# Fish Health News You Can Use

**Brought to you by the Pacific Region Fish Health Program**

**April 2020 edition**

## This Issue



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## In the Next Issue

- Digenes (grubs)
- Disinfectants
- PRFHP updates
- Mystery parasite of the day

## Intro to This Issue

In this issue of the Fish Health News we are talking about fish diseases and climate change. While it seems likely that climate change will cause changes in the prevalence and behavior of many diseases, we are especially concerned about a group known as the Myxozoa.

Myxozoans are a group of microscopic aquatic parasites that affect a wide range of fish species. They are in the phylum of Cnidaria, meaning that they are distantly related to corals, jellyfish, and sea anemones.

Figure 1: Jellyfish, a more typical cnidarian. *[photo by Luis Miguel Bugallo Sánchez (Lmbuga) / CC BY-SA (https://creativecommons.org/licenses/by-sa/4.0)]*

# Climate Change and Fish Diseases

In the wild, fish disease organisms (pathogens) are a normal part of ecosystems and they play an important part in maintaining the balance of nature. Fish and their local pathogens co-evolve to insure that neither the fish nor the pathogen is wiped out. It's a cold war détente kind of arrangement that limits hostilities to the advantage of both sides.

*Figure 2; Nixon and Brezhnev, classic Détente.*

Diseases tend to be mild because if the disease kills all of the fish then it has no way to propagate and persist. That's bad for the fish and for the disease organism. Likewise, the energy expenditure required for a fish population to completely wipe out a disease isn't worth the cost as long as the disease only kills a few fish or produces just an occasional mild disease outbreak. Both the fish and the pathogen do best when both sides limit the harm that they cause to the other side. As long as this balance is maintained, big disease problems are rare and epidemics only occur when the system is disturbed by changes that occur too quickly for the fish and pathogen to adapt.

Unfortunately, climate change is happening quickly and there are many ways that it is disturbing the balance of ecosystems. This includes changes in the delicate relationship between fish and their diseases.

*Figure 32: Fish and viruses in balance*

1. Changes in temperatures and hydrology are changing the ranges and migratory pathways of many animals. Tropical fish now seen on the coasts of Oregon and Washington are an excellent example. In their new homes, these newly-arrived fish species are exposed to pathogens that they haven't seen before. They may also bring with them diseases from their former homes that the Oregon and Washington fish have never seen.

*Figure 3: Mola mola (the Ocean Sunfish) a tropical fish now sometimes found on the Oregon coast*

2. Many parasites have non-fish hosts in their life cycles. Changes in temperatures or water flows may allow these non-fish hosts to become established in places that we've never seen them before. When that happens, the parasites that rely on these animals are able to colonize new areas and they may cause big losses in previously unexposed fish populations.
3. Fish are constantly adapting to changes in the water quality of their environments, but when the changes are major, or rapid, and especially when they push fish to their limits, fish may have to let some physiological functions go in order to support their most critical systems. Maintaining an immune system is very expensive for fish (it requires a lot of energy and protein) so fish often have to greatly reduce immune function when they are stressed.
4. Many bacteria that can infect fish can also live freely in the environment. Increasing water temperatures may increase the numbers of some of these bacteria to a point that they are able to easily overcome the fishes' defenses.

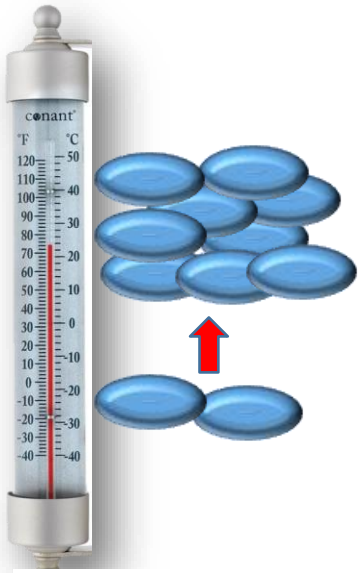



*Figure 4: Bacteria grow faster at higher temperatures*

So what do we worry about with fish diseases and climate change in the Pacific NW? The primary impacts of climate change in our Region will be warmer temperatures and more precipitation coming as rain instead of snow. This means higher summer water temperatures and lower flows. Based on our experience with recent periods of warm dry weather, and on what we see in watersheds to our south, we expect that the following things might happen:

More losses from bacterial diseases: Many important fish disease bacteria can also live freely in the wild. Warmer temperatures favor the rapid propagation of these bacteria both in the environment and inside fish. When increased pathogen numbers are coupled with poor immune function in warmer water, infections and disease outbreaks are more likely to occur. A striking example was fish losses from columnaris in the Columbia River in the summer of 2015. Other important bacterial diseases of salmon, including furunculosis and enteric redmouth, are more likely to happen at warmer temperatures.

