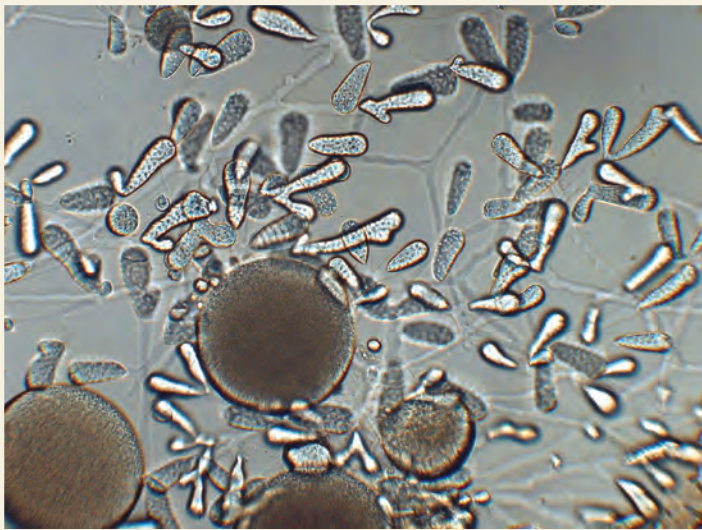# COMMON DISEASES OF WILD AND CULTURED FISHES IN ALASKA

Theodore Meyers, Tamara Burton, Collette Bentz and Norman Starkey

About This Booklet

This booklet is a product of the *Ichthyophonus* Diagnostics, Educational and Outreach Program which was initiated and funded by the Yukon River Panel's Restoration and Enhancement fund and facilitated by the Yukon River Drainage Fisheries Association in conjunction with the Alaska Department of Fish and Game. The original impetus driving the production of this booklet was from a concern that Yukon River fishers were discarding Canadian-origin Chinook salmon believed to be infected by *Ichthyophonus*. It was decided to develop an educational program that included the creation of a booklet containing photographs and descriptions of frequently encountered parasites within Yukon River fish.

This booklet is to serve as a brief illustrated guide that lists many of the common parasitic, infectious, and noninfectious diseases of wild and cultured fish encountered in Alaska. The content is directed towards lay users, as well as fish culturists at aquaculture facilities and field biologists and is not a comprehensive treatise nor should it be considered a scientific document. Interested users of this guide are directed to the listed fish disease references for additional information.

Information contained within this booklet is published from the laboratory records of the Alaska Department of Fish and Game, Fish Pathology Section that has regulatory oversight of finfish health in the State of Alaska. This second printing includes one added disease and some new photographs.

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Aquareovirus

I. Causative Agent and Disease

Aquareovirus is a recent new genus in the virus family Reoviridae. These icosahedral (60-80 nm) double-stranded RNA viruses (over 50) have been isolated from a variety of marine and freshwater aquatic animals worldwide including finfish, and bivalve molluscs. Genetic analyses have identified 7 different genotypes of aquareoviruses. Most of these viruses produce self-limiting infections of low pathogenicity and are not associated with extensive disease or mortality. Exceptions include isolates from 7 fish species that have been associated with fish mortality, most notably the grass carp reovirus. The viral agents are most often isolated from asymptomatic adult carrier fish during routine screening examinations.

II. Host Species

In the Pacific Northwest, specifically Washington, Oregon and California, adult Chinook salmon appear to be the most frequent species infected with aquareoviruses. The virus has also been isolated from adult coho and chum salmon and steelhead. Rainbow trout have been experimentally infected with the virus resulting in mild hepatitis with no overt disease or mortality. In Alaska, aquareoviruses have been isolated from Chinook salmon and geoduck clams.

III. Clinical Signs

Fish infected with aquareoviruses generally do not exhibit obvious clinical signs of disease. Viral replication can produce focal necrotic lesions in the livers of rainbow trout, chum salmon and bluegill fry. Exceptions to the relative non-pathogenicity of these viruses are reported for at least 7 species of fish where the viruses are associated with epizootic fish mortality. Most notably is the grass carp reovirus that produces severe hemorrhaging in fingerlings and yearlings resulting in up to 80% mortality.

IV. Transmission

Transmission is horizontal via water or from fish to fish. Isolates from bivalve mollusks likely represent virus that has been shed into the water column from a fish host that has been bioaccumulated into shellfish tissues by filter feeding.

V. Diagnosis

Detection of aquareoviruses is done by isolating the virus in cultures of susceptible fish cell lines that have been inoculated with infected tissue. The virus causes a unique cytopathic effect (CPE) characterized by focal areas of cellular fusion (syncytia) and cytoplasmic destruction creating a vacuolated or foamy appearance. Presumptive identifications are made based on the typical CPE and confirmed serologically, by electron microscopy or by polymerase chain reaction (PCR).

VI. Prognosis for Host

The prognosis for the fish host is good in the majority of cases where the virus is not a primary pathogen. There are no corrective therapies for viral infections in fish except avoidance.

VII. Human Health Significance

There are no human health concerns associated with aquareoviruses.
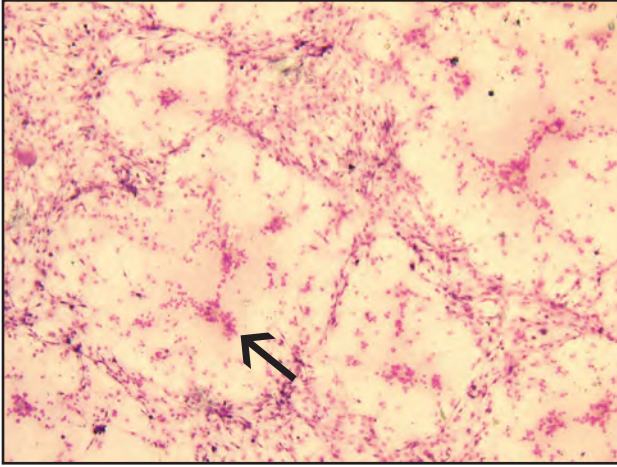
Syncytial cell CPE (arrow) of Aquareovirus in bluegill fry cells

Negative stain of Aquareovirus particles (arrow)

Erythrocytic Inclusion Body Syndrome (EIBS)

I. Causative Agent and Disease

Erythrocytic inclusion body syndrome (EIBS) is caused by an unclassified icosahedral virus (70-80 nm) that infects erythrocytes of several salmonid fishes. Typically, EIBS presents with single or multiple pale, basophilic, cytoplasmic inclusions (0.4-1.6 um) in the cytoplasm of erythrocytes in stained peripheral blood smears. Affected fish may be asymptomatic, but more often have varying degrees of anemia and secondary bacterial and fungal infections. In severe cases of uncomplicated anemia cumulative fish mortality over 20% has been reported with hematocrits less than 20%.

II. Host Species

EIBS has been found in Chinook, coho and Atlantic salmon in the Pacific Northwest, Japan, Norway and the British Isles. Other salmonid species showing variable susceptibilities by experimental injection with infected blood homogenates include cutthroat trout, masou salmon and chum salmon.

III. Clinical Signs

Fish are lethargic, anorexic and anemic with chronic mortality often associated with secondary infections by other pathogens. Five stages of EIBS have been described: pre-inclusion, inclusion body formation, cell lysis with low hematocrits, recovery with increasing hematocrits and full recovery.

IV. Transmission

The disease can be transmitted horizontally while surviving fish generally recover and develop an acquired immunity against reinfection that is transferable by passive immunization.

V. Diagnosis

Isolation and replication of the virus in available fish cell lines has been unsuccessful. Thus, diagnosis is by observation of the small pale blue inclusion bodies in the cytoplasm of infected erythrocytes with confirmation by transmission electron microscopy (TEM). The virus is found free in the cytoplasm or more commonly occurs in membrane-bound cytoplasmic inclusion bodies within erythrocytes.

VI. Prognosis for Host

Severe fish losses caused directly by EIBS are rare. However, fish become weakened from the anemia and mortality from other associated environmental stressors or secondary pathogens can be significant. The disease generally is self-limiting with recovery and immunity in survivors.

VII. Human Health Significance

There are no human health concerns with the EIBS virus.
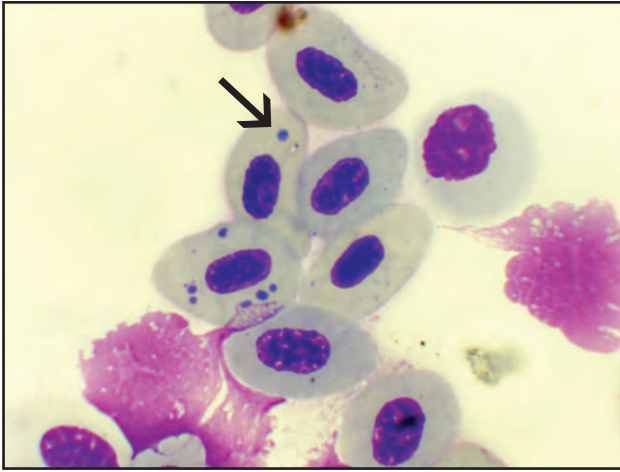
Erythrocytes of Chinook salmon with small basophilic cytoplasmic inclusion bodies (arrow) typical of EIBS
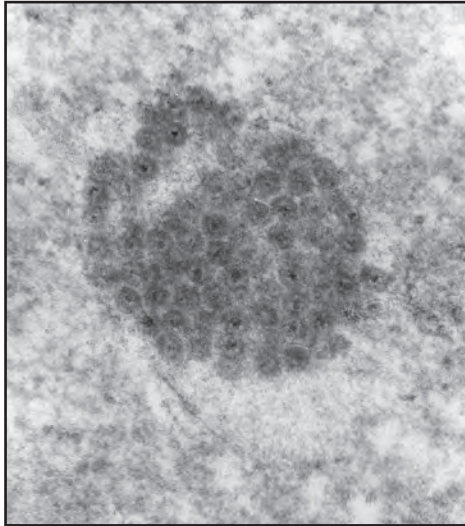
Ultrastructural section of inclusion body in erythrocyte composed of virus particles using transmission electron microscopy (TEM)

Erythrocytic Necrosis Virus (VENV)

I. Causative Agent and Disease

Erythrocytic necrosis virus, also called viral erythrocytic necrosis virus (VENV) is caused by several similar iridoviruses having double stranded DNA and an icosahedral shape ranging in size from 130-350 nm. The viruses infect erythrocytes causing a hemolytic disease often resulting in anemia and secondary infections by other pathogens.

