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Coldwater Disease Vaccine: Promising Preliminary Results (cont. from Oct. 2007)

Flavobacterium psychrophilum is the causative agent of bacterial coldwater disease (Holt 1987) and rainbow trout fry syndrome (Lorenzen, Salsgaard et al. 1997) in young salmonids. This bacterium was first isolated in 1948 from Coho Salmon in Washington State (Holt 1987) and has since been shown to cause significant losses in commercial aquaculture operations in the United States (Bernardet 1997), Europe (Lorenzen, Salsgaard et al. 1997), Chile (Bustos, Calbuyahue et al. 1995) and Japan (Wakabayashi, Toyama et al. 1994). The bacteria was first isolated from Utah's Glenwood Sport Fish Hatchery in 1996 and has since been found to cause significant fish mortalities throughout the state's captive rearing program. To address this problem, a research team at the state's Fisheries Experiment Station has been working to develop a vaccine against the bacteria.

In a recent trial (see "Ichthyogram", Vol. 18, Issue 2) the team demonstrated that mortality in juvenile rainbow trout, which were vaccinated with *F. psychrophilum* antigens and experimentally infected with live *F. psychrophilum* bacteria, could be significantly reduced. Since that time, efforts have been underway to assess whether a change in serum antibody may have contributed to reduced infection and increased survival among vaccinated fish.

To answer this question, an Enzyme Linked Immunosorbent Assay (ELISA) was used to measure *F. psychrophilum* specific antibody levels in fish sampled at different time intervals (week 0, 6, 12) across all experimental groups in this first trial. At week 0 there were no *F. psychrophilum* specific antibodies found in serum samples collected from naïve fish. At week 6, *F. psychrophilum* specific antibody levels were slightly elevated in the treatment group that received vaccine alone and in the group which received the vaccine mixed with an immuno-stimulating adjuvant (Fig. 1). At week 12, antibody levels were again slightly elevated in the vaccine alone group, but levels were significantly higher in fish that received the vaccine mixed with the adjuvant (Fig. 2). *F. psychrophilum* specific antibodies were not found in control fish that were injected with phosphate buffered saline (PBS) at weeks 0 and 6 (Figs. 1 and 2).

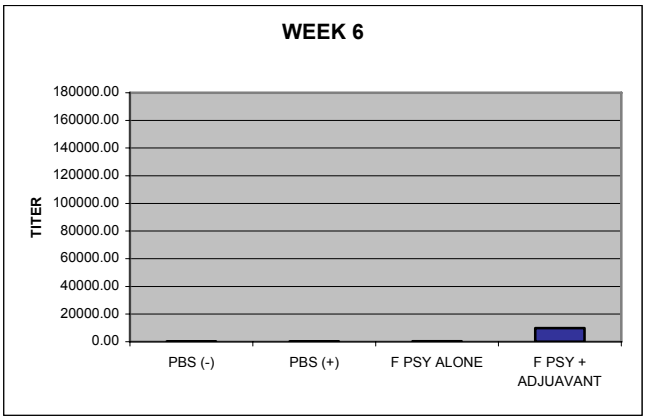

Fig. 1. Serum antibody in vaccinated fish at week 6

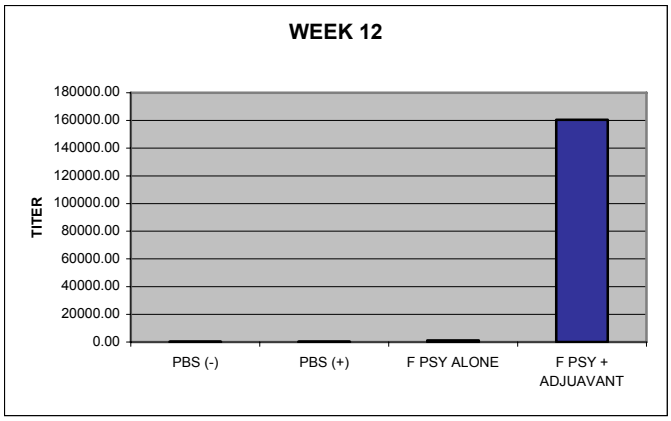

Fig. 2. Serum antibody in vaccinated fish at week 12

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Results from this analysis show that elevated *F. psychrophilum* specific serum antibodies were correlated with increased survival in fish under experimental conditions (see "Ichthyogram", Vol. 18, Issue 2). To confirm these findings, the team is currently working to replicate the trial described above. If successful, future efforts will include additional trials to identify whether a vaccine program can be developed to reduce infection and increase survival of fish that are exposed to *F. psychrophilum* in Utah's hatchery system.