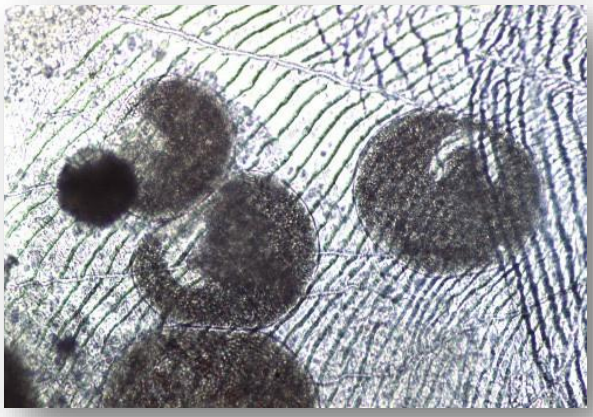


Figure 5: Ich parasites on a fish scale

Ich: As temperatures increase, Ich parasites are able to grow faster and multiply more quickly. In addition, salmon immunity declines rapidly as temperatures pass 65 F. At 55 F, Ich is rarely a problem in salmon. At 70 F, it is devastating.

The longer and warmer the summer season, the more likely Ich is to kill fish.

Myxospores: These parasites have complex life cycles that include both fish and aquatic invertebrates. Here in our region, the diseases (PKD and *C. shasta*) are widespread, but usually quite limited in their severity. However, as temperatures increase, it is possible that these invertebrate hosts will increase in number making it easier for the parasites to complete their life cycles. Coupled with warm water effects on fish immunity, there is the potential for infections by these parasites to become much more severe. We see evidence of this in the severity of *C. shasta* disease in the Klamath Basin in southern Oregon.

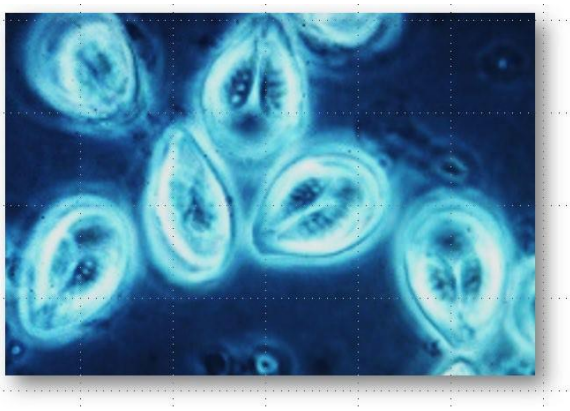


*Figure 6: Microscopic myxospore parasites*

Digenes: The digenes (black, yellow and white grubs and black spot) have very complex life cycles that involved fish, birds, and snails. Many fish species are susceptible and migratory birds spread the parasites throughout the US. The main limitation to the establishment and severity of digene infections is low or absent populations of specific snail hosts. Warmer temperatures may favor the spread of these snails while curtailing the fish's ability to mount an effective immune response. The apparently-increasing prevalence of black spot infections in fish migrating through tributaries of the southern parts of Puget Sound may be a sign of this happening.

Figure 7; Yellow grubs in a bluegill fin. These parasites have birds, fish, and snails in their life cycles.

Are there any silver linings? Higher water temperatures are also going to be a problem for some fish pathogens that are unable to propagate at higher temperatures even in fish with compromised immune systems. For example, the IHN and VHS viruses are unable to propagate at temperatures over 70 F and they persist in fish and in the environment only for short periods at these temperatures. Another example is the bacterial disease BKD which is caused by bacteria that prefer cooler temperatures. While that sounds good, we need to keep in mind that the complex interplay of the adverse effects of high temps on both the bacteria and the fish's immune systems makes the outcome hard to predict.

In conclusion: We expect that climate change in the Northwest will lead to higher water temperatures and lower summer flows that will weaken salmon immune systems and are likely to increase production of important bacterial pathogens and of Ich. We already see these impacts during periods of extreme weather. Less certain, but very troubling, is the potential for other parasites (especially *C. shasta*, PKD, and the grubs) to become much more serious problems in both wild and cultured fish.

## PKD

Proliferative kidney disease or "PKD" is caused by the myxozoan parasite *Tetracapsuloides bryosalmonae* and is an important disease of wild and cultured salmonids in North America and Europe. Proliferative kidney disease is primarily seen in the summer months when water temperatures rise. Mortalities can reach upwards of 95% and are typically highest above 54°F (12°C). The warming effect of climate change on rivers is causing PKD to be more commonly encountered in both wild and cultured fish. New cases of PKD in wild fish in Europe have been documented in recent years and the number and distribution of cases is expected to increase.