II. Host Species

The virus probably has several different representative strains present worldwide in the marine environment infecting a large variety of anadromous and marine fish species. In Alaska, VENV has been detected in Pacific herring from several locations but has not yet been observed in salmonids. Results from experimental infections and occurrence of epizootics in young-of-the-year Pacific herring indicate that juveniles are more susceptible than older fish of the same species.

III. Clinical Signs

Adult herring generally show no clinical signs of disease. In juvenile Pacific herring, fish are anemic exhibiting almost white gills and pale visceral organs. Livers may be green in color due to bilirubin breakdown. Hemotocrits may be as low as 2 to 10%, erythrocytes are fragile causing hemolysis of blood samples, and immature erythrocytes predominate in peripheral blood. High mortality with dead fish washing up on the shoreline accompanied by extensive congregations of predator birds may occur in areas where juvenile herring are weakened by the disease.

IV. Transmission

Transmission of this virus in the marine environment is likely horizontal from fish to fish based on experimental studies where the virus has been transmitted by waterborne exposure. Adult carrier fish of susceptible species are probably the reservoirs of virus that is transmitted to juvenile fish. Anadromous fish likely become infected during the marine segment of their life cycle.

V. Diagnosis

Diagnosis is made with blood smears and/or transmission electron microscopy (TEM). Characteristic eosinophilic inclusion bodies are present in the cytoplasm of erythrocytes when stained with Giemsa or Wright stains. Impression smears of kidney hematopoietic tissue can be used if blood is unavailable. The virus is confirmed by the observation of iridovirus particles associated with inclusion bodies using TEM. VEN viruses have not been successfully cultured in the laboratory for lack of a method to cultivate erythroid cells.

VI. Prognosis for Host

The virus in Alaskan juvenile herring caused one of the first natural epizootics to be reported associated with mass fish mortality. Chronic to subacute mortality in juvenile Pacific herring can also occur.

VII. Human Health Significance

No human health concerns are associated with VEN virus.

Anemic Pacific herring with very pale gills and green livers commonly seen in VEN
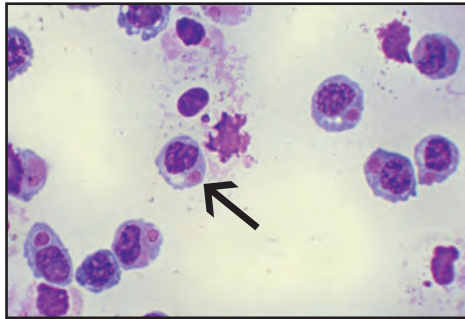
Erythrocytes of Pacific herring with large eosinophilic cytoplasmic inclusion bodies (arrow), some surrounded by pink lattice composed of virus particles
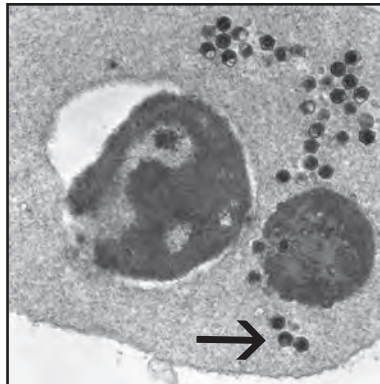
Ultrastructural section of erythrocyte showing large virus particles (arrow) comprising the lattice surrounding inclusion bodies in stained smears, TEM

Infectious Hematopoietic Necrosis Virus (IHNV)

I. Causative Agent and Disease

Infectious hematopoietic necrosis virus (IHNV) is a bullet-shaped novirhabdovirus that is enzootic to the North American Pacific Northwest, Italy, France and Germany. The virus was inadvertently introduced into Japan, Taiwan and other areas of the US (Snake River Valley, Idaho) where it has become established. IHNV can infect many salmonid species and has had a severe economic impact on intensively cultured salmon and trout. IHNV in Alaska has been limited primarily to sockeye salmon and rarely Chinook and chum salmon when they are exposed to water supplies containing infected sockeye salmon. Avoidance of IHNV and culture of sockeye salmon in Alaska has been successful through rigorous use of the Department of Fish and Game sockeye salmon culture policy. The disease, infectious hematopoietic necrosis (IHN), is an acute, systemic infection affecting the kidney tissues and other visceral organs causing extensive mortality in hatchery reared sockeye salmon juveniles as well as in wild stocks of outmigrating sockeye salmon smolts.

II. Host Species

Fish species susceptible to infection and disease by IHNV include sockeye, Chinook, chum, amago, yamame and Atlantic salmon, cutthroat trout and rainbow/steelhead trout. Brook and brown trout are experimentally susceptible to infection and mortality while lake trout are intermediately so. Arctic char and grayling are resistant while coho salmon are also resistant but can carry the virus when in the presence of other susceptible virus-infected fish species. Mortality is highest in young fish and resistance to infection and disease increases with age.

III. Clinical Signs

Infected fish may exhibit lethargy, whirling behavior, cranial swelling, abdominal swelling, exophthalmia, anemia and darkened body coloration, hemorrhaging of musculature and base of fins, fecal casts, pre-emergence in sac-fry, pale liver, spleen and kidney, stomach and intestine filled with milky or watery fluid and petechial hemorrhaging of mesenteries or visceral tissues.

IV. Transmission

Horizontal transmission through water via feces or sex products or carcass degradation is the most common route of infection. Virus occurs commonly in ovarian fluids and on the surface of eggs. Rarely, vertical transmission can occur within eggs (internal) and possibly with adhesion of virus particles to sperm during fertilization. Incubation and course of the disease is reported to be strongly influenced by water temperature in the Lower 48. Optimum temperature is 10°C-12°C but losses due to IHNV have been reported above 15°C. Mortalities occur within 4-6 days post-exposure with peak mortality occurring 8-14 days post-exposure. In Alaska, the disease can cause up to 100% mortality in sockeye salmon at water temperatures as low as 1-2°C but exponential mortality may take longer to occur at these lower temperatures. No natural reservoirs of IHNV have been confirmed other than those susceptible fish species that can become carriers of the virus. However, transient detections of IHNV have been reported in organic sediments, invertebrates, and some forage species of marine fish when associated with ongoing or recent IHNV epizootics in a susceptible salmonid species.

V. Diagnosis

Susceptible fish cell cultures are inoculated with kidney and spleen tissues (whole fry if small) or ovarian fluids from fish suspected of having IHNV. Presumptive diagnosis is made when characteristic cytopathic effect (CPE) occurs in cell monolayers from virus infection and lysis of the cells. Virus can be definitively identified with PCR.

VI. Prognosis for Host

Prognosis for infected fish is poor. Survivors of epizootics and non-lethal infections probably become lifelong carriers of the virus. There is no known therapy for fish that have been infected with IHNV. In Alaskan hatcheries, all infected lots of fish are destroyed. The occurrence of the disease is avoided through preventative measures including a virus-free water supply, rigorous disinfection, isolation of egg and fish lots and containment of diseased fish.

VII. Human Health Significance

There are no human health concerns associated with IHN virus.

Cephalic bumps on sockeye salmon fry are characteristic of IHN disease
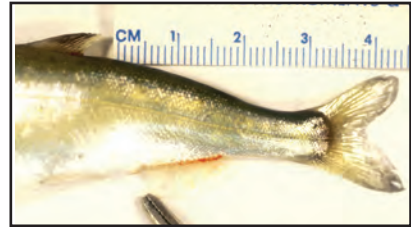
Hemorrhaging at the base of the fins sometimes observed in IHN disease
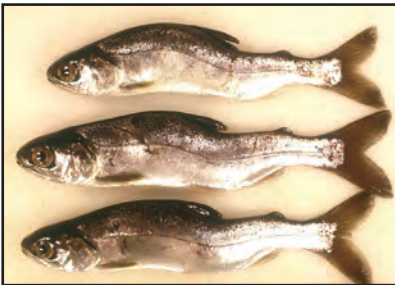
Scoliosis in sockeye salmon smolts surviving IHNV infection
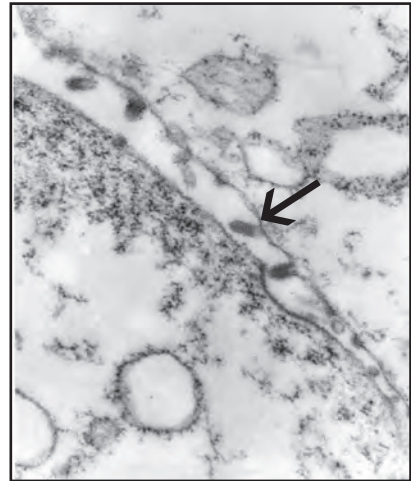
IHN virus particles (arrow) budding from cell membrane of a cultured fish cell

North American Viral Hemorrhagic Septicemia Virus (NA-VHSV)

I. Causative Agent and Disease

North American viral hemorrhagic septicemia virus (NA-VHSV) Type IVa is a bullet-shaped RNA rhabdovirus. It is molecularly distinct from a similar virus (Type IVb) found in the Great Lakes, USA that is pathogenic for a large number of non-salmonid fish species and different from the VHSV strains in Europe that are pathogenic for rainbow trout (Egtved virus) and much less so for some marine fish species.

