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From:
Nancy A. Erman
Specialist Emeritus
Aquatic ecology/ freshwater invertebrates
Department of Wildlife, Fish, and Conservation Biology
University of California, Davis
e-mail: naerman@ucdavis.edu
43200 East Oakside Place
Davis, CA 95616
530/758-1206

and

Don C. Erman
Professor Emeritus
Aquatic ecology/ fisheries biology
Department of Wildlife, Fish, and Conservation Biology
University of California, Davis
e-mail: dcerman@ucdavis.edu
43200 East Oakside Place, Davis, CA 95616

We are aquatic ecologists who have reviewed over the past several years many of the freshwater poisoning projects conducted or proposed by state fish and game agencies, by the US Fish and Wildlife Service (FWS), and as permitted by the USDA Forest Service throughout the western U.S. We have read the EPA risk assessment for the reregistration of Antimycin A (Young and Seeger undated). We have reviewed much of the literature on effects of antimycin on non-target species (aquatic invertebrates and amphibians). We are submitting these comments as private citizens in the public interest. We are commenting specifically on the environmental effects of antimycin (trade name Fintrol) when used as a "piscicide" in the nation's streams, rivers, and lakes.

We submitted comments and data to the EPA in April 2005 and April 2006 on the need to retain NPDES permits under the Clean Water Act for the use of pesticides in the Nation's waters and on the problems that rotenone poisons cause for non-target species when used to kill fish in streams and lakes. It is with some sense of futility that we submit these comments on antimycin. We have little hope that the EPA will take appropriate action to protect the freshwater environment and non-target species from the application of poisons by fish and game agencies to the nation's most sensitive and pristine waters. The recent action by the EPA to eliminate NPDES permits for aquatic pesticides is a major step backward in protecting the environment, aquatic species, and water quality in the US.

We assume that independent scientists and other members of the public will be allowed the same opportunity for comment after the EPA deadline that employees of fish and game agencies and other government agencies were allowed following the comment deadline for rotenone use last year.

Myths about antimycin

Two myths arise repeatedly in discussions of antimycin. One is that antimycin is an antibiotic (e.g., Dawson and Kolar 2003). The second is that it has no lasting impact on non-target species.

We know of no record that antimycin has ever been registered with the FDA as an antibiotic for either human or veterinary use. It has been known since

at least 1973 that it does not kill most bacteria, and is therefore not an antibiotic in the common sense (Lennon and Vezina 1973). However, it may have been the unfortunate title of that 1973 paper, "Antimycin A, a piscicidal antibiotic," that led fisheries managers to believe that it was an antibiotic. At any rate, the myth has continued and is often repeated, perhaps in the belief that calling a substance an "antibiotic" sounds better somehow than acknowledging that it is a poison that kills many forms of life. It seems odd that the Lennon and Vezina paper was not reviewed in a 2002 assessment of antimycin A use in fisheries (Finlayson et al. 2002), nor was it included in the EPA risk assessment.

In addition to its use by fish managers to poison freshwater life, antimycin, along with rotenone, has become a common agent used in biochemistry to block mitochondrial electron transport and inhibit the respiratory chain at known locations. Both chemicals are routinely used to kill cells (apoptosis) in experimental biochemical research (e.g., Campas, et al. 2006; Ding, et al. 2006).

The second myth, that antimycin has little or no lasting impact on other non-target aquatic animals, is less investigated and has not been proven. Antimycin, like the various formulations of rotenone, can not be referred to merely as a"piscicide," thereby implying that it kills only fish. In fact, antimycin acts as a poison on many non-target organisms. It readily kills aquatic invertebrates and amphibians, as the EPA risk assessment has acknowledged.

The problem

It was never the intention of the Endangered Species Act to attempt to save one species while putting other species at risk of extinction. Therefore, whether or not all species of aquatic invertebrates and amphibians are present and survive the use of aquatic poisons must be examined in detail. So, also, should the EPA examine the long-term or permanent success rate of aquatic poisons to "restore" the target fish species. It seems within the purview of the EPA to examine the policies of state fish and game agencies and the U.S. Fish and Wildlife Service that have led to the release of so many non-native fish species into U.S. waters. This form of biological pollution continues without environmental review. It leads to the professed need by these same agencies to

poison streams and lakes in our most pristine waters, that is, National Parks, Wilderness Areas, and Outstanding National Resource Water (ONRW).

Many of the "restoration" projects being proposed and conducted at present are in water most likely to have endemic and rare species of amphibians and invertebrates as well as rare species of fish. State fish and game agencies, the USDA Forest Service, and the Fish and Wildlife Service have been taking a single-species approach to these poisoning projects, poisoning everything in an aquatic system and then replacing the fish species they want. The projects are often large and have little chance of succeeding in eliminating the unwanted fish species over the long term. "Complete elimination of undesirable fish is the exception rather than the rule in larger lakes and streams" (Lennon 1970).

Inadequacy of studies and evidence of impacts to non-target species

In the studies we have examined, the questions being asked and the analyses being done are inadequate to determine the impact of antimycin on freshwater communities and non-target species. The fundamental questions arising from the application of antimycin and rotenone to aquatic systems should be, 1) are species of non-target animals disappearing from the single or repeated use of poisons over many years? 2) Is the community of species changing in terms of relative proportions and numbers of individuals? And 3) what are the aquatic and terrestrial food web effects of these changes or losses in the short- and long-term?