*Figure 8: Swollen kidney in a trout with PKD. Photo from Amberly Huttinger, FWS*

Proliferative Kidney Disease was first detected in the USA in 1981 at Hagerman State Fish Hatchery in Idaho in rainbow trout. The outbreak killed over 200,000 fish and resulted in the destruction of over 800,000 more. The disease has also resulted in significant fish loss at Merced Fish Hatchery in California. In 2016, an outbreak of PKD in the Yellowstone River in Montana killed thousands of mountain whitefish. The outbreak was attributed to warm summer water temperatures and low river flow that provided a "perfect storm" for the parasite to proliferate and infect fish. In order to reduce stress on infected fish and limit movement of the disease, Montana Fish, Wildlife, and Parks temporarily closed 183 miles of the Yellowstone River. This closure resulted in a significant economic loss for the area. Proliferative Kidney Disease has also caused fish losses in Washington and British Columbia but in recent years has not caused problems on any national fish hatchery in our Region.

The life cycle of *Tetracapsuloides bryosalmonae* involves two hosts: a fish and an aquatic sessile colonial invertebrate known as a bryozoan (Figure 10). Bryozoans are a diverse group of organisms with over 4,000 species found all over the world in freshwater, brackish, and saltwater environments. Colonies are made up of individual bryozoans and range from a centimeter to over a meter in length. These colonies grow on many different types of surfaces including ice. The bryozoan that is involved in the lifecycle of *Tetracapsuloides bryosalmonae* grows on the surfaces of river rocks and logs. Bryozoans infected with the parasite contain many parasitic spores that can be released into the environment and infect fish through their gills and skin. Bryozoans produce seed-like structures called statoblasts that can be spread in the environment by birds.

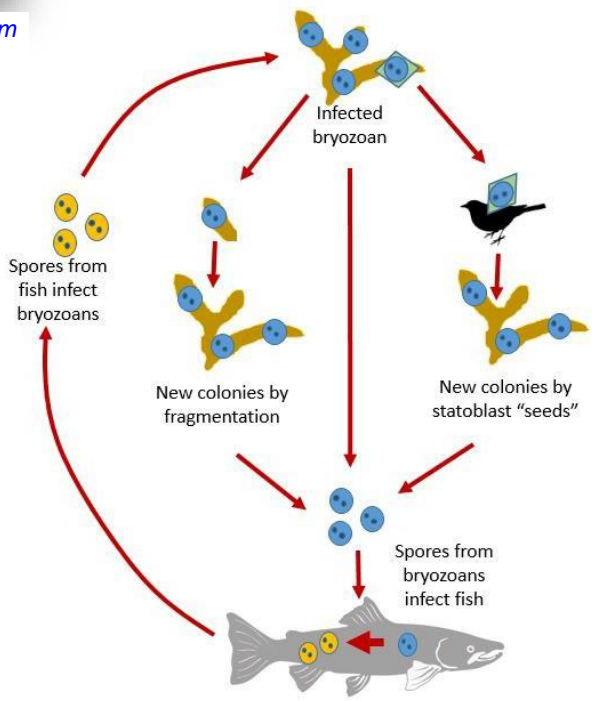



*Figure 9: The PKD life cycle.*

These statoblasts may contain *Tetracapsuloides bryosalmonae* spores. A recent study examining statoblasts showed up to 39% of the structures carried *Tetracapsuloides bryosalmonae*. Bryozoans also spread in the environment by fragmentation. Bryozoans are not present in every watershed, and nutrient availability and temperature may influence the distribution of bryozoans. Waterways in agricultural areas in Europe have been shown to have more bryozoans than less nutrient-rich waters.

*Figure 10: PKD Bryozoan Fredericella sultana Image credit: Ondrej Korábek Public Domain*

Infection with *Tetracapsuloides bryosalmonae* is normally tolerated by fish and does not always cause disease. In other words, infection with *Tetracapsuloides bryosalmonae* does not mean the fish will become sick with PKD. Fish that develop clinical PKD typically have experienced external stressors, such as warming water, that overwhelm the fish's ability to fight disease. Warm water temperatures allows replication of the parasite in the fish resulting in a significant inflammatory response. Warm water also indirectly increases likelihood of clinical PKD by causing the release of infective spores from the bryozoan host. Fish with clinical PKD have extensive kidney swelling, abdominal distention, bulging eyes, and may also develop gill lesions. Juvenile fish are most susceptible to disease. Diagnosis is made through microscopic examination.

Management of PKD in brown trout is Europe is commonly done with the development of protective immunity, a natural vaccination process. Fish that have not previously been exposed to *Tetracapsuloides bryosalmonae* are brought on station in summer as water temperatures rise. These fish develop clinical PKD but water temperatures drop before fish experience high mortality. Fish develop antibodies to the parasite and are resistant to future PKD. While this method has been effective, researchers in Europe are currently developing a *Tetracapsuloides bryosalmonae* vaccine and diagnostic tools to better control PKD in cultured fish.