II. Host Species

NA-VHSV Type IVa infects a wide range of marine host species in the northern Pacific Ocean including anadromous coho and Chinook salmon. In Alaska, the virus has been found in Pacific herring, Pacific cod, Pacific hake and walleye pollock but has not occurred naturally in salmonids. The virus has been associated with epizootic mortality, mostly in Pacific herring and sardines and is enzootic in populations of Northern Pacific herring. Experimental studies have shown that juvenile Alaskan Chinook, coho, pink and sockeye salmon are refractory to the virus by waterborne exposure.

III. Clinical Signs

Detection of NA-VHSV in anadromous salmonids in Washington and Oregon has generally been at very low levels and prevalences of virus and has not been associated with clinical disease. In Pacific cod, secondary VHSV infection has been detected at low levels in skin erosions and ulcers caused by other primary pathogens, but does not become systemic. In Pacific herring the virus can be acutely lethal for up to 100% of exposed juvenile herring with lower chronic mortality occurring in adult fish.

Infected juvenile herring develop hemorrhages of the skin around the mouth and isthmus and/or at the base of fins while occasional hemorrhages occur in adult fish along the flanks that may progress to ulcers in some cases. Fin erosion and lethargic swimming behavior may be present as well. Experimentally infected juvenile rainbow trout exhibited darkened body color and hemorrhaging at the base of fins and vent associated with low mortality.

IV. Transmission

Transmission of VHSV is horizontal through ambient seawater from fish to fish and likely by ingestion of infected fish. Individual infected juvenile Pacific herring can shed up to $10^{6.5}$ plaque forming units (PFU) of virus per ml. Primary virus infection is through the epidermis and possibly gill tissues followed by systemic infection (viremia). Because VHSV in the Pacific Northwest is indigenous to Pacific herring and other forage species utilized by salmon, these prey are the likely source of VHSV periodically detected in adult coho and Chinook salmon in Washington State.

V. Diagnosis

Cultures of susceptible fish cell lines are inoculated with kidney, spleen, liver, ovarian fluids or epidermal lesions from suspect fish. Presumptive diagnosis is made when characteristic cytopathic effect (CPE) or lysis of cells in cell monolayers occurs from virus infection. Virus identification is confirmed by PCR.

VI. Prognosis for Host

Susceptible juvenile herring sustain mortality up to 100% while mortal-

ity may not occur in adult fish or if so mortality is lower and more chronic. Herring that survive virus infection develop apparent immunity to reinfection. Noteworthy is that low-levels of VHSV are generally found in a small percentage of apparently healthy herring from most populations. Clinical disease and mortality from the virus is variable but generally low in other forage species.

VII. Human Health Significance

There are no human health concerns associated with NA-VHS virus.

Skin hemorrhaging in infected Pacific herring often caused by VHSV

Pacific herring with typical VHS lesion
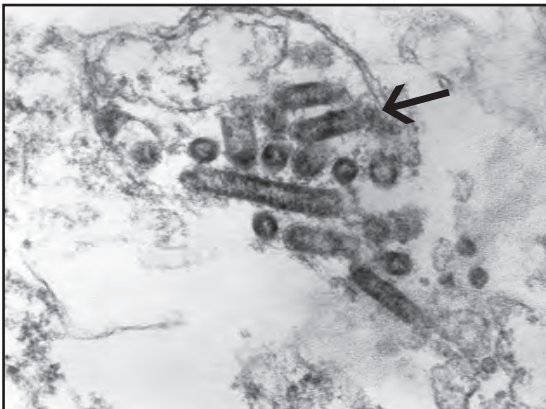
Ultrastructural section of virus particles (arrow) in cultured fish cell, TEM

Chinook Salmon Paramyxovirus

I. Causative Agent and Disease

Chinook salmon paramyxovirus is a large enveloped single-stranded RNA virus in the family Paramyxoviridae. The virus is of low virulence and, in most cases, not associated with disease or mortality. The viral agent is most often isolated from asymptomatic carrier fish during routine screening examinations.

II. Host Species

In North America this virus has been exclusively isolated from adult Chinook salmon from Alaska, Oregon and Washington. Elsewhere, a paramyxovirus has been isolated from seawater reared Atlantic salmon in Norway.

III. Clinical Signs

No gross clinical or histopathological signs of disease are associated with fish infected by this virus except for a paramyxovirus in Norway reportedly associated with the disease syndrome, proliferative gill inflammation (PGL).

IV. Transmission

The mode of transmission is horizontal by water or fish to fish. A marine reservoir for the virus is suspected.

V. Diagnosis

Detection of paramyxoviruses is done by isolating the virus in cultures of susceptible fish cell lines that have been inoculated with infected tissue. The virus causes a cytopathic effect (CPE) characterized by retracted and rounded cells after an extensive incubation period. Presumptive identifications are made by observing the typical CPE. This virus exhibits the unique characteristic of being the only fish virus known to hemagglutinate erythrocytes from fish, some mammals (human, rabbit, horse, guinea pig and swine), and birds. This ability to hemagglutinate is consistent with its placement in the Paramyxoviridae and is used to confirm viral isolates as paramyxoviruses. Confirmatory identification of the viral agent is also done by electron microscopy, fluorescent antibody test (FAT) and PCR.

VI. Prognosis for Host

The prognosis for the host is good regarding the nonpathogenic nature of the North American isolates of this virus. The Norwegian isolate is implicated as causing PGL resulting in fish mortality. In this case there are no corrective therapies for viral infections in fish except avoidance.

VII. Human Health Significance

There are no human health concerns associated with paramyxoviruses in fish.
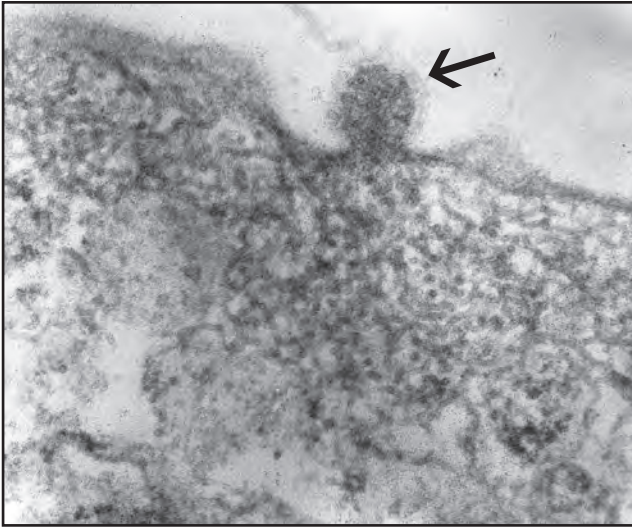
Ultrastructural section of cultured fish cell showing paramyxovirus particle (arrow) budding from cell membrane, TEM

Bacterial Coldwater Disease

*I. **Causative Agent and Disease***

The causative agent of bacterial coldwater disease, *Flavobacterium psychrophilum*, is a Gram-negative very proteolytic bacterium that causes systemic disease in cold waters. Disease associated with this bacterium usually occurs below 12°C and in Alaska often occurs in extremely cold water temperatures of 1°C. The bacterium was originally classified in the genus *Cytophaga*, which was changed to *Flexibacter*, and is now assigned to the genus *Flavobacterium*. The species name *psychrophilum* means "cold loving". Bacterial coldwater disease is most often characterized by tissue necrosis of the fins that progresses to complete destruction of the caudal peduncle exposing the vertebrae by ulceration and necrosis of the surrounding area. Other common names for this condition are peduncle disease or low temperature disease.

*II. **Host Species***

Bacterial coldwater disease can affect salmonids in temperate salmonid producing regions worldwide. Juvenile coho and Chinook salmon are particularly susceptible.

*III. **Clinical Signs***

Fingerlings showing darkening of the peduncle region, where water temperatures are between 4-12°C, may have up to 50% mortality prior to the occurrence of more chronic peduncle erosion. When acute, lesions appear in the areas of increased pigmentation on the peduncle region, or elsewhere. Ulcers are deep, and if fish survive, the caudal fin may erode completely exposing the vertebral column. When present, internal lesions may consist only of mild petechial hemorrhages within the adipose tissues surrounding the pyloric caeca. Chronic coldwater disease can result in lordosis and scoliosis ("crinkleback") and an abnormal swimming posture from the destruction of muscle bundles adjacent to the vertebral column. Another sequella to the disease is bacterial invasion of the brainstem causing erratic swimming, darkened posterior body and sudden death from damage to nervous tissues, vertebral cartilage, and bone.

*IV. **Transmission***

Transmission is horizontal through the water column and vertically through the eggs of infected adult salmonids. *Flavobacterium* sp. are also common inhabitants of aquatic ecosystems. The bacteria have been isolated from internal organs and gonadal fluids of returning adult salmon suggesting they may carry the infection during their seawater phase but reinfection upon entering freshwater is also a possibility.