Instead, however, the few studies that have been conducted on antimycin effects on aquatic invertebrates have asked, "Are invertebrates present again in the stream or lake following poisoning within a relatively short period of time (usually one year or less)?" The answer to that question will always be "yes" because some species of invertebrates are adapted to almost any environmental condition and will inhabit even the most disturbed sites.

Few studies on the effects of antimycin on non-target species have been published in peer-reviewed journals. Most are unpublished agency reports based on monitoring before and after the application of antimycin. Most of these

reports do not contain the raw data. None have been done at a species level. Antimycin has been used to poison aquatic habitats in the US for 40 years.

To our knowledge, no inventories of species have been done anywhere in the western US prior to a stream or lake poisoning operation. And we suspect the same is true for the eastern US. The monitoring studies done in co-ordination with poisoning operations are conducted at broader taxonomic levels than species, that is, at genus, family, order, and class levels. Total taxa and EPT (Ephemeroptera, Plecoptera, Trichoptera) measurements are not precise enough to answer the most fundamental questions about the outcomes of poisoning. Some species will be highly sensitive to antimycin and will disappear; others will be less so. Some species will rapidly inhabit a recently vacated ecological niche and will expand in numbers. Not all species of mayflies, stoneflies and caddisflies, the EPT, (incidentally, these are orders of insects, not families as stated in the EPA risk assessment) are sensitive to all impacts. Some are highly tolerant to some conditions (see, for example, a discussion in Erman 1996). Nor do we necessarily know that these groups of insects are the aquatic invertebrates most sensitive to antimycin. They may be, but data do not exist to make that assessment. Diptera, for example, are far more diverse (more species) in freshwater habitats than the EPT and some may be as sensitive to antimycin or more so than are some species of mayflies, stoneflies, or caddisflies.

In the study of a small trout stream poisoned with antimycin to remove non-game fish in Wisconsin, Jacobi and Degan (1977) found that the cranefly genus *Antocha*, a Diptera, decreased after antimycin exposure and continued a downward trend two years after the application of antimycin (Fig. 1). *Antocha* showed a similar response to rotenone poisoning in the Great Basin National Park (NP) where it was still missing three years after the poisoning (Darby et al. 2004). It was probably not the same species as that in Wisconsin, but illustrates the extreme sensitivity of some Diptera to aquatic poisons.

In the Great Basin NP study a species of mayfly was as sensitive to rotenone as was *Antocha* and also was still missing after three years.

Many species of invertebrates were significantly depressed immediately following the antimycin poisoning in the Wisconsin study (Jacobi and Degan

1977). The crustacean *Gammarus pseudolimnaeus* recovered rapidly and increased in biomass over its pre-antimycin levels (Fig. 1).

The same study reported dramatic changes in the amount of plant cover on the stream bottom and the total biomass of benthic macroinvertebrates. Both measures increased substantially up to two years after antimycin poisoning compared to the control stream (Fig. 2). In other words, in the stream poisoned with antimycin the community structure and food pathways became much altered up to 2 years after poisoning compared to a control stream. We were unable to evaluate other changes because data for only the most common 18 taxa out of 38 were presented in the report (Jacobi and Degan 1977). The study was conducted for only two years. Therefore, it is unknown whether or not the aquatic invertebrate and plant community ever recovered from this poisoning.

The EPA must recognize that following a large disturbance, a common response in streams at some point is an increase in abundance or biomass of some species. This response has been known since the earliest days of pollution monitoring, and should not be confused with a "recovery" of the stream ecosystem. The EPA risk assessment in reviewing a macroinvertebrate monitoring study in Great Basin National Park states, "However, by 9 months post-treatment, invertebrate populations had returned to pre-treatment conditions and in some cases exceeded pre-treatment abundance by over 300%…." This particular study was conducted for only one year after poisoning. There is no way to know whether or not the stream community and species recovered. The 300% increase in abundance of something can not be considered a recovery, but is rather indication of a disturbance.

Cosmopolitan, less sensitive, or "weedy" colonizer species tend to increase in numbers following a disturbance: poison released into a stream or lake is a disturbance.

There also seems to be some misunderstanding in the EPA risk assessment and in some of the studies we have reviewed about the meaning of the word "taxa." It refers to any level of taxonomic resolution. It is not synonymous with "species." If a taxon higher than the species level disappears, we know that at least one species is gone, but the taxon may have represented several or many

species. In most cases, the broader the taxon, the more species it represents. For example, a family represents far more species than a genus (with a few exceptions).

Most aquatic insects can be identified to species only by their adult forms, and mature forms are necessary for species identification for most other aquatic invertebrates (such as snails, clams, aquatic worms, crustaceans, etc.). A study reported by the National Park Service in Moore et al. (2005) states in the Executive Summary that "after one year all aquatic macroinvertebrate species were at or above pre-treatment levels." However, the rapid bioassessment methods of aquatic forms that were used in the study could not have determined species.