Proliferative Kidney Disease is an important disease in fish and has had significant impacts on fisheries in North America and Europe. With warming rivers, we could experience the "perfect storm" of temperature and low flows seen in the Yellowstone River in Montana that lead to a PKD outbreak. Although we have not seen PKD in hatcheries in our region, climate change may produce conditions that favor the disease. Hatchery staff should continue to monitor fish and notify fish health staff of any concerns.

*Figure 11: PKD spores in bryozoan. Sudhagar et al., Pathogens 2020, 9(1), 16; CreativeCommons.org*

## Ceratonova shasta

*Ceratonova shasta*, previously known as *Ceratomyxa shasta*, is a myxosporean parasite that infects Pacific salmonids and causes a disease called ceratomyxosis or "gut rot." The parasite was first identified in Shasta County, California, but has since been detected in Washington, Oregon, Idaho, Alaska, and British Columbia. The distribution of *C. shasta* is limited to systems in which the intermediate host (an aquatic worm) and the definitive host (salmonids) overlap. *C. shasta* can infect all life stages of coho (*Onchoryhnchus kisutch*), Chinook (*O. tshwytscha*), pink (*O. gorbuscha*), chum (*O. keta*), sockeye (*O. nerka*), and steelhead and rainbow trout (*O. mykiss*), as well as cutthroat trout (*O. clarkii*). There are currently three distinct genetic types of *C. shasta* which are adapted to infect specific salmon species.

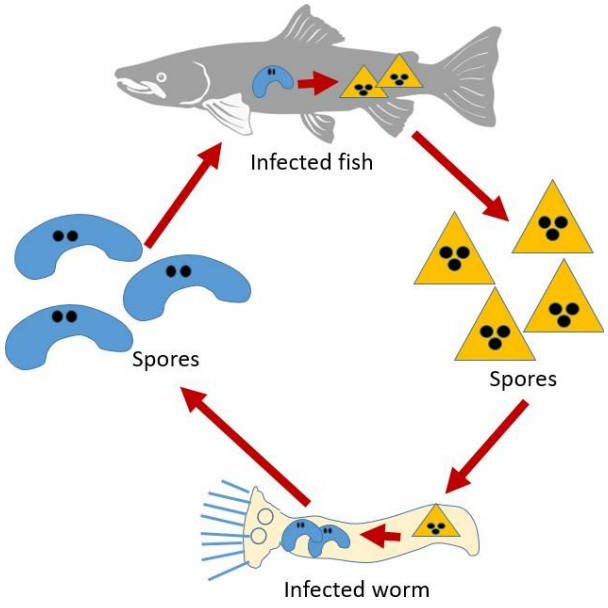



*Figure 12: The C shasta life cycle*

Like other myxosporeans, the lifecycle of *C. shasta* is complex and requires an invertebrate host and a fish host. When an infected fish dies it sheds *C. shasta* spores into the water, these spores are not infective to fish and cannot spread disease alone. The spores must first infect *Manuyunkia speciosa*, a small, freshwater worm. Within the worm hosts' epidermis *C. shasta* spores transform. Spores within this next phase of the lifecycle are infective to salmon and can infect fish via the gills directly from the water column. Neither horizontal (fish to fish) nor vertical (parent to offspring) transmission has been observed for *C. shasta*, indicating that *M. speciosa* is necessary for transmission of infection. Infection rate and severity is amplified in warmer water temperatures (≥10°C), making the disease of particular concern with regard to climate change.

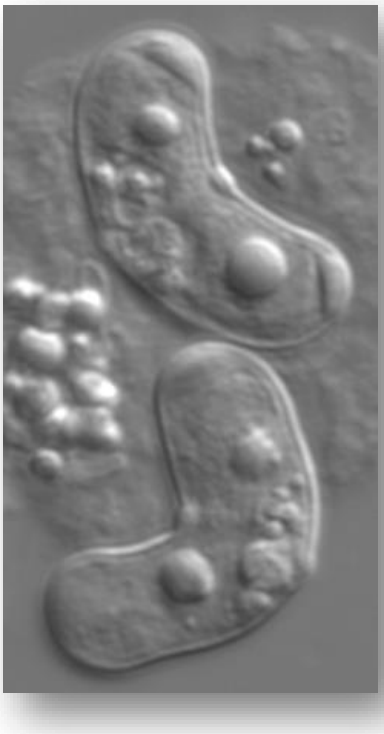


*Figure 13: C shasta spores between worm and fish. Picture by Steve Atkinson*

*C. shasta* causes hemorrhagic necrosis of the intestines of infected salmon, hence the name "gut rot." After proliferating in the intestinal wall of the fish host *C. shasta* can spread to and develop in the kidneys, liver, pyloric cecae, gall