Christine Swan

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Graduates Complete Intensive Fish Culture Class at FES

Pictured below are recent graduates of the fish culture class held in Logan from February 11-22, 2008. The class is held periodically to train new fish culture recruits within the Division of Wildlife Resources. The class this year was the largest ever, with thirteen students participating. In addition to the hatchery crew, regional biologist Aaron (Gimpy) Webber and Ute Tribal biologist Jay Groves attended. For the first time, college credit was made available to students through Utah State University. A wide variety of topics was covered on subjects as diverse as chemical safety, water quality, fish health and animal welfare issues.

A special thanks to the many instructors including Rick Hartman, Don Bone, Tim Miles, Eric Wagner, Anna Forest, Larry Dalton, Quentin Bradwisch, Christine Swan, Doug Routledge, Roger Mellenthin, Doug Lukes, Robert Schmidt, Wade Cavender, Chris Wilson and also Catherine Smith for making arrangements.

Pictured below, first row: David Barnhurst, Jared Cook, Matt Bartley, Randy Oplinger. Second row: Wes Pearce, John Coombs, Erin VanDyke, Jason Tull, Zane Olsen, Travis Dees, Aaron Webber, Mike McCarty and instructor Chris Wilson. Not present: Jay Groves.

Got worms? A review of *Tubifex tubifex* resistance to *Myxobolus cerebralis* infection

Whirling disease is caused by the parasite *Myxobolus cerebralis*. The parasite has a two-host life cycle, requiring both salmonid fish and *Tubifex* worms as hosts (Markiw and Wolf 1983). To become infected, a fish has to come in contact with triactinomyxons (TAMs), which are produced by the oligochaete host, which has ingested the myxospore stage (Wolf and Markiw 1984; El-Matbouli and Hoffmann 1989). *Tubifex tubifex* has been identified as the only oligochaete worm species capable of producing the TAM stage (El-Matbouli and Hoffman 1989). The myxospores are ingested by *T. tubifex* and the parasite develops extracellularly between epithelial cells of the gut where it grows and reproduces; after about 3 months, mature TAMs are released in the feces of the worm. A fish becomes infected when it comes into contact with TAMs, initiating the development of new myxospores to continue the cycle.

Whirling disease has caused dramatic and highly publicized reductions in some populations of wild rainbow trout, *Oncorhynchus mykiss* (Nehring and Walker 1996; Vincent 1996). Yet some populations of wild trout are not affected as dramatically even when they appear to have the same environmental conditions (Nickum 1999). The dose of TAMs is one of the single most important factors that determine the severity of whirling disease among wild trout populations (MacConnell and Vincent 2002). Recent studies have indicated that differences in worm abundance and susceptibility may be responsible for variation in TAM doses, and in turn, variation in fish population effects (Arsan et al. 2007).

This brief review focuses on the oligochaete host, *Tubifex tubifex,* and differences in susceptibility to infection by *M. cerebralis* among various populations of *T. tubifex*. As Arsan et al. (2007) summarized in their study, there are 6 cryptic mitochondrial lineages of *T. tubifex* (lineages I- VI) (Sturmbauer et al. 1999; Beauchamp et al. 2001). North America is known to harbor lineages I, III, V, VI and, most recently identified in Alaska, lineage IV (Beauchamp et al. 2001; Arsan et al. 2007). Europe has lineages I-V (Sturmbauer et al. 1999). Worms of lineages I and III are susceptible to the parasite and produce TAMs; worms of lineages V and VI do not produce TAMs (Beauchamp et al. 2002; Dubey et al. 2005). Research in Colorado has indicated that lineage III is the most widely distributed of the various genotypes and is found in the same waters as lineage I, V, and VI (Thompson 2007; Nehring 2007). A qPCR process developed by John Wood of Pisces-Molecular Inc. is currently available for simultaneous testing and quantification of mDNA from four different lineages.

The newest efforts in whirling disease control have been to take this information and study how resistant worms can combat the problem. The basic theory is that an infected waterway that harbors a population of the susceptible strain of *T. tubifex*, lineages I and III, can be replaced or augmented with a population of resistant worms, lineages V and VI. Even if the susceptible worms are not entirely eradicated, the resistant worms still consume and "deactivate" the spores in the sediment, lessening the total production of TAMs.
This theory is supported by previous studies. Areas highly affected by the disease are predominated by susceptible worms, whereas areas less affected by the disease have higher populations of the resistant strains (Beauchamp et al. 2005). In another study by Beauchamp et al. (2006), they reported a 70% reduction in total TAM production in a mixed colony verses a colony of all susceptible worms.