Monitoring of larval forms at genus levels and higher can often indicate impacts from a disturbance. It can not, however, tell us what species or how many may be lost from poisoning. Monitoring is not mitigation for poisoning. There is no mitigation for the loss of a species. And monitoring is not the same thing as a species inventory.

A healthy stream system may have 200 or more species of aquatic macroinvertebrates in it.

Stream poisoning is a special risk to species in springs, seeps and headwater streams. Many of the projects we have reviewed have poisoned these habitats. Such habitats are highly likely to contain rare, endemic, or relict species. Many have narrow distributions and narrow environmental tolerances. Many are not found lower in the stream system (Erman and Erman 1990, 1995; Erman 1998). They can not be replaced by downstream drift of larvae from upstream or by adults flying upstream to deposit eggs. And, of course, species that do not fly or have limited flight capability have even less chance of repopulating poisoned streams or lakes.

The terms "short-term" and "long-term" when referring to impacts on aquatic invertebrates are not defined by the EPA or in the studies we have reviewed. We have found no data collected on antimycin effects on non-target species for longer than two years following poisoning. We suggest that any impact still obvious one year after a poisoning event should be considered a

long-term impact, but that monitoring should continue as long as changes are apparent. That period may be five or ten years or more.

A study in California, South Fork of the Kern River, on drift of invertebrates following antimycin application showed major drift as a result of the poisoning (Stefferud 1977). Drift occurred as dead or dying invertebrates lost their hold on the bottom substrate and drifted in the water column. "The data gathered in this study indicate that use of antimycin as a piscicide has a definite effect upon the aquatic invertebrate community in cold mountain streams" (Stefferud 1977). "Dead or dying tadpoles were also collected in the drift nets" (Stefferud 1977). Funding for the planned continuation of that study was apparently withdrawn, and no further data were collected after the first year of results.

The EPA risk assessment seems to have relied uncritically on interpretations of data and studies provided to them from proponents of antimycin and rotenone for fish management. In our review of the Moore et al., 2005, report on the Sam's Creek study from the National Park Service, we found that there were few data presented to fully evaluate statements and conclusions. Different methods of sampling, different methods of taxonomic identification, and different levels of expertise were used to obtain data for number or identity of taxa. We were unable to differentiate what data were obtained under the various methods. We also found many errors in the data and missing sampling periods.

The few data that are presented reveal major problems in the report. There are three figures (Figures 8, 9, 10 in Moore et al. 2005) from the Sams Creek macroinvertebrate study and some additional numbers given in the text. There were 5 control and 4 treatment sites in the study. The so-called Treatment Site 9, however, was outside the boundaries of the antimycin exposure zone (i.e., downstream from the project boundary at stream barrier 646 m). The authors claim the station was affected in 2001 by the potassium permanganate detoxification process but (perhaps?) not antimycin. In either case, it did not receive the same treatment as the three other treatment stations.

There are no data on concentrations of antimycin A or $KMnO_4$ reported from samples in Sams Creek to judge the exposure of macroinvertebrates from

site 9. The change in total taxa number (Fig. 8) and EPT taxa number (Fig. 9) for site 9 suggests something happened.

A portion of Sams Creek and Starkey Creek (the farthest upstream locations) was poisoned with antimycin in October 2000. Two macroinvertebrate Treatment Sites (site 2 and site 4) lie within the treatment area of this poisoning event. Three different releases of antimycin occurred because the NPS personnel considered the dose insufficient to kill all fish (see p. 15-17 in Moore et al. 2005). Nevertheless, the report summarized "The observations of October 25, 26, and 27, 2000, provided evidence that the antimycin was eliminating rainbow trout but that it was only effective over a much shorter vertical distance..." (Moore et al. 2005, p. 17). We are unable to determine from the report whether or not the data reported in Fig. 10 for the period Sept./Oct. '00 was before or after the antimycin release. Thus, the interpretation of subsequent samples at site 2 and 4 taken the following year in September 2001, and considered "before" conditions is unclear. It is possible that antimycin released in the upstream reaches in October 2000 affected sensitive taxa of macroinvertebrates. Taxa loss or replacement of sensitive species may have already occurred at these sites. Thus, after another antimycin exposure in 2001 further changes in taxa in October 2001 and September 2002 would be confounded.

If stations 2 and 4 were poisoned in 2000, they are not "before" treatment stations for the purposes of Figures 8 and 9. For some reason the "before treatment" data referred to in the report and collected in 1996-97 were not used in these figures.

We are told nothing about the use of potassium permanganate in 2000 and do not know if it affected station 9 at that time as well.

Data presented (Figures 8, 9, 10) are internally inconsistent from one figure to another, and do not correspond to text references to the "same" data.
For example, Fig. 10 summarized the total number of taxa collected at all sites for all dates. These values can be compared for the dates of September 2001 and October 2001 shown in Figure 8. Of the nine values representing the number of total taxa before poisoning (September 2001), seven are different between Fig. 8 and Fig. 10, and for the 9 values representing October 2001, four appear

different. In other words, 61% of the supposed same data for two sampling periods differed between Figs. 8 and. 10.