bladder, gonads, heart, gills, muscle tissue, and within the eye. Clinical signs vary between species and ages of fish, but generally include weight loss/emaciation, lethargy, skin darkening (especially in steelhead and rainbow trout), abdominal distention, exophthalmia (pop eye), and protrusion of the vent. On necropsy, infected fish may have swollen, discolored, or hemorrhagic intestinal tracts, ascites (free fluid in the coelom), and/or thick, white material in the stomach and pyloric cecae. Chinook, in particular, may developing perforating lesions throughout the intestinal tract. Abscesses may be apparent in the liver, kidney, muscle (especially coho), and spleen in affected adults. These clinical signs are very similar to bacterial kidney disease (BKD) in salmon, so it's important to perform diagnostics to differentiate the two infections. The prognosis for clinical ceratomyxosis is poor and mortality is usually high. Resistant fish that survive infection can become subclinical carriers of *C. shasta* and shed myxospores in the water.

*Figure15: The worm host of C shasta. Picture curtesy of Mark Atkinson*

Preliminary diagnosis of ceratomyxosis is made by visualizing *C. shasta* organisms on wet mounts of free abdominal fluid, intestinal wall, or lesions within other organs or by visualizing the parasite on impression smears of affected organs. Confirmation of suspected *C. shasta* is commonly made by direct visualization of the mature parasite spores in wet mounts or histological sections of tissues.

*Figure 146: Salmon with C shasta. Lesions are hard to see, but the kidney is swollen. Picture by Steve Atkinson*

Blood tests and molecular tests are also available as molecular methods of diagnosing *C. shasta*. Preventing exposure to the infective parasite is the only way to control ceratomyxosis, there is currently no effective treatment for or vaccination protocol against infection. Removal of the worm host and infected salmon carcasses from water systems have been investigated as means to control infection, but neither method has been found to reduce infection burden enough to be viable. Cooler water temperatures (ie: chillers in adult holding) have been successful in slowing the progression of clinical disease in infected fish, but has not been successful in clearing the infection. *C. shasta* infection may make salmon filets unsightly but the parasite poses no known risk to human health and does not appear to infect other species of freshwater fish.

During the 2014 Chinook spawning season there was increased mortality in adults in holding ponds and a marked decline in returning adults to Warm Springs National Fish

Hatchery (WSNFH) in Warm Springs, Oregon; adults that did return had clinical signs of ceratomyxosis and significant *C. shasta* burdens. This was particularly concerning as this was the first time that ceratomyxosis was encountered on a large scale in the Deschutes River Basin. A subsequent review of monitoring data revealed that less than half of the juvenile Chinook that were released from WSNFH were surviving to pass over Bonneville Dam after release; a fish health investigation found that *C. shasta* was the likely culprit in the alarming increase in juvenile mortality as well. A 3-year, multi-agency investigation involving USFWS, the Confederated Tribes of the Warm Springs Reservation, Oregon Department of Fish and Wildlife, Pacific Gas and Electric, and Oregon State University found levels of *C. shasta* throughout the Deschutes Basin (with the exception of Warm Springs River) which greatly exceeded the threshold known to be lethal to salmonids.

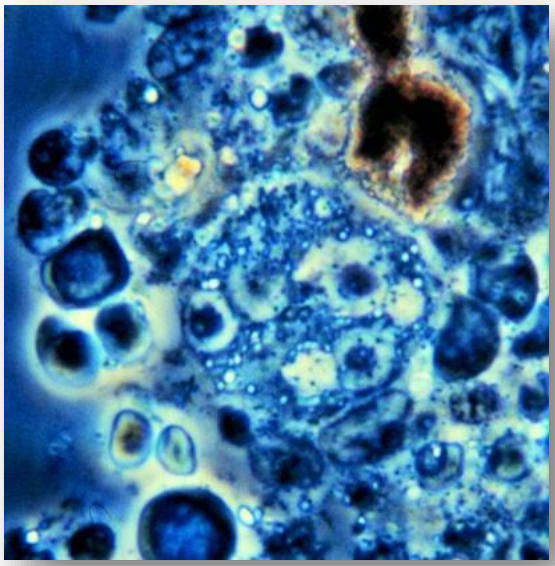


*Figure 16: C. shasta (center) developing in a fish. Picture courtesy of Jerri Bartholomew.*

The increase in parasite density in the Deschutes basin was likely multifactorial. In 2009 a Selective Water Withdrawal tower was installed above Round Butte Dam to discharge water from Lake Billy Chinook into the lower Deschutes River. The water coming from Lake Billy Chinook was not only warmer but also more nutrient-rich than the water in the Deschutes River; the influx of warm, turbid

