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LETTER TO THE EDITOR . . .

Salmonid Whirling Disease: Status in the United States, 1985

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In a recent description of the life cycle of *Myxosoma cerebralis*, the etiologic agent of whirling disease in salmonids, Wolf and Markiw (1984, Science 225: 1449–1452) demonstrated that a tubificid oligochaete was the obligate alternate host and that an organism produced in the worm and having the unique morphology of a triactinomyxon was the long-sought infectious stage for fish. That report presented the first scheme of a myxosporean life cycle that was supported by experimental data instead of authoritative opinion. Since then, inquiries from administrators, managers, and biologists have suggested a need to review the new findings as they relate to fisheries resources in the United States where this exotic disease was once considered rare, but where it now must be recognized as firmly established. My purpose here is to consider some aspects of whirling disease in the light of the new knowledge that tubificids are required in the life cycle, and of how that information relates to existing measures of control and to additional measures that should be considered.

Whirling disease is a chronic, noncontagious infection of salmonid fishes that is causally related to a protozoan parasite known as *M. cerebralis*. Because salmonids are propagated widely in the United States in areas where whirling disease is now established, the infection is found commonly in the highly susceptible young—fingerling to subadult—of rainbow trout (*Salmo gairdneri*) and brook trout (*Salvelinus fontinalis*). Clinical cases show external signs that are first evident as dark pigmentation of the caudal peduncle and caudal fin—the so-called black tail sign. Next to appear is the abnormal tail-chasing behavior from which the disease derives its name. In time, structural deformation of the head or body develops. However, fish with infections of low intensity show none of these signs and often appear and act normally.

Histologically, focal to regional areas of lysis and damage occur in cartilaginous tissue, and spores of *M. cerebralis* are present in or near such lesions. The metabolic activity of the parasite is credited with causing the lesions, which eventually result in structural deformation during bone deposition.

The presence of *M. cerebralis* spores is universally considered to be pathognomonic for whirling disease.

Whirling disease was introduced accidentally into the United States, presumably from its origin in Europe, during the early 1950's—to Nevada in the West and to Pennsylvania in the East. Within watersheds, the disease can spread naturally, but it has been disseminated also by man—sometimes unwittingly, but sometimes covertly. Consequently, whirling disease is now known to have been spread to 10 other states: California, Connecticut, Massachusetts, Michigan, New Hampshire, New Jersey, New York, Ohio, Virginia, and West Virginia. New Hampshire was added to the list in 1980 and New York in 1984.

No state has yet been successful in eradicating *M. cerebralis* from its waters. Unquestionably, the application of appropriate control measures can lower the prevalence of whirling disease, as well as

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reduce the more insidious problem of subclinical infection. However, once the organism is present in natural environments, eradication—a complete stamping out—will require extraordinary effort.

Although whirling disease has been known and studied in Europe since the early 1900's, the life cycle has remained an enigma. The breakthrough was made in the United States during the early 1980's as the culmination of a sequence of experimental research findings. First, Markiw and Wolf (1983, J. Protozool. 30: 561–564) found that a tubificid oligochaete was required for *M. cerebralis* to yield the stage that was infective for fish. Next, the long-sought infectious stage of the organism was reported and the life cycle was defined (Wolf and Markiw, 1984, op. cit.).

In brief, mature spores of *M. cerebralis* reach the environment when infected fish die and decompose or are consumed by scavengers. Alternatively, the spores are released in feces in viable condition when infected fish are cannibalized or eaten by a predator (Taylor and Lott, 1978, J. Protozool. 25: 105–106).

The freshly released small disk-like spores of the myxosporean *M. cerebralis* are not infectious for trout. Instead, the spores infect the tubificid and in the gut of the worm undergo a slow but dramatic morphologic change to a much larger three-tailed and grapple-shaped organism known as an actinosporean and more specifically as a member of the group with the genus name *Triactinomyxon*. Triactinomyxons have been recognized as parasites of annelid worms for more than 90 yr, but have never been shown to initiate autoinfection in their host. Instead, the triactinomyxon—for which the name *T. gyrosalmo* was proposed (Wolf and Markiw, 1984, op. cit.)—produces whirling disease when infected worms are eaten by a susceptible trout or when fish encounter the organism when it is waterborne. The grapple-like appendages are thought to enable waterborne organisms to lodge in fish gills and there to effect transfer of the parasite's internal bodies or sporozoites to the respiratory capillaries of the fish.