In addition, the authors' text reference to data (p. 23) concerning (treated) site 9 stated total taxa declined from 61 in September 2001 to 40 taxa in October 2001. These values do not correspond to data in either Figure 8 or 10 (58 to 46 and 61 to 47, respectively).

Nevertheless, the National Park Service carried out its own analysis of variance on the number of total taxa (and EPT taxa) before (Sept. 2001) compared to 1 year after (Sept./Oct. '02) antimycin exposure using all 9 stations for treatment and control. They found no significant differences (although no ANOVA table was presented.) We are unable to fully replicate the analysis they performed without the full original (correct) data. However, we used the difference in number of total taxa shown in their Figure 10 between Sept. '01 and Oct. 01 and Sept./Oct. '02 (Fig. 3).

Our results suggest that there were differences before and after antimycin and treatment and control. In the ANOVA of just the 1-year difference in total taxa, the result is a significant difference at $p=0.0901$. We reject a null hypothesis at less than $p= 0.05$ because of the very weak power of the test with so few degrees of freedom and other uncertainties about the data.

Additional uncertainties about the study appear in the report. In the section on methods for the macroinvertebrate study, the report states "In the laboratory, aquatic insects were identified to the lowest taxonomic level possible" (Moore, et al. 2005, p. 9), and also "teams of experienced collectors used a multi-habitat approach to conduct aquatic macroinvertebrate sampling for each sample collected." But, later, explaining variation in samples from control stations, the report states: "Variation between samples occurred because: 1) the same collectors were not available for each sample, 2) each collector did not have the same field identification expertise for a particular taxon, or 3) were uncertain of how many potential taxa might be represented by what appeared to be a single taxon in the field" (p. 21). These contradictions leave us questioning, were identifications made in the laboratory or in the field? Were collectors experienced

or were they not? They also represent another large inconsistency in the methods and, therefore, in the data.

Without seeing the original data we can not answer the many questions raised by the Moore et al. report. If the EPA is going to rely so heavily on these studies to make their determination of risk assessment, we strongly recommend they obtain and analyze the original data and send it out for independent peer review by scientists who have no connection to, or interest in, promoting the use of aquatic poisons.

In our analysis of studies on rotenone effects in California, we found that the California Department of Fish and Game final reports to the Regional Water Quality Board misrepresented invertebrate impacts that were obvious in the raw data (see Erman and Erman comments on rotenone submitted to the EPA, April 2006)

Problems with Antimycin Application

Agency personnel have difficulty correctly applying the target dose of antimycin to streams. Recent examples are revealing. During the project in Sams Creek in the Great Smoky Mountain NP (Moore et al. 2005), personnel were unable to regulate antimycin dosage for two days in the initial stream poisoning in October 2000. "Unfortunately, the bottle containing the correct amount of antimycin for Sams Creek was inadvertently switched with the bottle for Starkey Creek" (Moore et al. 2005, p. 16). Personnel repeatedly tried different applications, new batches of antimycin, and increasing concentrations because sentinel trout failed to die as fast as expected. These procedures were eventually halted by the third day when "...additional concerns related to *Neophylax kolodskii* (a caddisfly thought to exist only in the treatment area) were raised as was the issue of not completing the project within allotted time frames..." (Moore et al. 2005, p. 16).

It is worth noting that actual measurement of antimycin in the stream sections was not conducted (is it possible with existing technology?), and there is no further information in the report concerning the fate of the endemic caddisfly species. We also wonder whether the detoxification station, cued by dye in the

water and not the presence of antimycin, might also have operated for a period of time with unknown effects on downstream invertebrate populations.

This episode at Sams Creek is reminiscent of a project in Wisconsin in 1972 in which errors in calculating dosage and equipment failure resulted in four times the concentrations administered over the "target" values (Jacobi and Degan 1977).

When antimycin poisoning of Sams Creek was resumed in September 2001, the project lasted over 11 days during which time potassium permanganate was used on nine days at the single detoxification station for a total of 64 hours (Moore et al. 2005, Table 2, p. 21). The authors state that in treatment site 9, below the detoxification location, "Apparently the cumulative effect of nine days of treatment with this strong oxidizer eliminated the Ephemeroptera (mayfly) taxon and all but one individual in the common stonefly family Peltoperlidae from this sample site" (Moore et al. 2005, p. 23).

The target concentration of antimycin relies on estimates of stream flow, among other factors. Measurement of stream discharge by velocity-cross section techniques is known to have uncertainty. Under ideal conditions errors in discharge can be as small as 2% (standard error of the estimate) or as large as about 20% when conditions are poor. (Sauer and Meyer 1992).

Poison drip stations are allocated along a stream course according to "best guesses" of past experience elsewhere for how far a lethal concentration will travel (e.g., Moore et al. 2005). It is common practice not only to drip rotenone or antimycin into the stream but also to deliver additional unknown quantities to springs, seeps, side channels, pools, and back eddies (Darby et al. 2004, Moore et al. 2005). For example, in the project in the Great Basin National Park, Darby et al. 2004 stated "...rotenone dry powder was mixed with sand and gelatin with handfuls deposited in rivulets that fed the main channel from seeps and springs" (p. 5). And elsewhere "Concentrations of antimycin averaged 8 μg/L. Concentration within various headwater reaches often exceeded 25 μg/L to compensate for spring and seep inflows between drip stations. Back eddies of the stream and adjacent springs and seeps were treated with 250 ml of Fintrol using a backpack sprayer" (Darby et al. 2004, p. 5).