*Figure 158: Construction sequence of the new selective water withdrawal and fish collection facility at Round Butte Dam, Lake Billy Chinook, Deschutes River, Madras, Oregon. Photo by Scott Carlon, NOAA Fisheries*

water altered downstream water quality and temperature, which changed the community of aquatic invertebrates in the lower Deschutes, most notably by increasing the prevalence of *Manayunkia speciosa*. (Challenges to water quality such as climate change and human activities upstream caused a downstream change in water temperature, turbidity, and quality, making the Deschutes more hospitable to aquatic worms.) The increase in worm population coupled with higher water temperatures exponentially increased the density of infectious *C. shasta* spores in the water column. Furthermore, the release of infectious spores from *M. speciosa* worms synchronized with salmon migration times, meaning that juvenile fish were heavily

impacted on their outmigration to the ocean while adults were being exposed to extraordinarily high numbers of parasites on their migration back to WSNFH. Warmer-than-normal water temperatures further compounded the mortality event in adults that had returned to holding on station by accelerating the induction and progression of clinical ceratomyxosis in infected fish. Investigations into the Deschutes River *C. shasta* outbreak are ongoing and will provide important insight to managing this parasite in the Pacific Northwest in the future.

While *C. shasta* is unlikely to become a fish health problem on station because of the absence of the requisite worm host it is an important disease to keep in mind for out-migrating smolts and returning adults. Particularly in situations where brood stock may be held on station for longer periods (weeks), complications associated with ceratomyxosis should be considered in areas where the disease exists; management plans including keeping holding pond water as cool as possible, reducing stocking density, and reducing other fish stressors may be helpful in reducing mortality associated with *C. shasta*. Because of its initial similarity to BKD a thorough work-up, including definitive diagnosis, is indicated to guide subsequent management decisions. The appearance of *C. shasta* in areas where it was not previously detected may signal important shifts in the local ecosystem and new outbreaks may warrant in-depth investigations.

## Myxofactoids

The involvement of worms and bryozoans in the life cycle of *C. shasta* and PKD was not clear until the 1990s. Before that, the fish and invertebrate portions of their life cycles were given different unrelated names.

Myxozoan spores come in all kinds of fantastic shapes designed to keep spores suspended in the water column in an optimal way to intersect a fish.

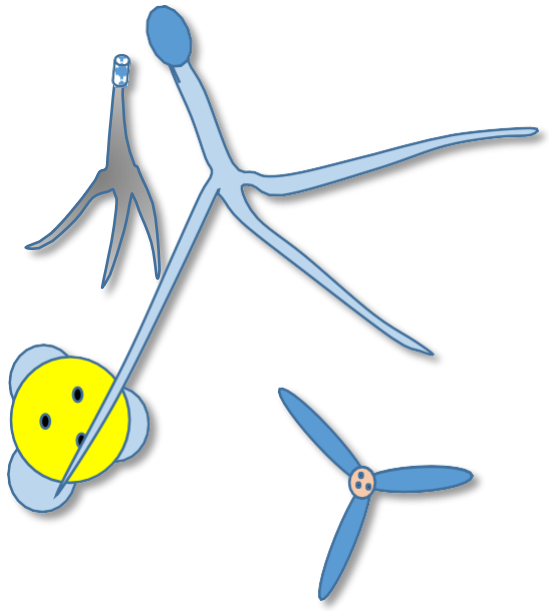


*Figure 17: Assorted myxozoan spores*

The myxozoan Kudoa infects salmon muscle and remains fairly innocuous until the fish dies. After death the spores trigger protein digesting enzymes that break down the muscle and release the spores. The same thing happens to fillets from infected fish that are caught by sport and recreational fishers.

*Figure 20: The "Magnificent bryozoan" makes gelatinous blobs the size of beach balls and is featured in a creepy FWS video on You-tube titled "Attack of the Blobs". https://www.youtube.com/watch?v=qbllejaU-lw*

Myxozoans like *C. shasta* and *T. bryosalmonae* are most closely related to a jellyfish family called the Cnidaria. The myxozoans use the same stinging cells that the Cnidaria use, but they use them to attach the spore to the fish's intestinal wall to make it easy for the parasite to invade the fish.

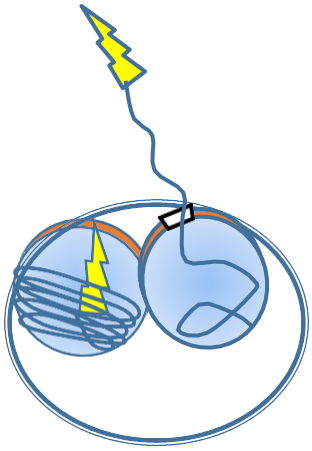


*Figure 18Figure 19; A jellyfish nematocyst. In Myxozoans, the harpoon springs out to fasten the spore to the fish.*

A myxozoan known and Henneguya invades the gills of catfish. In channel catfish the immune response is so strong that the catfish destroys its own gills.