*V. **Diagnosis***

Presumptive diagnosis is made by isolation of long, filamentous, Gram-negative bacteria that are non-motile or have gliding motility from kidney tissues or typical skin lesions of fish reared in cold water. The bacterium grows well on Cytophaga and TYES agars, with optimum growth at 15-16°C. Typical colony morphology is bright yellow with convex centers and a thin spreading periphery resembling a "fried egg" or an entirely convex colony with no spreading periphery. Colonies turn orange-red when KOH is added indicating flexirubin pigment. Confirmatory diagnosis can be done using PCR.

VI. Prognosis for Host

Bacterial coldwater disease begins as an external infection on young fish that eventually becomes systemic and generally results in mortality. Within the hatchery, populations of fish can be treated for the infection with antibiotics.

VII. Human Health Significance

There are no human health concerns associated with *Flavobacterium psychrophilum*.
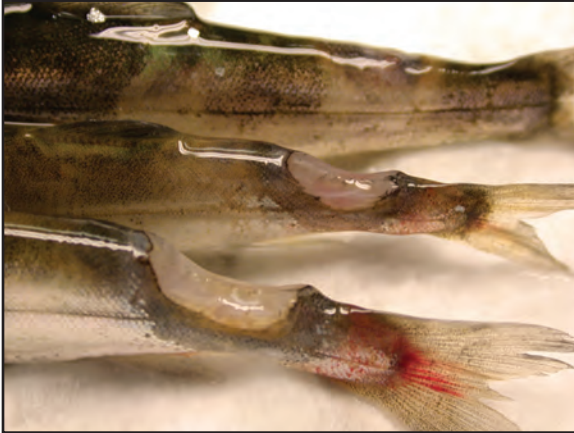
Deep, scooping ulcers characteristic of bacterial coldwater disease on coho salmon fingerlings
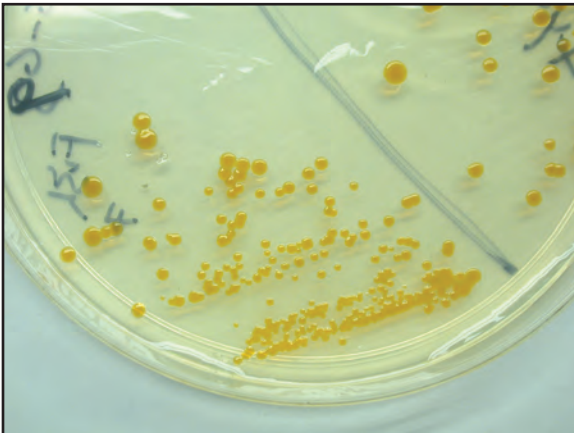
Typical yellow colonies of *Flavobacterium psychrophilum* on TYES agar

Bacterial Gill Disease

I. Causative Agent and Disease

Bacterial gill disease (BGD) is most commonly caused by filamentous bacteria within the genus *Flavobacterium (branchiophila)*. In previous taxonomy these bacteria were known as members of the Myxobacteria and were first placed in the genus *Cytophaga*, later changed to *Flexibacter* and now *Flavobacterium*. The syndrome of this disease includes swollen gill lamellae caused by proliferation of the epithelial cells sometimes resulting in lamellar fusion. The epithelial proliferation is a response to irritation from the large numbers of filamentous bacteria found attached to the gill surface. The thickened epithelial layer results in decreased gas exchange for respiration triggering explosive epizootics with high fish mortality of up to 25%.

II. Host Species

All cultured salmonids are susceptible and the disease is found worldwide. In Alaska, sockeye, Chinook and coho salmon appear to be most susceptible. Adults and yearlings are less susceptible than fry and fingerlings.

III. Clinical Signs

Fish with BGD show a loss of appetite, orient to the water current for increased flow over the gills and exhibit exaggerated opercular movements. An increase in mucus on the head and upper body may also be noted. BGD usually affects fry or fingerling salmonids in high density culture conditions. Therefore, the disease is often associated with sub-optimal water and environmental quality such as overcrowding that result in excessive ammonia, low dissolved oxygen levels and excess suspended organic matter. Such stressors can predispose fish to infection by these bacteria that are present at low levels in the aquatic environment. BGD typically can occur in the spring with the feeding of starter mash that irritates delicate gill tissues of swim-up fry. The resulting gill hyperplasia (excessive cell division and thickness) interferes with normal gas exchange while secondary infections from fungus or other opportunistic pathogens may occur.

IV. Transmission

Transmission occurs horizontally through the water from fish to fish. Predisposing factors for epizootic outbreaks are sub-optimal environmental conditions and suspended solids or abrasive feeds. The incubation period can be as little as 24 hours or up to several weeks, most commonly during periods of colder water temperatures below 5°C.

V. Diagnosis

Fish with BGD have pale, swollen gills, flared opercula, are listless and do not feed well. Large numbers of filamentous rod shaped bacteria are found attached to the gills causing epithelial hyperplasia and possibly fusion or clubbing of gill lamellae. The causative filamentous bacteria are Gram-negative, non-motile (or have gliding motility) and grow on Cytophaga or TYES agars.

VI. Prognosis for Host

Early intervention in the progression of the disease may reduce fish mortality which can be significant. In a hatchery setting external chemical treatments with hydrogen peroxide may help control the bacteria. If gill tissue is severely damaged, fish may not survive the treat-

ments. Preventative measures for BGD include maintaining the water supply free of fish (especially adults), mud and silt, reducing stress such as overcrowding, avoiding low dissolved oxygen or high ammonia levels and avoiding excessive fish handling.

VII. Human Health Significance

The causative bacteria of BGD are of no human health concern.
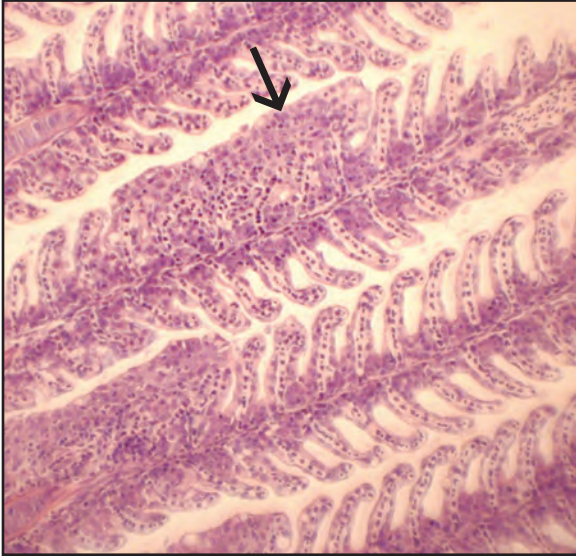
Histological section of gill lamellar fusion (arrow) caused by external bacteria

Higher magnification showing filamentous bacteria (arrow) on gill

Bacterial Kidney Disease (BKD)

I. Causative Agent and Disease

Bacterial kidney disease (BKD) is caused by *Renibacterium salmoninarum* (Rs) that can replicate extracellularly and intracellularly within macrophages. BKD, also known as Dee Disease, is a systemic bacterial infection caused by a small, non-motile, Gram-positive coccobacillus.   Typically, the course of the disease results in slow chronic fish mortality that occurs in Alaska at much colder water temperatures of 1-2°C than reported elsewhere (11°C).

II. Host Species

All salmonids are considered susceptible and the disease usually occurs in fish 6 months or older, i.e., juvenile and adult fish.

III. Clinical Signs

In the acute stage, fish may die without exhibiting any clinical signs of disease. In the more typical chronic form of BKD fish may exhibit exophthalmia, petechial hemorrhages and/or vesicles of the skin, and abdominal distention due to the accumulation of ascitic fluid in the abdominal and pericardial cavities. The kidney, which is the target organ, is often enlarged and edematous and may exhibit off-white nodules varying in size. The whole kidney may appear gray, corrugated and swollen. White foci may also be present in other organs, chiefly the liver and spleen.

IV. Transmission

The Rs bacteria can be transmitted horizontally from fish-to-fish or from a water supply containing infected fish. In early fish culture, feeding raw, unpasteurized viscera of infected fish to other fish increased the incidence of the disease in hatcheries. Unlike many other bacterial pathogens of fish, *R. salmoninarum* can also be transmitted vertically within the egg. The bacteria gain access during egg formation or more commonly enter the yolk through the micropyle after ovulation from contaminated ovarian fluids of the female parent. Transmission from contaminated male seminal fluids during fertilization is another possible route. The organism may survive free in the environment for long periods of time.

V. Diagnosis

Presumptive diagnosis of BKD is sometimes possible by observation of the gross pathology and the presence of intracellular and extracellular Gram-positive, small, non-acidfast, non-sporeforming coccobacilli in Gram stained impression smears of infected tissues. The organism does not grow on TSA but requires a specialized media at 15-20°C for 10 to 21 days of incubation. Organisms can be definitively identified with a specific fluorescent antibody test, enzyme linked immunoabsorbent assay (ELISA) or by polymerase chain reaction (PCR).

VI. Prognosis for Host

This disease results in chronic fish mortality in both freshwater and seawater and can have a detrimental impact on fish populations, generally during the later stages of rearing. Once infected, fish are carriers for life. In Alaska, losses of coho and Chinook salmon fingerlings from BKD can range from 2-5%/month during final months of freshwater or seawater rearing. In numerous watersheds within Alaska, Rs antigen has been detected by ELISA in both wild and hatchery coho, Chinook, chum and pink

salmon. Prevalence usually is less than 10%, but some systems have had carrier rates as high as 90%. Trout, char and grayling in "wild" systems often show prevalences of up to 100%.

VII. Human Health Significance

There are no human health concerns associated with the bacteria.