In a more recent project in Arizona, antimycin was applied by the usual drip stations and also by antimycin laden sand into pools and by backpack sprayers to isolated water bodies, backwaters, and vegetated stream margins "...with renovation crews instructed to approximate an application of 50 $\mu g/L$" (Dinger and Marks, in press). These procedures hardly constitute rigorous control of application rates and given the fact that few projects report actual (rather than "target") concentrations over time; true exposure values are speculative. If, as suggested in the EPA risk assessment, some limitations will be recommended for frequency of application, we suspect that agencies will merely substitute higher dose rates to insure lethal conditions. In other words, more projects would operate at the manufacturers legal limit on the label for antimycin. Already, as seen in the Fossil Creek project, state agency personnel in Arizona opted for levels of 50 to 100 $\mu g/L$ antimycin A because of concerns that water quality would reduce efficacy and the desire to have total fish kill on the first try (Dinger and Marks, in press).

The Dinger and Marks study (in press) reported that antimycin killed invertebrates, many taxa were still missing after 5 months, and there was a shift to "more tolerant taxa." No changes in taxa occurred at the control station during those five months. But the study was marred by a permanent change in flow after the first five months. Nevertheless, the authors continued collecting samples for two years after the antimycin poisoning. Some taxa had not recovered after two years. The authors do not report taxa numbers or type at the control station after two years. Whether or not the taxa missing after two years were from antimycin or the change in flow is unknown.

The Dinger and Marks study is the highest "target" rate of antimycin application reported in invertebrate studies we have reviewed, but there were insufficient instream measurements of actual concentrations to determine what levels were reached in the past. In our review of rotenone projects, for example, we found that in Silver King Creek, CA, the target level of rotenone (which is measured) was 25 $\mu g/L$. Concentrations measured on several occasions at a single downstream monitoring station, however, showed rotenone plus the first decay product (rotenolone, also poisonous) reached 40 $\mu g/L$ (Flint et al. 1998).

Those measurements did not include the equal amounts of other cube resins, also poisons, in the Nusyn-Noxfish.

The routine procedure of adding “handfuls” of poison-laced sand, of using backpack sprayers “to approximate an application rate,” and of other uncontrolled methods of dispersing poison render meaningless approximations of actual instream concentrations. In addition, we urge caution in making the judgement that a single high concentration of antimycin is more toxic than repeated releases of a lower concentration. The issue for animal survival is exposure to a poison, that is, time and concentration.

We have noticed in recent environmental assessments that agencies do not want to reveal or decide on the poison or formulation they will use. In a recent Finding of No significant Impact on an extremely large poisoning project in New Mexico, in the Rio Costilla watershed (over 150 miles of stream, 25 lakes, and a reservoir) the poisons and formulations to be used are not specified in the public document. The same tactic is being used in the Lake Davis watershed in California by the California Department of Fish and Game. (Antimycin is not proposed for use in the California study, however, because at present it is not allowed in California.) That watershed and reservoir was poisoned about 10 years ago and is now slated to be poisoned again because of a total failure to eliminate the targeted fish species. We must assume that agencies may use more than one poison, as often as they want, in amounts as high as they want, and without monitoring or oversight by any independent agency.

The Fintrol label (FIFRA approved) does not restrict concentration at present. It recommends up to roughly 25 μg/L if cold temperatures and high pH exist in the receiving water. It says the only way to determine lethal dose is to perform a bioassay. It does not contain an explicit legal limit.

The EPA draft risk assessment states on p. 18, "Although maximum treatment rates are not stated on the label, this risk assessment is based on an upper-bound treatment rate of 25 μg/L applied once per year." The EPA Table 3 (p. 18) also reiterates that the maximum rate per application is "roughly" 25 ppb (μg/L). But in an Addendum to the EPA risk assessment much higher levels of antimycin and more applications per year are listed.

Further, the paper by Dinger and Marks states, "However, the label allows for treatment outside this limit when 2 conditions are met: 1) bioassays indicate the need for higher levels, and 2) permission from the state game and fish agency are [sic] required. For Fossil Creek both conditions were met (the treatment was performed by AZGFD), ensuring legality" (note: AZGFD means Arizona Game and Fish Dept).

Therefore, at present, the EPA has removed the requirement of NPDES permits and the FIFRA label says that if more than "roughly 25 μg/L" is applied, the agency doing the poisoning can determine whether or not to use more poison than recommended. There is no independent monitoring and no oversight by other agencies.

A statement appeared in a 2006 Decision Notice for a poisoning project on Crawford Creek, Montana: “Antimycin (another EPA registered piscicide) will not be used in this project because of recent information related to quality control of product and reduced effectiveness.” If the product has poor quality control and is ineffective, why is it being used in natural waters at all; and why are these problems not part of the EPA risk assessment discussion?

<u>Interactions with other pesticides present in water</u>.