The exact advantageous mechanisms with the resistant strains are not clearly defined. In some laboratory settings, the resistant strains have been shown to have a competitive advantage over the susceptible strains by completely replacing the susceptible strain with the competitive strain after 3 months of cohabitation. Beauchamp et al. (2006) showed that field-collected *T. tubifex* cultures, which contained multiple lineages, resulted in cultures that were dominated by the laboratory-introduced resistant lineage V. An important consideration is the infection and resulting parasitism effect of *M. cerebralis* on the *T. tubifex* population. The reproduction rates, biomass, and abundance decrease when the worms are exposed to the myxospores (Stevens et

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al. 2001; Kerans et al. 2004; Arsan et al. 2007; Steinbach-Elwell et al. 2006).

Interestingly, recent studies examining competition between susceptible and resistant strains seem to differ in results. Dubey et al. (2005) found the resistant strain out-competing the susceptible strain. Winkelman et al. (2007) found the opposite, although they were comparing different lineages for the resistant strain (VI verses V). When comparing between lineages III and VI, VI seems to have a wider tolerance of temperature based on its growth and reproduction at experimental temperatures (Dubey et al. 2005). Dubey et al. (2005) also determined that photoperiod had no or little effect on either lineage. Infection level within lineage III has a wide range, even at similar temperatures (Dubey et al.2005; Blazer et al. 2003; Table 1). Beauchamp et al. (2005) also reported infection rates ranging from 2.3%-30.0% for lineage III Windy Gap *T. tubifex*. Highest survival was seen at a temperature of 17°C and was significantly higher for the lineage VI (Dubey et al.2005; Beauchamp et al. 2002). Although contrary to other findings, Dubey et al. (2005) found no difference in growth or reproduction with exposed verses unexposed for both lineage III and VI.

TABLE 1.- Infection rates of *M. cerebralis* of lineage III *T. tubifex* reported for Dubey et al. 2005, and Blazer et al. 2003. Exposure was at 500 and 350 spores per worm for Dubey et al. 2005 and Blazer et al. 2003, respectively.

Temperature °C	Infection rates for Lineage III	
	Dubey et al.	Blazer et al.
5	4.30%	
9		8.3-16.7%
13		6.3-22.9%
17	3.30%	12.5-33.3%
20		0%
27	0%	

When comparing between lineages III and V, the susceptible strain (lineage III) is not as clearly disadvantaged. Winkelman et al. (2007) has studied the competition both in field and laboratory settings. The data is still preliminary, but field results show that the lineage V populations are surviving but not migrating up or downstream. In the laboratory, the susceptible lineage III has shown a competitive advantage over lineage V when no infection is occurring (Winkelman et al. 2007). However, Kowalski and Bergersen (2003) noted that lineage III worms had lower LC_{50} levels for two chemicals (Baylucide and TFM) than lineage V worms.

Another consideration is each lineage's microhabitat preference. Even less research has been done on specific lineage preference within a stream. To date, Winkelman et al. (2005) has shown that proportions of fine and coarse sand influences the lineage composition. Lineage V worms preferred greater depths and higher proportions of coarse sand and gravel, but lineage VI was more associated with greater depths than both lineage III and V. Lineage I, III, V, and VI have been found at elevations from 6,000 to 11,000 feet (Nehring 2007). Flow and depth seemed to be a better of an indicator of overall *T. tubifex* abundance than specific lineage composition (Winkelman et al. 2005). The factor of microhabitat preference puts another variable into the equation. Most of the previous work has not considered the sediment type and its influence on competition among the lineages.

Much of the research currently in progress is comparing lineages and trying to single out what microhabitats and parameters are optimal for each lineage. Lineage VI worms have been stocked in Colorado, but their performance is still being evaluated. As these recent studies show, there is potential in using the resistant strains to help reduce, prevent, or contain the whirling disease parasite. The more that we understand the lineages, their ecology, and their interaction with other species and each other, the better informed we will be when making worm-stocking decisions. We need to insure that we are not stocking something that will be a host for some other pathogen or parasite that will be worse than *M. cerebralis*. Given the infamous history of species transfers such as *Mysis* shrimp, cane toads, and mosquitofish, we need to be cautious. Nonetheless, stocking of resistant strains could be a useful tool for aquatics managers to help combat the impact of whirling disease.

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At the Fisheries Experiment Station we have imported some lineage I and VI worms from Colorado. We will be using these for some upcoming experiments and to potentially stock into the Colorado River drainage where we have *M. cerebralis* present.