Petechial hemorrhages in a salmonid fish with BKD
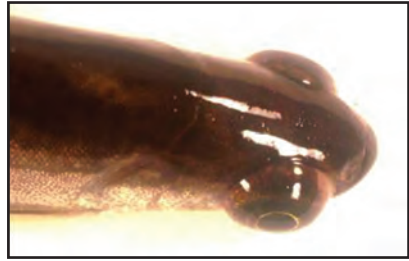
Exophthalmia or pop-eye is commonly seen in fish with BKD and other infections that affect kidney function

White nodules in the kidney of a fish with BKD is a typical clinical sign of this disease
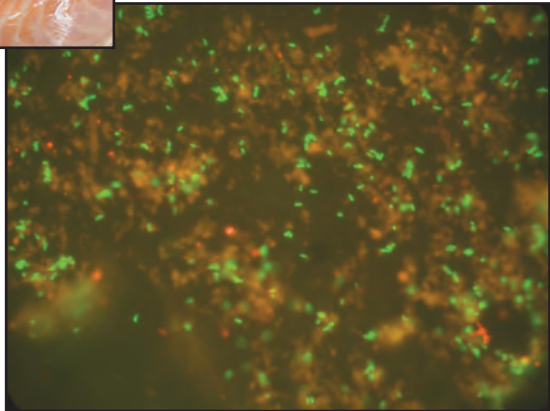
BKD bacteria, *Renibacterium salmoninarum* stained with a fluorescent dye (green) in the fluorescent antibody test

Enteric Redmouth Disease (ERM)

I. Causative Agent and Disease

Enteric redmouth disease (ERM) is caused by Gram-negative, motile bacteria known as *Yersinia ruckeri*. The name ERM is derived from the inflammation and petechial hemorrhages of the lower hind gut and in and around the mouth of infected fish that are not necessarily unique characteristics of infection by this bacterium. ERM is an acute septicemia in salmonids with bacterial foci, necrosis and inflammation in all tissues. In Alaska, two serotypes of the bacteria, known as Type I and Type II, can cause the disease. The two serotypes are differentiated from each other based on biochemical and/or serological tests. The virulence varies considerably within each serogroup but *Y. ruckeri* Type I has been more pathogenic in Alaskan salmonids. The bacteria are found in North America and Europe and there are a total of 6 serotypes worldwide.

II. Host Species

Rainbow trout are the most susceptible host, but all salmonids and several other fish species are susceptible to infection.

III. Clinical Signs

Externally, clinical signs can be similar to other bacterial septicemias. Infected fish are often lethargic and dark in color. Inflammation and petechiation are prominent in and around the mouth, the isthmus and on the opercula. Petechial hemorrhages are commonly seen at the base of the fins. Fish often exhibit exophthalmia and a distended abdomen. Internally the stomach is often filled with watery fluid and petechiation may be present in the musculature and visceral organs, most notably in the hind gut and liver.

IV. Transmission

The bacterium is horizontally transmitted from fish to fish via the fecal oral route and often becomes localized in the lower intestine of fish surviving a disease outbreak. Bacteria can remain viable for a limited time in ambient water to infect susceptible fish. Other reservoirs of the bacteria include fish eating birds reported near aquaculture facilities.

V. Diagnosis

Presumptive diagnosis is made by the cultivation of a Gram-negative, oxidase negative, motile bacterial rod from blood, kidney, or lesions when inoculated on bacteriological media. Diagnosis is confirmed with biochemical tests or fluorescent antibody tests specific for *Yersinia ruckeri* Types I and II.

VI. Prognosis for Host

Under aquaculture conditions, diseased fish generally die if there is no antibiotic intervention. Prognosis for the population is good if the condition is recognized early so that antibiotic therapy can be initiated.

VII. Human Health Significance

There are no human health concerns associated with *Yersinia ruckeri*.

Petechial hemorrhages of the liver as seen in enteric redmouth disease
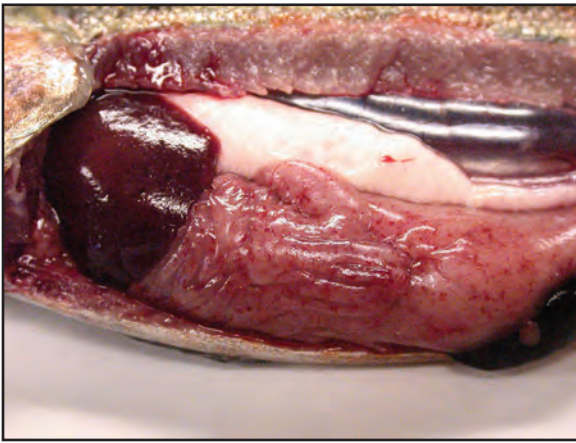
Severe internal hemorrhaging typically seen in bacterial septicemias like enteric redmouth disease

Furunculosis

I. Causative Agent and Disease

Furunculosis is caused by a Gram-negative bacterium known as *Aeromonas salmonicida* and is probably the most commonly encountered bacterial pathogen in cultured salmonids. The disease occurs worldwide in freshwater and has also been reported in the marine environment. It is known to occur in North America, Europe, Asia, and Africa. Furunculosis, is characterized by a generalized bacteremia with focal necrotic swellings in the muscle tissue called furuncles.

II. Host Species

All salmonid species are susceptible. Rainbow trout show some resistance. Young fish are the most susceptible, especially when the water temperatures are > 8° C. In hatcheries, pink and chum salmon are less likely to develop furunculosis since they are not reared long before being released to seawater. Many non-salmonid species of fish in both marine and freshwater are also susceptible to infection by *A. salmonicida*, some strains of which are atypical.

III. Clinical Signs

In acute septicemia where rapid death may occur, gross clinical signs may not develop. In subacute and chronic infections, body darkening, lethargy and loss of appetite are associated with the typical focal necrosis in the muscle, often visible as a swelling under the skin. These lesions eventually ulcerate producing deep craters. Erythema, petechiation and exophthalmia may be present and the abdomen of the fish may be distended with internal ascitic fluid. Bloody fluid may be discharged from the anal vent and the kidney, liver and/or spleen may be enlarged.

IV. Transmission

Horizontal transmission to susceptible fish is via the water column or by the fecal-oral route. Diseased or carrier fish are point sources of infection. Increasing water temperature exacerbates the incidence and intensity of infection. Vertical transmission of the bacteria has not been demonstrated.

V. Diagnosis

Presumptive diagnosis is made by culture of a Gram-negative, oxidase positive (an oxidase negative isolate has been described), non-motile bacterial rod from blood, kidney, or lesions on TSA or furunculosis agar with the production of a brown diffusible pigment. Some strains of bacteria may not produce pigment. Diagnosis is confirmed by biochemical tests, slide agglutination and fluorescent antibody tests specific for *A. salmonicida*.

VI. Prognosis for Host

In nature, the disease usually results in mortality. In a hatchery, prognosis for the fish population is good if the condition is caught early and antibiotic therapy is initiated.

VII. Human Health Significance

There are no human health concerns associated with *A. salmonicida*.

Typical furuncle lesion on adult sockeye salmon with furunculosis

Early furuncular lesion on young salmonid fish with furunculosis
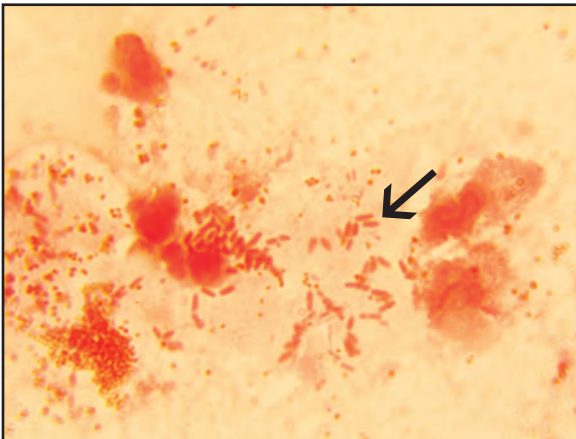
Kidney impression showing Gram negatively (red) stained bacteria (arrow) of *Aeromonas salmonicida* causing furunculosis

Marine *Flexibacter*

I. Causative Agent and Disease

Flexibacter maritimus (Tenacibaculum) is a filamentous, Gram-negative bacterium that does not possess flagella, but moves by gliding motility. These marine bacteria are opportunistic pathogens of fish producing external infections such as bacterial gill disease, fin rot, or eroded mouth disease. Infections are often initiated by physical trauma, pinheading and adverse environmental conditions. Resulting fish mortality can be significant.

II. Host Species

All marine fish worldwide are potentially susceptible to infection by *Flexibacter maritimus* which has been isolated from a variety of salmonid fishes, Dover sole, sea bass, turbot, bream, halibut and sardines. In Alaska, this bacterial pathogen has caused mortality of juvenile Pacific salmon in seawater netpens during the winter and early spring.

III. Clinical Signs

Diseased fish have ulcerated skin lesions, frayed or eroded fins and tail. Moderate to severe erosions of the head and mouth may also occur. Infected epidermal tissue may appear pale yellow to white due to the presence of large numbers of bacteria. Infected gills of fish may produce excessive mucus, have pale color and exhibit lamellar hyperplasia. Secondary systemic infections by other bacteria commonly occur through any open lesions.

IV. Transmission

Flexibacter is a naturally occurring marine bacterium. The mode of transmission is horizontally through the water from fish to fish and generally requires minor external trauma or other environmental stressors.

V. Diagnosis

Diagnosis of *Flexibacter* infections is made by observing large numbers of filamentous bacteria in wet mount preparations of lesion material. The bacteria can be cultured on seawater Cytophaga or TYES agars with 1.5% NaCl added and incubated at 15°C. The colonies are often yellow in color.