The EPA risk assessment has evaluated antimycin as if there are no other complicating chemicals in the environment that may increase toxicity. Antimycin works by interfering with the electron transport system in cell mitochondria (Dawson and Kolar 2003). With many toxins, such as rotenone and antimycin, the effect on the transport system is mediated by an organism’s natural defenses. But when certain compounds are also present in the environment, toxicity is increased because the natural defense system (cytochrome P450) is reduced (Li et al. 2007). This result is well established for the role of piperonyl butoxide (PBO) as a synergist in formulations of rotenone and other insecticides. However, it is also known that other pesticides themselves may function much like PBO (in blocking cytochrome P450) and, hence, increase substantially the toxicity of insecticides. The EPA is aware of these relationships, and in their rotenone risk assessment cited the work by Bills et al., 1981, for example, that showed PCBs multiplied the

toxicity of rotenone to fish. There is other work that has established similar relationships among a range of pesticides and herbicides (e.g., Bielza, et al. 2007). There is also strong evidence that residues of common herbicides and insecticides (and PBO) may remain in aquatic sediments (Woudneh and Oros 2006) or in the water, even in remote national parks (LeNoir et al. 1999, Angermann et al. 2002).

It is likely that low level residues of pesticides are present now in many aquatic habitats, and these levels may increase without the further review or analysis previously required by NPDES permits. At present, we are unaware of any fish poisoning project that has analyzed water or sediments for low level pesticide residue prior to applying rotenone formulations or antimycin.

Has the EPA considered the role of potential synergists on the toxicity of antimycin in its risk analysis, and are these risks to non-target aquatic invertebrates and amphibians accounted for under the proposed reregistration?

Summary and conclusions

Antimycin clearly affects non-target species and probably eliminates some and, possibly many, invertebrates and amphibians. Some species may be permanently exterminated. No studies to date have proven that antimycin is harmless. Several studies have shown impacts to non-target animals and communities at broad taxonomic levels.

The EPA was wrong to eliminate National Pollution Discharge Elimination System (NPDES) permits for the use of stream and lake poisons. NPDES permits, issued under the Clean Water Act, allowed projects to be evaluated by an independent agency (in California, Regional Water Quality Boards and the State Water Board) on a site-specific basis, at the local level, and to include monitoring requirements. In California, the NPDES review assures that projects are in compliance with the Basin Plans for each regional water district. The NPDES permit review also determines whether or not a project is likely to cause harm to non-target species and whether or not the project protects beneficial uses of water.

Stream and lake poisoning projects, being conducted and proposed by agencies at present, are large covering many stream miles and many lakes. They are often in the most pristine areas of the country—Wilderness Areas, National Parks, and Outstanding National Resource Waters. These areas deserve the greatest protection and are most likely to have endemic and/ or rare non-target species.

Stream and lake poisoning projects to eliminate unwanted fish species have a poor record of long-term success. Agencies poison waters for two or three years, unwanted fish return within about 10 years, and the agencies begin poisoning again. Agencies have a long record of errors and mishaps with their poisoning operations.

We recommend that antimycin reregistration be denied for all but small, artificial ponds and self-contained fish farm ponds that have no outlets.

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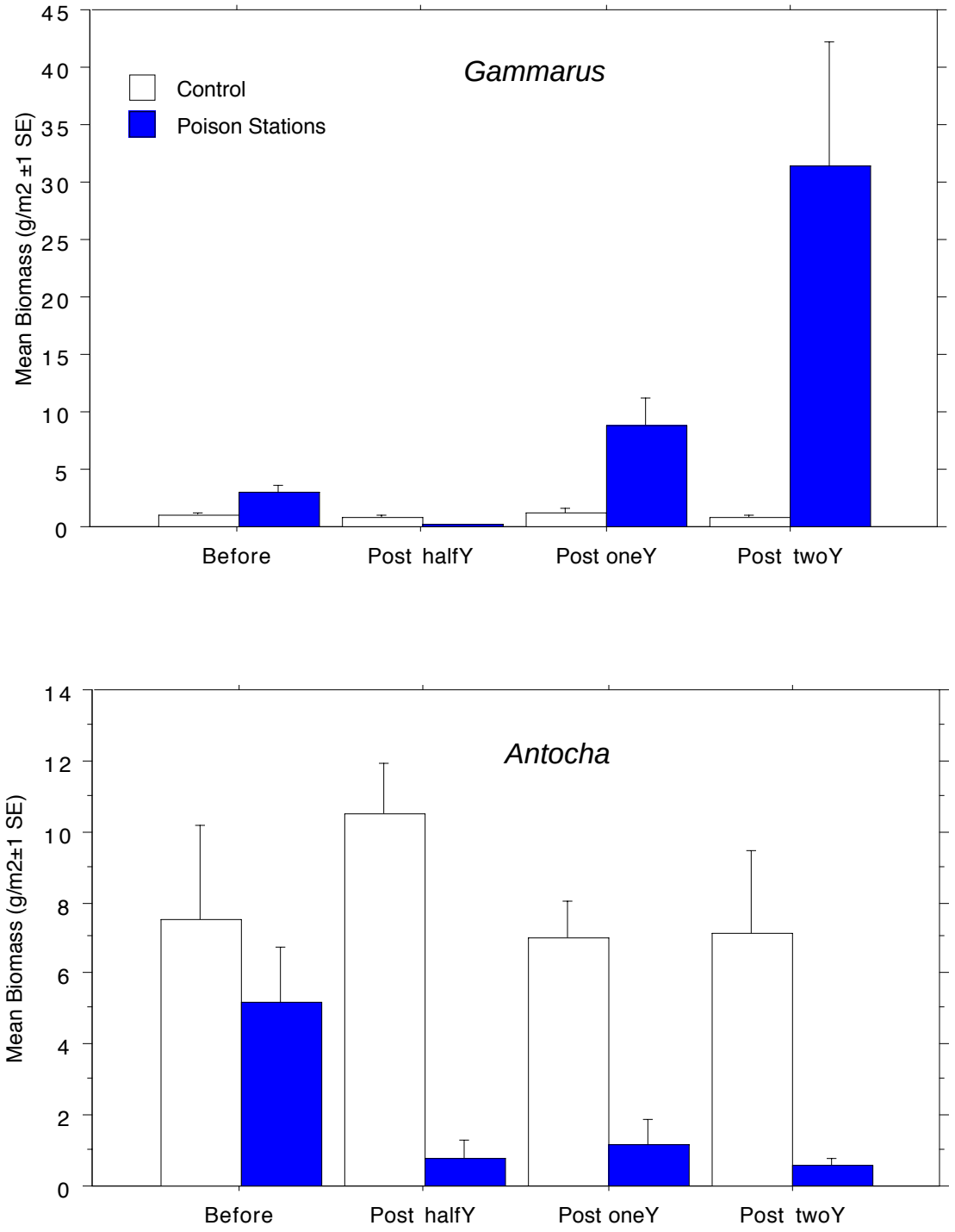