Tubificids are normal inhabitants of aquatic environments and are particularly abundant in rich organic soils. Dense populations are commonly considered to be an indication of organic enrichment or (in the general sense) pollution. Tubificids are typically abundant and occur in dense red patches in settling basins and streams that carry effluent (bearing residues of food and feces) from trout hatcheries. More important, the worms find ideal habitat in earthen ponds and raceways where cleaning is both difficult and infrequent. Once introduced, the whirling disease organism readily becomes established in such habitats. In contrast, concrete or plastic-lined hatchery facilities that are kept clean and have an uncontaminated water supply can be kept free of the disease.

Knowledge of the life cycle logically leads one to consider measures that could be applied to prevent whirling disease. Such measures include eradication of the essential tubificid or the waterborne triactinomyxon. In theory, tubificids can be killed by thorough drying or chemical treatment of the aquatic soil. Although the life cycle was not then known, Hoffman and Hoffman (1972, J. Wildl. Dis. 8: 49–53) conducted laboratory tests of several compounds and provided presumptive evidence that chemical disinfection—that is, killing the spores of *M. cerebralis*—was possible. In actual practice at hatcheries, however, chemical disinfection has been only partly successful and has not resulted in eradication of the disease. The biology of the tubificid, its burrowing behavior, and the nature of aquatic soils virtually ensure survival of some worms beyond the reach of chemicals that can be applied and maintained economically in adequate concentration and to sufficient soil depth. Considering that rainfall is appreciable at

most locations where trout are reared, the drying of pond soils is not a realistic eradication measure.

Selectively lethal chemicals have a potential for achieving eradication, although no such compounds are yet known. Application would probably have to be prolonged to kill deeply buried worms or the progeny worms inside cocoons. Moreover, the safety of other aquatic biota must be considered. The overall requirements for registration of such compounds by regulatory agencies of the United States government are formidable.

Decontamination of water supplies to remove or kill the triactinomyxons is an alternative approach to eradication. Cleanup can be effected by filtration, chlorination, ozonation, or ultraviolet irradiation—alone or in combination.

Although the actual infectious stage was not known, Hoffman (1975, J. Wildl. Dis. 11: 505–507) showed in laboratory tests that filtration and ultraviolet irradiation (UV) reduced waterborne infectivity, and that a combination of 25-μm filtration and irradiation at 27,650 microwatts/sec/cm^2 was most effective. Quantification of ozone and chlorine needed for disinfection of water supplies has not been determined.

Although two methods of decontamination have been partly successful, all of the various systems require an energy source, and thus are vulnerable to interruption. Accordingly, back-up fail-safe systems are required. Filtration is additionally vulnerable to heavy incursion of silt and debris that can occlude the devices and negate the effects of UV irradiation.

Drugs and chemicals are used routinely to treat and, in some instances, to help prevent certain bacterial and parasitic conditions in fishes under husbandry. Drugs are often highly successful in controlling infection or reducing mortality, but their use neither eliminates the pathogen nor prevents its entry into some fish; in fact, some fish are almost certain to become infected and to harbor the pathogen.

Year-long continuous feeding of six drugs to young trout held in water known to be contaminated with the infectious stage of the whirling disease organism showed that although clinical signs did not appear, infection was not prevented (Taylor et al., 1973, J. Wildl. Dis. 9: 302–305). One antibiotic, one nitrofuran, one sulfonamide, and three antiprotozoal compounds were tested. Best results were achieved with the nitrofuran, which partly inhibited spore formation but did not prevent infection.

In many regions of the United States whirling disease has yet to be found; the stocking of such regions with trout that are minimally or even potentially infected with the whirling disease organism is wholly unacceptable.

Significant but nonetheless incomplete control of certain insect pests of agriculture and forestry has been achieved by introducing predator insects or bacterial or viral pathogens. Prospects for biological control of whirling disease are dim, inasmuch as the host fish themselves are prime predators of the tubificids and demonstrably contract the infection by ingestion. The tubificids undoubtedly have their own array of pathogens, but the identity of virulent bacteria or viruses is not yet known.

Now that the actual infective stage, the triactinomyxon, has been identified and produced experimentally, the application of immunologic methods holds promise for future control of whirling disease. As a prime example of progress in parasitology, researchers are looking toward vaccination of man against malaria as a new weapon to eradicate the disease. Although not yet developed, malarial vaccine is to be targeted against the infective stage produced by the mosquito.

The antigenic components of the triactinomyxon must be determined and

methods must be developed by genetic engineering to mass produce the antigen economically. Appropriate methods of administration would have to be determined, as well as safety and efficacy. Eventually such a product would require licensing by the U.S. Department of Agriculture.

The task of developing a practical vaccine against whirling disease is, as seen from the present vantage point, formidable but not impossible. Whether such a product would evoke complete and population-wide protection cannot now be determined. Beneficial but less-than-absolute protection will affect the disposition of fish stocks involved and the waters to be stocked.