VI. Prognosis for Host

This external bacterial infection can cause significant mortality, especially if fish are stressed. Treatment has been successful with oral antibiotic therapy.

VII. Human Health Significance

There are no human health concerns associated with *Flexibacter maritimus*.

Caudal fin lesion on a halibut caused by infection with a marine *Flexibacter*

Severely eroded head of coho salmon smolt due to marine *Flexibacter* infection

Motile *Aeromonas* and *Pseudomonas* Septicemia

I. Causative Agent and Disease

Motile bacterial septicemias are caused by Gram-negative bacteria including *Aeromonas* and *Pseudomonas* with the *Aeromonas hydrophila*-complex and *Pseudomonas fluorescens* being the most common species. The *A. hydrophila (liquefaciens)* complex contains numerous biotypes and serotypes of *A. hydrophila* as well as *A. sobria , A. caviae, A. shuberti and A. veronii*. These bacteria are ubiquitous in the aqua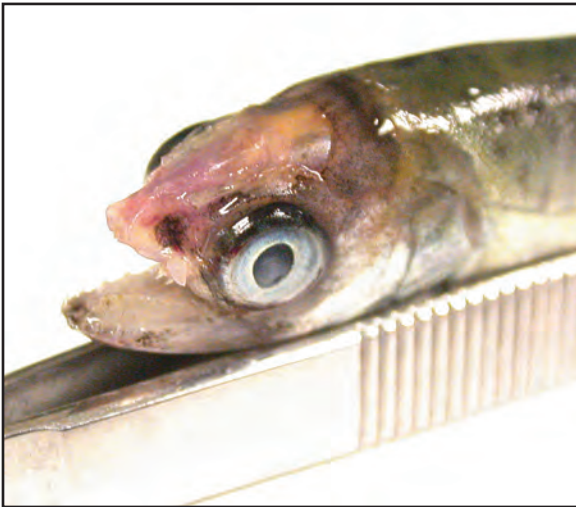tic environment and are found around the world in both fresh and brackish water, but more commonly in freshwater. These bacteria generally cause a systemic, hemorrhagic disease in fish. Most of these bacteria are considered opportunistic pathogens causing disease in fish compromised by stress or other pathogens. Some species, most commonly *P. fluorescens* and *A. hydrophila*, have been reported as primary fish pathogens in systems of high intensity fish culture.

II. Host Species

When less than optimum conditions prevail, all freshwater fish species are likely susceptible to these bacteria. Among salmonids, rainbow trout and Chinook salmon are probably the most susceptible to the *A. hydrophila* complex. Both *Aeromonas* and *Pseudomonas* are pathogenic for other cold-blooded vertebrates including frogs and reptiles and will infect mammals including man through wounds or when they are immunocompromised.

III. Clinical Signs

Lethargy, low-level mortality, and occasional cutaneous lesions on the body surface may occur. Inflammation and erosion in and around the mouth with hemorrhaging and necrosis of the fins is common. Exophthalmia and abdominal distention with ascitic fluid may also be present. Internally, the kidney may be soft and swollen and the spleen enlarged. Petechial hemorrhages may be present internally in many tissues and the intestines may be inflamed and filled with yellow mucus or bloody fluid.

IV. Transmission

These bacteria are among the normal flora of healthy fish and are ubiquitous in the aquatic environment. They are particularly abundant in organically polluted waters while infected carrier fish and other aquatic animals can serve as reservoirs. Transmission is horizontal from fish to fish or from contaminated water. Water temperatures 10°C or above favor these opportunistic pathogens.

V. Diagnosis

A presumptive diagnosis is made when fish exhibit characteristic clinical signs with tissue imprints, squashes or blood smears containing Gram-negative, motile rod-shaped bacteria. A definitive diagnosis is made by isolation of the organism on appropriate bacteriological media followed by identification from biochemical tests.

VI. Prognosis for Host

Severely affected fish will die. However, these bacteria are generally weak pathogens. Poor environmental conditions predispose fish to disease outbreaks which are self resolving without intervention by antibiotic therapy when the source of stress is removed. When necessary, antibiotic therapy can be ef-

fective, except some pseudomonads are resistant to available aquaculture drugs.

cause disease in humans through wounds or when immunocompromised.

VII. Human Health Significance

Some bacteria in these genera can
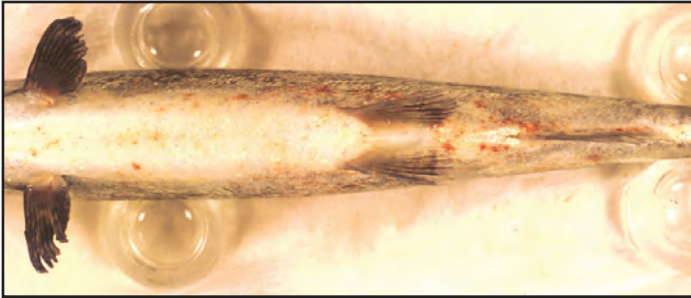
Petechial hemorrhages on ventral surface of a salmonid fish with bacterial septicemia
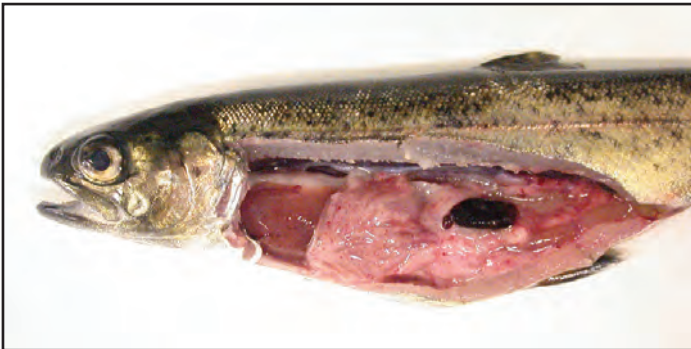
Petechial hemorrhages of liver, pyloric caeca, gut, visceral fat of a salmonid fish with bacterial septicemia

Unidentified Fusobacteria-like Agent

I.   Causative Agent and Disease

An external skin and/or gill infection is caused by long, non-motile, Gram-negative bacterial rods that are sharply pointed at both ends. The bacteria are commonly referred to as fusobacteria and infect cultured salmonid fish in fresh water during periods of very cold water temperatures less than 5°C.

II.   Host Species

This organism has been detected on cultured salmonid fishes at various life stages from alevin to pre-smolt in the Pacific Northwest and Alaska. It has most commonly affected Chinook and coho salmon.

III. Clinical Signs

The skin of infected fish have excessive mucus production and gill infections result in lamellar hyperplasia and increased respiration.

IV. Transmission

These bacteria are probably transmitted horizontally through the water from fish to fish.

V.   Diagnosis

Diagnosis is made by observing Gram-negative, non-motile, bacterial rods with characteristic attenuated ends on the skin and/or gills of infected fish. The biomass of bacteria on the fish surface is often extensive. This bacterial organism has not been cultured successfully on conventional bacterial media but some temporary success has been achieved in nutrient broth at low pH.

VI. Prognosis for Host

External infection by these bacteria results in high fish mortality if there is no intervening therapy. One or two external applications of formalin or hydrogen peroxide have been successful treatments.

VII. Human Health Significance

There are no known human health concerns associated with this fusobacteria-like agent.
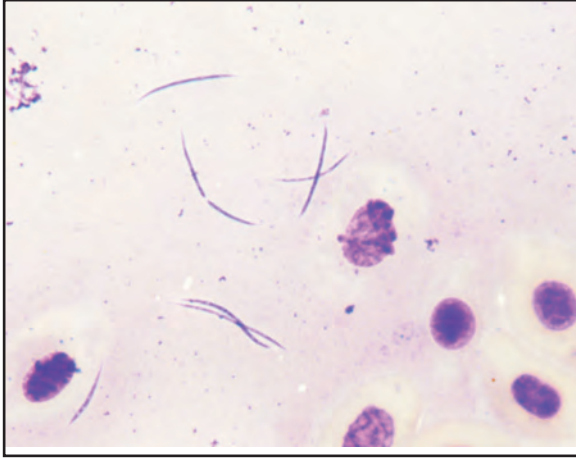
Fusobacteria stained with Giemsa showing typical fusiform shape with pointed ends

Vibriosis

I. Causative Agent and Disease

The genus *Vibrio* contains significant bacterial pathogens of marine fish that cause vibriosis, an acute bacterial septicemia. The primary pathogens include *V. (Listonella) anguillarum, V. ordalii* and *V. salmonicida*. In addition, *Vibrio alginolyticus* may occur as a secondary invader and *V. vulnificus* is generally restricted to European and Japanese waters. *Vibrio salmonicida* is reported from Atlantic Canada and Maine in North America and in Norway, Shetland Islands and Faroe Islands in Europe causing cold water vibriosis or Hitra disease mostly in Atlantic salmon. These bacteria are ubiquitous in the marine environment causing typical Gram-negative acute septicemias with bacterial foci, necrosis, hemorrhaging and inflammation in most fish tissues.

II. Host Species

Because vibriosis has occurred in an extensive number of fish species worldwide, most marine fish species are likely to be susceptible. All species of Pacific salmon and trout are susceptible to vibriosis that quite often involves *V. anguillarum*. Coho salmon seem to be more resistant while chum and Chinook salmon are very susceptible. *V. ordalii* and *V. salmonicida* are principally associated with Pacific and Atlantic salmon, respectively, while *V. vulnificus* most often infects eels causing red pest disease.