Fig.1. Change in biomass of the crustacean *Gammarus* and crane fly larvae *Antocha* before and 0.5, 1, and 2 years after treatment with antimycin (Data from Jacobi and Degan 1977).

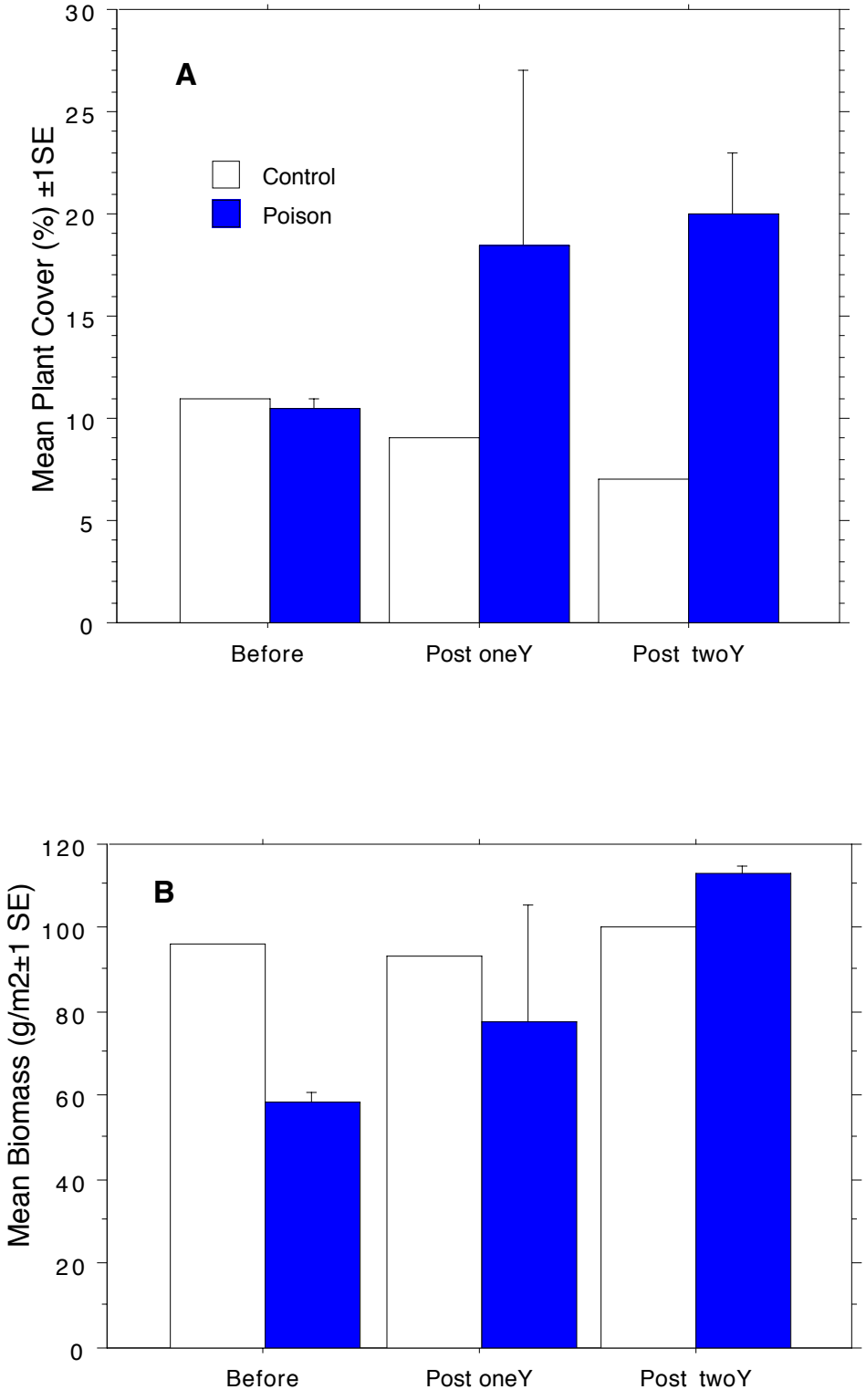

Fig.2. Percentage of stream bottom covered by aquatic plants (A) and total benthic macoinvertebrate biomass (B) in treated and control streams before, 1 year and 2 years after antimycin po.
(Data from Table 6, Jacobi and Degan 1977)

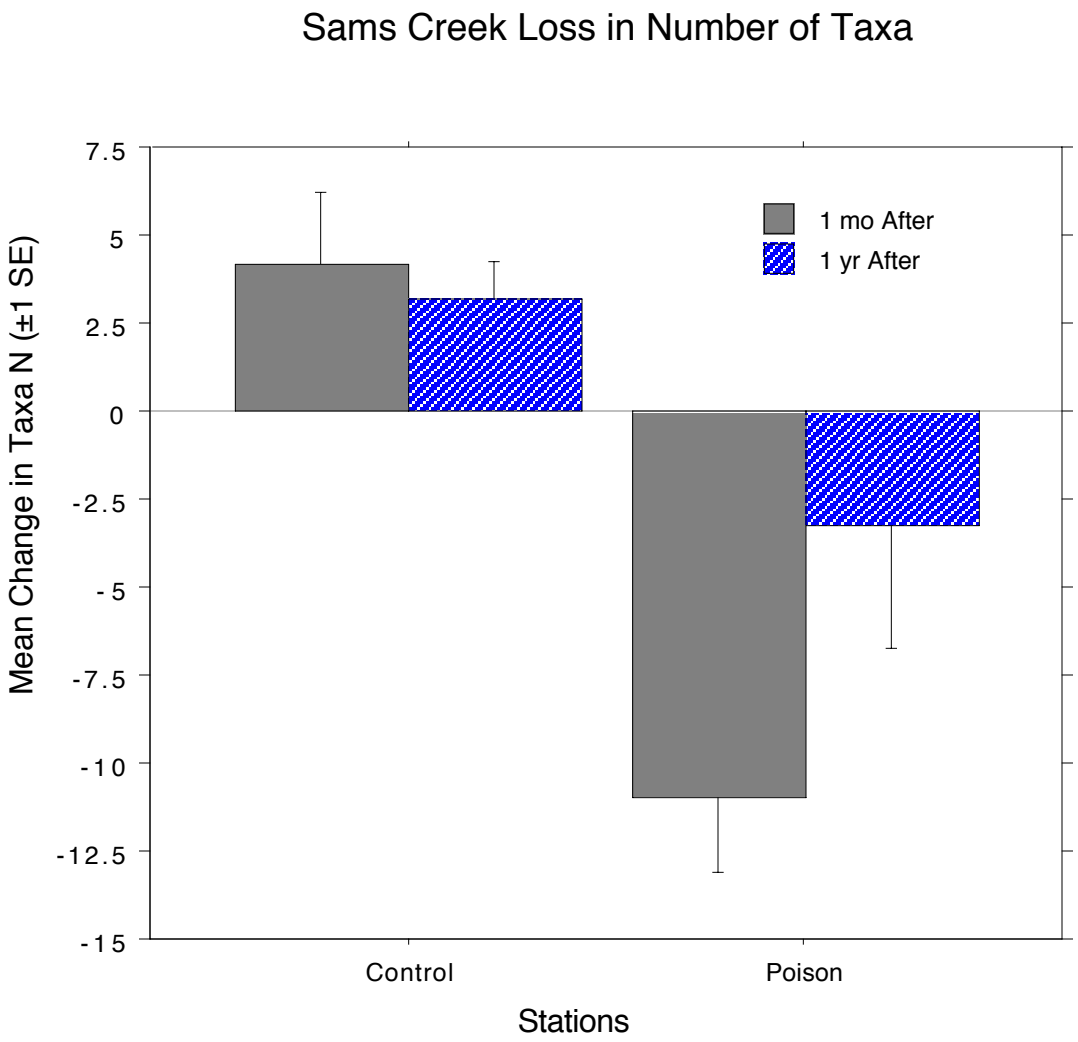

Fig. 3. Change in total number of taxa at control and antimycin treatment stations in Sams Creek. Bars represent the number of total taxa collected before antimycin treatment in September 2001 minus the total number of taxa collected in October 2001 (1 mo after) and September 2002 (1 yr after) in control and treatment sites. (Data from Fig. 10, Moore et al. 2005). The treatment sites averaged 11 taxa lost in the month after poisoning and 3.2 taxa 1 year after antimycin while the control sites gained 4.2 and 3.2 taxa for the same periods.