Matt Bartley and Eric Wagner

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Lethal Effects of Hydrogen Peroxide on New Zealand Mud Snails

Introduction

New Zealand mud snails (*Potamopyrgus antipodarum*; syn. *Hydrobia jenkinsi*; NZMS) are an aquatic nuisance species that were first reported in the western United States in 1987 (Bowler 1990). As of 2007, NZMS were reported in all western states except New Mexico (Montana State University 2007). Ecologically, NZMS are considered problematic because their densities in the wild can reach 800,000 individuals / m^2 (Dorgelo 1987). They can be responsible for consuming up to 75% of the gross primary production (Hall et al. 2003) and may account for up to 92% of the invertebrate production in a stream (Hall et al. 2006). As a result, they may have a significant influence on fish populations. In November 2007, NZMS were reported in the Utah Division of Wildlife Resources (UDWR) Loa State Fish Hatchery (Wayne County, Utah). In this article, we report the results of research that the UDWR Fisheries Experiment Station has conducted to determine the efficacy of using hydrogen peroxide to eliminate NZMS from the Loa Hatchery.

Methods

To determine the lethal effect of hydrogen peroxide on NZMS, two experiments were conducted. In the first experiment, replicate groups of 25 NZMS were placed into 400 mL beakers and were exposed to a 30,000 ppm solution of hydrogen peroxide for either 1, 10, 15, 25, 50, or 100 minutes (four replicate for each duration, eight replicate groups of 10 and 100 minutes). As a control, eight replicate groups of 25 NZMS were held in water for 10 minutes. After exposure, the solution was poured out of the beaker, NZMS were rinsed twice with hatchery water, and the beaker was re-filled with water and held for 24h to assess survival. In the second experiment, groups of 25 NZMS were placed into 400 mL beakers and were exposed to 100, 250, 500, 1,000, 1,500, 10,000, and 30,000 ppm hydrogen peroxide solutions for 15 minutes (four replicates per concentration with four control replicates where NZMS were held in water). Similar to the first experiment, at the end of the exposure period, the solution was poured out of the beaker, NZMS were rinsed, and held in hatchery water for 24h to assess survival. For both experiments, the percentage of NZMS that survived is reported. Also, LD-50 and LD-90 values were computed using the PROC Probit procedure in SAS and reported.

Results and Discussion

Overall, 3 of 100 (97%) of NZMS exposed to a 30,000 ppm solution for 1 minute and 1 of 200 (99.5%) of snails exposed to the same solution for 10 minutes survived (Table 1). We observed 100% mortality of NZMS that

Table 1: Percent survival of New Zealand mud snails exposed to a 30,000 ppm solution of hydrogen peroxide for various durations (Experiment 1), and percent survival of New Zealand mud snails exposed to various concentrations of hydrogen peroxide for 15 minutes (Experiment 2).

Experiment 1			Experiment 2		
Exposure Time (min)	% Survival	Standard Error (%)	Concentration (ppm)	% Survival	Standard Error (%)
1	3.0	1.9	100	100.0	0.0
10	0.5	0.5	250	99.0	1.0
15	0.0	0.0	500	100.0	0.0
25	0.0	0.0	1,000	98.0	2.0
50	0.0	0.0	1,500	85.0	10.0
100	0.0	0.0	10,000	0.0	0.0
Control	100	0	30,000	0.0	0.0
			Control	100.0	0.0

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were exposed to the 30,000 ppm solution for all other durations. We estimate that the LD-50 and LD-90 values for NZMS exposed to a 30,000 ppm solution are under 10 seconds. Therefore, a 30,000 ppm solution of hydrogen peroxide is very lethal to NZMS, however, longer (>10 minute) exposures are required to kill 100% of NZMS.

When hydrogen peroxide was tested with 15 minute exposures at various concentrations, we only observed significant NZMS mortality at concentrations greater than 10,000 ppm (Table 1). We estimate that the LD-50 value for a 15-minute exposure is 2660 ppm and the LD-90 value is 5520 ppm.

These results demonstrate that hydrogen peroxide can potentially be used at Loa or other hatcheries to control NZMS, or to prevent snail establishment. Because previous studies have shown that hydrogen peroxide is an effective egg disinfectant (Arndt et al. 2001), it is possible that it can be used during wild egg take operations to kill inadvertently collected snails. In addition, we observed that NZMS treated with high concentrations (>10,000 ppm) of hydrogen peroxide float. This indicates that hydrogen peroxide can possibly be used to remove snails from difficult to access areas such as the inside of pipes or sunshine systems. It is encouraging that hydrogen peroxide can be used to kill or remove NZMS. Of the chemicals that have been shown to be lethal to NZMS, hydrogen peroxide is the only chemical that is approved by the FDA for use at hatchery facilities.

Randy Oplinger

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