III. Clinical Signs

Characteristic clinical signs of vibriosis include inflammation and reddening along the ventral and lateral areas of the fish with petechial hemorrhaging that develops at the base of fins, vent and within the mouth. Acute cases exhibit a darkened body with swollen, cutaneous lesions that ulcerate, releasing blood. There may also be corneal opacity followed by evulsion of the orbital contents. Internally, the intestine may be distended with a clear, viscous fluid. Hemorrhaging is common in the viscera and around the intestines, with swelling and necrosis of the kidney and spleen.

IV. Transmission

Horizontal transmission occurs from organisms in the water, or contact between fish. Outbreaks have occurred in freshwater fish fed carcasses of marine fish. In Alaska, disease does not usually occur until seawater temperatures reach 8°C.

V. Diagnosis

Presumptive diagnosis is made by observing motile, curved Gram-negative bacterial rods in spleen squashes or peripheral blood smears of marine or anadromous fish. Bacteria can be isolated on tryptic soy agar sometimes requiring 1.5% NaCl. Confirmatory diagnosis is made using biochemical or slide agglutination tests.

VI. Prognosis for Host

Epizootics of vibriosis in wild fish populations are rare but result in significant fish mortality. When cultured salmonids are reared in seawater netpens the disease is common resulting in high mortality if not treated with antibiotics. Several licensed vaccine preparations for aquaculture have been effective in the control of vibriosis.

VII. Human Health Significance

The *Vibrio* species associated with most fish diseases such as *V. anguillarum, V. ordalii* and *V. salmonicida* are not considered to be human pathogens. However, several other vibrios are of human health

concern including *V. cholerae, V. vulnificus, V. parahaemolyticus* and occasionally *V. alginolyticus.*

Bloody ascites (arrow) in abdominal cavity commonly seen in fish with vibriosis
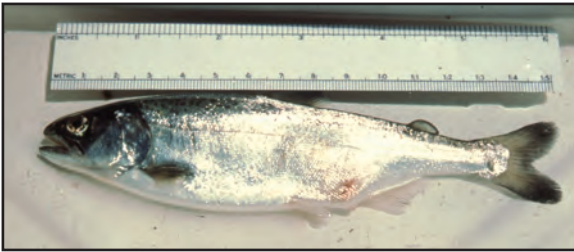
Coho salmon smolt with small posterior external hemorrhage due to vibriosis
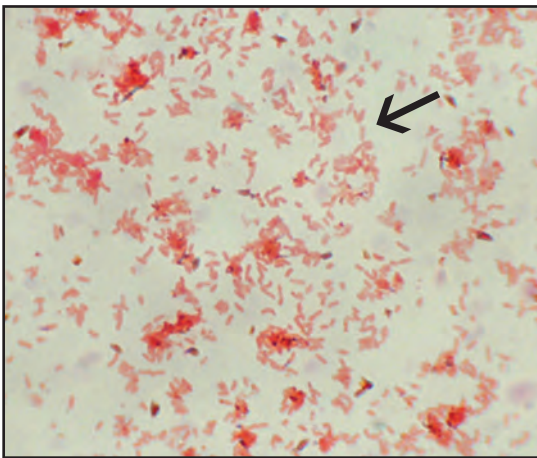
Gram-negative *Vibrio* bacteria (arrow) cultured from infected fish

Phoma herbarum

I. Causative Agent

Phoma herbarum causes a systemic mycotic infection in salmonids and is normally a pathogen of plants. This fungus is a member of the fungi imperfecti which lack sexual reproductive stages and is in the order Sphaeropsidales. The fungus infection is characterized by mycelial invasion of the air bladder and/or digestive tract. The fungus invades other organs becoming systemic resulting in gut obstruction, peritonitis and visceral necrosis with severe hemorrhaging.

II. Host Species

The disease has been found in cultured fry and fingerling coho, Chinook and sockeye salmon, lake trout, steelhead/rainbow trout and Arctic grayling in Alaska and the Pacific Northwest.

III. Clinical Signs

Affected fish may swim on one side or in a vertical position with tail down or may rest on one side at the bottom of the rearing container. Fish often have swollen and hemorrhagic vents and the abdominal area can be laterally compressed in what is referred to as a "pinched abdomen". Fish may also exhibit hemorrhage of the caudal fin and/or petechial hemorrhages on the lateral and ventral body surfaces.

IV. Transmission

Phoma herbarum is a weakly infectious facultative fish pathogen that likely invades either by entrance of conidia or hyphae into the air bladder via the pneumatic duct connecting the esophagus, or by entering with food into the lower gastrointestinal tract where the primary focus of infection may develop. Therefore, transmission of infectious stages is suspected to be oral with food or with gulping air to inflate the air bladder.

V. Diagnosis

Diagnosis is based on typical gross clinical signs and septate fungal hyphae present in the lumen of the air bladder or gut and/or the presence of visceral hyphae. The fungus is cultured by aseptically removing material from the abdominal cavity of an infected fish and plating onto sabouraud dextrose or potato agar and incubating at 16-20°C. Colonies appear as light buff turning to light pink and finally to greenish-gray to black as pycnidia are formed. Pycnidia produce hyaline unicellular conidia. Hyphae are fine in diameter and septate (have cross walls).

VI. Prognosis for Host

There is no known treatment for systemic mycosis in fish. In most cases only a small percentage of the population will become infected. Those fish that are infected will eventually die. In natural infections cumulative mortality is generally low (<2-5%) but can be up to 12%.

VII. Human Health Significance

There are no human health concerns associated with *Phoma*.

Perforated body wall near vent and "pinched abdomens" in fry with *Phoma* infection
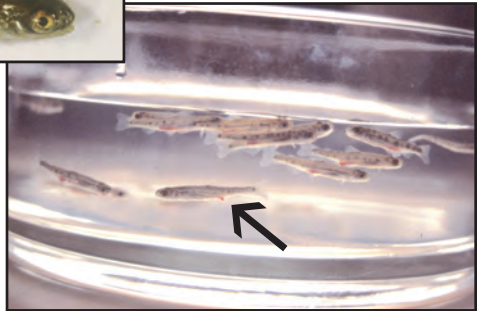
Anal prolapse (arrow) and hemorrhage of sockeye salmon fry infected with *Phoma*
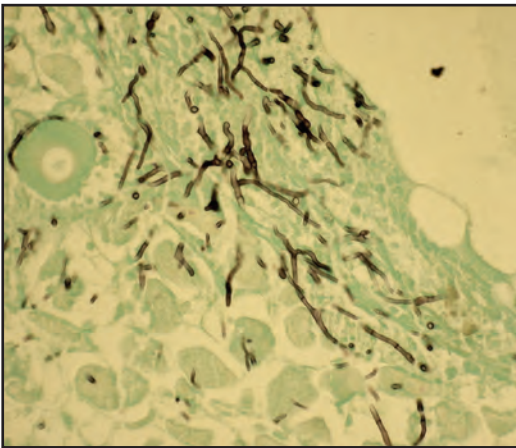
Hyphae of *Phoma* (black) invading fish muscle, stained with Grocotts fungus stain
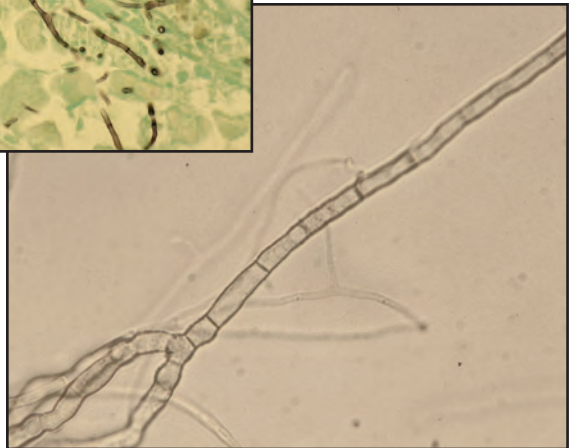
Phoma hyphae exhibiting typical septa or crosswalls

Saprolegniasis – Cotton Wool Disease

I. Causative Agent and Disease

The disease saprolegniasis is caused by freshwater fungi usually in the genus *Saprolegnia*. These fungi are classified in the family Saprolegniaceae, otherwise known as water molds. Saprolegniasis often is used indiscriminately to describe any cotton-like growth of fungus adherent to skin or gills which may include any one of several genera of molds. The fungi are found worldwide in freshwater, although some species may occur in brackish water less than 2.8 parts per thousand salinity. Most species are saprophytes naturally present in the environment (water, sediment) and are considered opportunistic pathogens and secondary invaders requiring prior injury of external tissues from mechanical abrasion or other primary pathogens. Some species of *Saprolegnia (parasitica)* can produce a systemic mycosis and are considered primary pathogens.

II. Host Species

All freshwater fish species and incubating eggs are susceptible to saprolegniasis.

III. Clinical Signs

Externally, the fungus appears as focal white to brownish cotton-like patches on the surface of the skin and/or gills. Early lesions consist of pale foci with peripheral areas of erythema and a central zone of lifted scales which frequently becomes ulcerated, exposing underlying musculature. Systemic infections are characterized by mycelial masses in the gut and surrounding viscera causing peritonitis with extensive hemorrhage, necrosis and adhesions. In smaller juvenile fish external signs of bloating caused by gut obstruction may progress to perforation of the abdominal wall.

IV. Transmission

External fungal infections are transmitted through ambient water by infectious biflagellated zoospores released from hyphal sporangia. Systemic infections in cultured fish occur by ingestion of uneaten food that has been colonized by fungal hyphae. Factors of environmental stress play an important role in the etiology of the external disease. Outbreaks occur primarily after minor injury from handling or during crowded conditions when environmental quality is suboptimal. Adult salmon migrating to spawning areas have weakened immune systems and often have external infections of *Saprolegnia*. Also, cold water temperatures predispose fish to fungal disease because development of zoospores and sexual stages are favored while host tissue repair and the inflammatory response are slowed by the lower host metabolism.