*Figure 22: A gap in a catfish gill filament caused by an excessive immune reaction to a myxozoan parasite.*

Other myxozoans are present in trout and salmon in the northwest. The most famous is the parasite that causes whirling disease. It develops in the cartilage of trout and causes deformities.

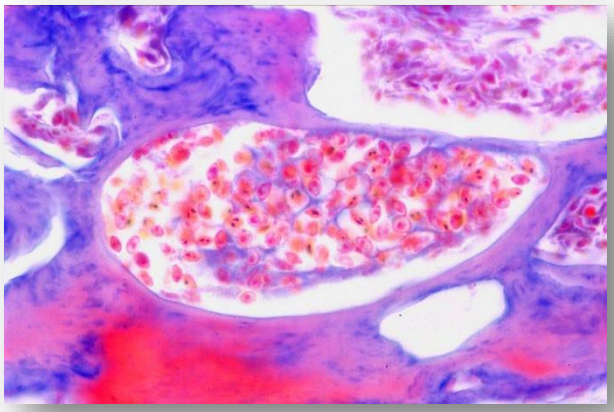


*Figure 19: Whirling disease spores (orange, center) in the bone (purple) of a rainbow trout.*

## PRFHP Update

PRFHP Veterinarian Dr. Trista Becker (based at Leavenworth NFH) has accepted a new position as the Project Leader of the FWS Fish Health Center in New Mexico and will be leaving us in April. Tim Bundy (also at Leavenworth) will be carrying on the fish health work with support from other PRFHP veterinarians and staff. We will hire a new veterinarian as soon as possible. Trista has been awesome. We'll miss her.

The rest of the PRFHP staff is working hard to maintain fish health services while dealing with the complexities of the coronavirus pandemic. We had not expected to put our expertise in epidemiology and biosecurity to work in this way, but we are determined to keep both fish and people healthy. To our fish hatchery

"clients" thank you for working with us to find new and creative ways to get our work done.

## Thiamine Update

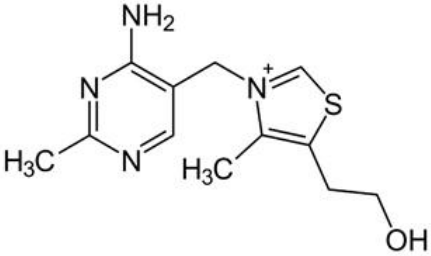



*Figure24: Thiamine. (Public domain, USG)*

Thiamine is suddenly a hot topic on hatcheries in the Northwest. Thiamine is a B vitamin required for carbohydrate metabolism and proper functioning of the nervous and cardiovascular systems. It is an essential nutrient for humans and fish. In humans, a thiamine deficiency causes a disease called beriberi. In fish, a deficiency of this vitamin can cause low reproductive success in adults, early mortality in swim-up fry, and immunosuppression. Requirements for Coho and Chinook salmon are 10-15 mg/kg feed. In the wild, thiamine is produced by bacteria, algae, and plants and gets to salmon through the food chain.

In the Great Lakes researchers documented thiamine deficiency in Lake Trout and some salmon species back in 1968. The main cause of the deficiency was the forage fish consumed. The alewives and rainbow smelt eaten by the top predators were an exotic invasive species having a high level of an enzyme called thiaminase. This enzyme breaks down the thiamine causing the predator to be thiamine deficient. Afflicted adults may have trouble reaching the spawning grounds, their spawning may not result in viable eggs, and severe deficiencies can kill them outright. Thiamine-deficient adult females also pass the deficiency to their eggs causing the fry to hatch without the thiamine reserve that they need to swim normally and to begin feeding. In one study where Lake Trout females were fed only the exotic alewives, the Lake Trout eggs contained 2.5-nmol thiamine/g and 20% of the fry died. When Lake Trout were fed bloaters (a native fish species that does not contain thiaminase), their eggs contained 12-nmol thiamine/g and no fry died.

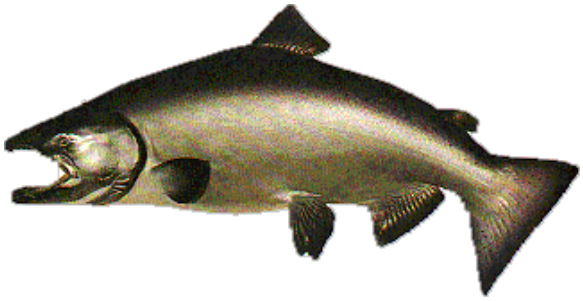
This year fry from eggs of the fall and spring Chinook salmon returning to Federal and state hatcheries in California, and adult steelhead returning to Oregon hatcheries, have exhibited signs of thiamine deficiency (fry laying in their sides, failure to feed, and mortality). After a thiamine bath, afflicted fry were up and swimming, schooling, and feeding within an hour or two.