In Europe the rainbow trout is propagated mainly as a table or food fish and in some places water supplies needed for the necessary large-scale production are ineradicably contaminated with the infective stage of whirling disease. Trout producers in such situations incubate eggs and rear the young—usually to fingerling stage or larger—in pathogen-free spring or well water. That practice exploits the fact that, although even yearling trout might become infected and produce spores of *M. cerebralis*, older fish seldom develop clinical signs of the disease. The rearing of trout to at least fingerling size in noninfective water takes them past the highly vulnerable fry stage. When carrying capacity of available pathogen-free water is exceeded and fish must be removed and exposed to the pathogen, their response can be tolerated. Fish become infected, but severe signs such as deformities are avoided, and the product can be successfully marketed because it is normal in appearance.

An alternative method has been applied in the United States. Brown trout (*Salmo trutta*) and coho salmon (*Oncorhynchus kistuch*) have high, but not complete, resistance to whirling disease. The brown trout is generally considered to be the original host of *M. cerebralis*, and hence its resistance is a feature of selection and adaptation.

In places where the only water supplies are contaminated, production of the highly susceptible rainbow and brook trout has been discontinued. Instead, the resistant brown trout and coho salmon (O'Grodnick, 1979, Trans. Am. Fish. Soc. 108: 187–190) are propagated. Even these resistant species are reared for 6 mo or longer in noncontaminated water—just as is done in the European method.

Statistically valid samples (5% prevalence) of trout and salmon so produced have been examined and found free of spores of *M. cerebralis*. However, resource managers of areas or regions where whirling disease is absent nevertheless look at exposed populations hypercritically. Quite understandably, fish of such exposed stocks should not be stocked in environments that are free of the infection.

Attempts to arrive at the overall significance of whirling disease lead to mixed results. From an ethical or conservative viewpoint—that of responsible resource protection—whirling disease in the United States is an exotic infection. Accordingly, if it cannot be eradicated, every effort should be made to confine it to areas or regions where it now exists; it should not be introduced to environments, regions, or areas where it is not now present. The responsible viewpoint is rational, safe, and readily defended, for it is axiomatic that existing fish resources should be protected. Also, it is more effective and less costly to exclude a pathogen than to eradicate it once it is established.

"What harm does whirling disease do in the wild—or even in the hatchery?" "Is it measurably harmful or just theoretically harmful?" "Is *Myxosoma cerebralis* really that bad?" "How can you justify ranking whirling disease with viral hemorrhagic septicemia?" It is true that whirling dis-

ease infections of high intensity in a hatchery kill some of the young, cripple survivors, and result in reduced growth. Yet, some hatcheries with enzootic whirling disease have continued to operate (and presumably profitably) for years. In the absence of the disease, the profit margin could probably be increased—but by how much? The answer is not known, and data are not available.

The effect of whirling disease in wild populations is totally unknown, but one can speculate that, because population densities are lower than in hatcheries, the risk of intense infection, clinical disease, and serious impact is reduced.

Questioning the significance of whirling disease is not just an academic exercise. North America has had the problem for nearly 30 yr and the formerly rigid attitudes seem to be softening. In relation to commercial operations (not equivalent to natural fishery resources) in the United Kingdom, official attitudes changed when it became evident that the disease was widely disseminated. As a consequence in early 1984, whirling disease was taken off the list of notifiable diseases.

Judging by the small number of diagnostic cases submitted to our laboratory and by the paucity of reports of new occurrences, whirling disease seemingly has not spread much during the past several years. That judgment should not lead to a complacent attitude, because one must ask, "Is anyone looking?" and the answer must be "Probably not—unless there are signs of the disease or other reasons to scrutinize fish."

Whirling disease exists in streams of several of the states, but just how extensively is not known. Determination of the extent of the infection depends on the methods used for detection. The most widely used method of detecting spores of *M. cerebralis* is the plankton centrifuge method that was popularized by O'Grodnick (1975, J. Wildl. Dis. 11: 54–57) and modified for greater sensitivity by Markiw and Wolf (1980, Can. J. Fish. Aquat. Sci. 37: 2225–2227). Unquestionably, the most reliable method of determining the presence or absence of whirling disease infection is that of exposing susceptible young trout as sentinel animals.

The significance of whirling disease in various kinds of natural environments with different kinds of resident, or short-term, salmonids needs to be determined. Eradication procedures will undoubtedly be considered under some circumstances. Where that approach is to be applied, it should be directed against both host animals of the parasite's life cycle—the salmonid fish and the tubificid worm.

Until reliable eradication procedures are developed, it is highly probable that man will have to tolerate the presence of whirling disease in wild waters of the United States and in fish hatcheries where it is already present.