenone tends to breakdown more readily in the shallow epilimnion of water bodies (Bradbury 1986). Schnick (1974) reported that each increase in depth of 1 foot in a pond increased length of rotenone toxicity to bluegill by 2 days. Not only is the epilimnion usually warmer than the deeper hypolimnetic waters but it also gets more light than the hypolimnion. These two factors act to increase the rate at which rotenone will degrade in such waters.

As mentioned earlier, the early studies of rotenone degradation showed that rotenone broke down to two simple products: dehydrorotenone and water (Bradbury 1986). Further study by Cheng et al. (1972), using photodegradation, identified that rotenone decomposes to least 20 degradation products, most of which are rotenoids. They reported that only one product, 6 $\alpha\beta$, 12 $\alpha\beta$ -rotenolone, is toxic. The fact that the other 19 or more degradation products aren't toxic is one more reason why aquatic managers feel rotenone is justifiable for use on many of our nation's waters.

Receptors

Early rotenone fish poisonings of the 1930's reported that the death of the fish was caused by the constriction of the gill capillaries preventing blood flow to the tissue (Schnick 1974). Similar findings conclude that rotenone poisoning acts to destroy gill tissue. However, Oberg (1964) found that severe rotenone poisoned fish appeared to have normal gill circulation and that the destruction of gill tissue was apparently due to secondary changes appearing in the late stages of poisoning. Later research proved

the toxic action of rotenone actually takes place at the cellular level, where it blocks oxidative phosphorylation (Fukami et al. 1967). Oberg (1961) found that the specific site of action of rotenone is in the electron transport system. Fajt and Grizzle's 1998 study on carp made it clear that rotenone acts to increase blood oxygen content. They noted that fish death is most likely a result of rotenone inhibiting mitochondrial NADH-ubiquinone reductase, which makes oxygen unavailable for respiration. Further evidence of rotenone affecting fish respiration is exhibited by fish behavioral changes, including reduced opercular movement, erratic swimming, and loss of equilibrium. After this noted behavior, the ventilation rate of fish slows and they sink to the bottom where they remain until death (Oberg 1967).

Rotenone has the ability to inhibit cellular respiration in almost every living organism, including mammals, fish, amphibians, insects, and even plants (Bradbury 1986). So the next logical question is why rotenone is only toxic to certain organisms, mainly fish and insects. Fukami et al. (1969) concluded that the selective toxicity of fish, mammals, and insects to rotenone is due to the ease of entrance to the cellular level. The high susceptibility of fish to rotenone stems from rotenone's efficient entry through the gills (Oberg 1964; 1967). The gill-like tracheae, the cuticle, and the mid-gut of aquatic macroinvertebrates make them just as susceptible to the toxic effects of rotenone (Bradbury 1986). One advantage aquatic macroinvertebrates have is that they can often escape rotenone poisoning due to their ability to burrow into the sediments. The insolubility of rotenone in water and the relatively high lipid content of gills combine to create a gradient that essentially "pulls" rotenone in as it encounters these large surface area structures. Once in the bloodstream, rotenone associates with

lipids and lipoproteins, favoring its buildup in organ tissues. Mammals are not highly susceptible to rotenone because they are protected by effective oxidizing enzyme systems (Schnick 1974), and inefficient gastrointestinal absorption (Bradbury 1986).

Controls

Several environmental and regulatory measures aid in the mitigation of rotenone exposure. The Environmental Protection Agency (EPA) has registered twenty-nine formulations of rotenone from 18 different companies for aquatic and agriculture use (Schnick 1974). There are continual investigations in progress to update information on the rotenone formulations. These investigations include short and long-term effects of rotenone on invertebrates in the laboratory and in the field, toxicity tests on fish, counteraction, and factors influencing inactivation and degradation of rotenone.

The toxic action of rotenone can be reversed depending on the amount absorbed by the organism (Bradbury 1986). Fukami et al. (1969) concluded that natural detoxification can occur of sublethal rotenone doses. The basic process involves the oxidation of the compound by microsomal mixed-function oxidase (mfo) enzymes.

Many studies have shown that rotenone poisoned fish can be revived by one or a combination of treatments (Bradbury 1986). Solutions of potassium permanganate or chlorine can be used to detoxify rotenone (Schnick 1974). Rotenone can be removed from the water with activated carbon. Oberg (1967) suggests that simply using untreated freshwater baths can revive fish. He reports reviving rotenone-poisoned cod in untreated water. Oberg (1961) also showed that methylene blue solutions could reduce respiratory inhibition due to rotenone. The use of methylene blue is often

discouraged because it is toxic to aquatic macrophytes and encourages bacterial infection on fish (Bradbury 1986).

Potassium permanganate has been reported to be the most effective and practical method of rotenone detoxification (Bradbury 1986). It can be used to reverse the toxicity in the affected fish or to accelerate the natural breakdown in water. To reverse fish toxicity, the affected fish are placed in a potassium permanganate solution for about 20 seconds and then placed in fresh, non-toxic, hyperoxygenated water where they will have a higher chance of survival. It has been hypothesized that potassium permanganate works by neutralizing residual rotenone on the gills and body surface of the fish. One downfall of using this treatment is that it has a tendency to leave fish more vulnerable to bacterial and fungal infections. One way potassium permanganate has been used to detoxify water is in outlet streams that flow from treated lakes. This is crucial so that rotenone will not impact non-target fish species occupying the stream habitat.

Many rotenone treatments have been used to clean up municipal water supplies but have received criticism because they can cause water to have a bad odor and taste (Bonn and Holbert 1961). Tests have shown that using 1mg/l applications of activated carbon for each threshold odor number produced can control these undesirable effects.

Certain managerial decisions can also act to control harmful effects of rotenone. In North America, many fish managers choose to apply rotenone in the warm, summer months. As stated earlier, heat and light are the best at degrading rotenone, both of which the summer tends to favor.

Conclusions

The sources, pathways, receptors, and controls of rotenone have been studied in detail by many researchers. This report has attempted to highlight the most important findings of each category so that each individual who handles this compound can understand its toxicity and potential effects in detail. Careful and responsible application of this toxic compound will allow for its continued use. Managers need to be aware that rotenone is not only toxic to target organisms, like fish, but also potentially toxic to non-target organisms, including humans. Continued studies regarding rotenone use can ensure heightened awareness to managers responsible for application and bolster confidence in the public who rely on these managers for the safety of now and future generations.

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