Why now? One cause could be the fact that the Pacific forage fish stock has changed. The sardine stocks have failed and anchovy numbers have increased. The thiamine deficiency in Chinook may have been caused by fish feeding heavily on anchovies before going upstream to spawn. However, the cause may be more complicated. Sampling plankton communities off the coast of California, scientists have found lower amounts of B vitamins, including thiamine. One theory is that warming waters have caused marine bacteria to consume more of the vitamins that they produce. This makes for lower levels of vitamins available for phytoplankton and zooplankton to take up and pass up the food chain to forage fish and eventually salmon.

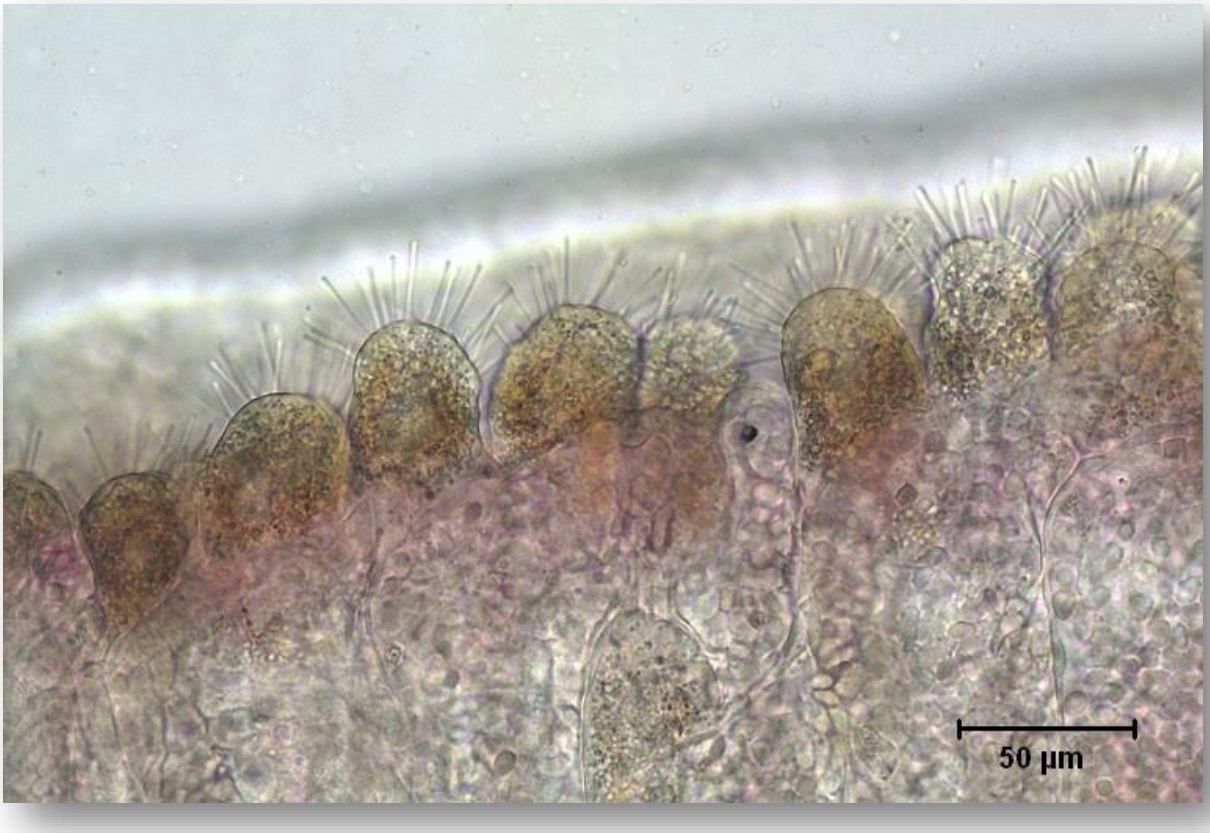
We can combat thiamine deficiencies in hatchery fish by using thiamine baths as needed, but populations spawning in the wild are a much more complex issue. To quote one researcher, "Thiamine supplements are not a realistic solution to ameliorate whole ecosystems."( L. Balk in *Deadly Deficiency in the*

*Heart of an Environmental Mystery,* 2018 PNAS).

We are working now on a potential thiamine deficiency at Quinault NFH. If you are seeing any problems in fry, be sure to contact your fish health person for an accurate diagnosis. The PRFHP staff is working closely with Ann Gannam (Fish Nutritionist at the Abernathy FTC) to evaluate and treat any problems that arise. If you have any questions about thiamine, call Ann or the PRFHP staff.



# Mystery Parasite of the Day




**For the answer, click Here.**