V. Diagnosis

Diagnosis is based on typical gross clinical signs where the skin, gills and other surfaces of an infected fish or eggs become covered with white, cottony tufts of fungal hyphae. Wet mounts of fungal mycelium from lesions show large, branching, non-septate hyphae. Terminal ends of older hyphae form club-shaped sporangia containing biflagellated zoospores. The fungus can be isolated on cornmeal or potato agar.

VI. Prognosis for Host

When external infections are extensive and/or involve the gills death of the host is likely from fluid imbalance and peripheral circulatory failure (shock). In the hatchery environment external fungus infections can be treated success-

fully with formalin drips. There is no treatment for systemic mycoses that are rapidly fatal.

VII. Human Health Significance

There are no human health concerns associated with *Saprolegnia*.
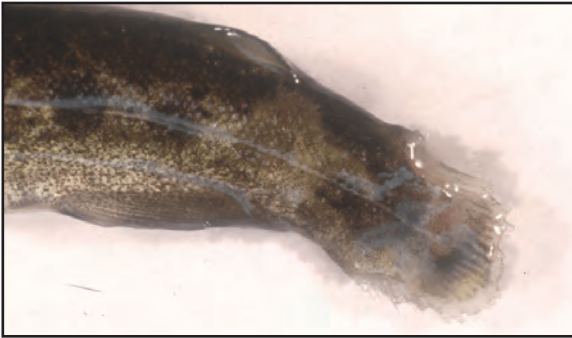
Tail rot due to infestation with *Saprolegnia* fungus
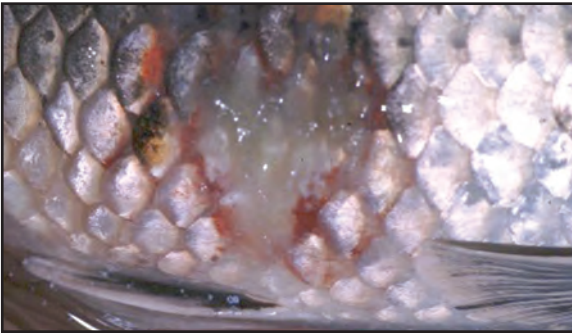
Typical skin lesion due to *Saprolegnia* fungus

Hyphae and sporangia of *Saprolegnia* fungus, phase contrast microscopy

Ceratomyxa shasta

I. Causative Agent and Disease

The parasite *Ceratomyxa shasta* is a protozoan member of the class Myxosporea that produces crescent-shaped or U-shaped spores 14-23μ long by 6-8μ wide at the suture line. A single spore contains two refractile polar capsules, each with an extensible coiled filament. Vegetative trophozoites and spores may be found producing necrotic lesions within various tissues of salmonid fishes but the parasite has a tropism for the gastrointestinal tract, especially the intestine. High mortality may occur in susceptible juvenile fish and the parasite contributes to significant prespawning mortality of infected adult salmon. Depending on the host species and stock, natural exposure to the parasite may allow some fish populations to develop resistance to infection and severity of the disease. Ceratomyxosis occurs seasonally (May to November) becoming more severe when water temperatures reach or exceed 10°C.

II. Host Species

This organism parasitizes several different species of freshwater and anadromous salmonids and is restricted to the Pacific Northwest (PNW) and British Columbia. *Ceratomyxa shasta* has also been found parasitizing wild adult chum and coho salmon, rainbow trout and Dolly Varden in Alaska within several south-central and interior drainages including the Yukon, Naknek, Wood, King Salmon, Togiak and Sushana Rivers as well as Lower Talarik, Mortenson and Russell Creeks. However, no clinical signs of disease have been evident in parasitized wild fish nor has the parasite been found in any hatchery stocks of Alaskan salmonids.

III. Clinical Signs

Parasitized fish typically appear darkened in color with swollen or hemorrhaged vents and abdomens distended by ascites. Although lesions are variable by age and fish species, the entire digestive tract may be affected with granulomas and abscesses (boils) causing tissue necrosis that may spread to major visceral organs and skeletal musculature. These lesions contain developing multicellular trophozoites and spores. Each trophozoite forms a pansporoblast usually containing two spores.

IV. Transmission

Ceratomyxa shasta is transmitted to fish by infectious actinosporean Tetractinomyxon-like stages in the water column that are shed by parasitized freshwater polychaete worms of the species *Manayunkia speciosa* that serve as the intermediate host. The worms become parasitized by ingestion of mature spores released by parasitized live or decomposing fish hosts. However, unlike other myxozoans, the parasite develops within the alternate host epidermis rather than within the intestinal epithelium.

V. Diagnosis

Tissue lesions or intestinal scrapes are examined for spores having the typical size and shape of this parasite. Identity can be confirmed with fluorescein or enzyme conjugated antibody tests and by PCR specific for *Ceratomyxa shasta*.

VI. Prognosis for Host

Depending on the fish species, stock and water temperature, prognosis may be poor with high fish mortality. Major epizootics of juvenile salmonids in PNW

hatcheries have commonly occurred when exposed to surface waters where the parasite is enzootic. Resistant fish in enzootic areas can become subclinical carriers of *Ceratomyxa shasta* with spores occurring in the lower intestinal tract. Prevention of exposure to the parasite is the only effective method of control.

VII. Human Health Significance

Although parasitized fish tissues may be aesthetically displeasing, there are no human health concerns with *Ceratomyxa shasta*.

Stained spore of *Ceratomyxa shasta* showing polar capsules (arrow) and the medial suture line

Ceratomyxa shasta spore with two polar capsules; bar = 10 microns

Coho salmon with swollen prolapsed vent due to infection with *Ceratomyxa shasta* (Photo: R. Holt, Oregon Dept. of Fish and Wildlife)

Bloating due to ascites in fish infected with *Ceratomyxa shasta* (Photo: R. Holt, Oregon Dept. of Fish and Wildlife)

Epistylis (Heteropolaria)

I. Causative Agent and Disease

Epistylis is a sessile, ciliated protozoan that propagates as colonies at the ends of non-contractile stalks on the skin, and sometimes the gills, of fish. *Epistylis* is not a true parasite but an epibiont that utilizes fish only as a substrate for attachment. The disease is one of biofouling rather than infection causing smothering and stress allowing for the invasion of secondary pathogens. The protozoan exists worldwide.

II. Host Species

All species of salmonids are susceptible. Egg masses of catfish and other warm water fish species may also be affected.

III. Clinical Signs

Flashing is a nonspecific sign of external infestation by any protozoan. Infested fish may also produce excessive external mucus.

IV. Transmission

This organism is horizontally transmitted from fish to fish. Slow water flows with high organic loads favor the growth of *Epistylis*.

V. Diagnosis

Diagnosis is made by observation of the protozoa in wet mounts of skin scrapes. The colonies appear like a cluster of bluebells growing on a stalk attached to the fish by a disc.

VI. Prognosis for Host

The prognosis for an infested fish is good if organism numbers are low and fish are not stressed. Heavy colonial growth in a hatchery setting must be treated with chemicals (formalin or hydrogen peroxide) to reduce numbers of protozoa and prevent secondary infections by bacteria and fungi.

VII. Human Health Significance

There are no human health concerns associated with *Epistylis*.
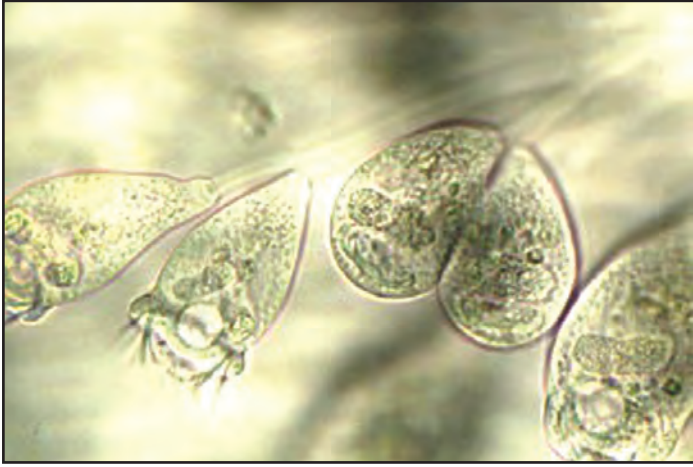
Stalked ciliates of the genus *Epistylis*

Henneguya

I. Causative Agent and Disease

Henneguya is a protozoan parasite in the class Myxosporea. The genus has about 119 different species, some of which are very host and tissue specific. The parasite is found in fish as an ovoid spore (11 x 9 µm) with two anterior polar capsules and two long posterior tail-like processes (26-40 µm). The most common species in Alaska is *H. salminicola*. The spores of this parasite are found in the muscle and under the skin of Pacific salmon causing a condition sometimes referred to as "milky flesh" disease because of the creamy white fluid (spore suspension) that oozes from the cysts (pansporoblasts) during filleting. It is also known as "tapioca" disease from the many
small round spore containing cysts in the flesh.

II. Host Species

Many species of anadromous, marine and freshwater fishes are susceptible to the several different species of *Henneguya* worldwide.

III. Clinical Signs

Fish infected with *Henneguya* have numerous white pansporoblasts (cysts) in the target tissues that are filled with the spores.

IV. Transmission

Henneguya salminicola is transmitted by an infectious stage in freshwater. Pacific salmon become infected as juveniles and the parasites reach the muscle via the circulatory system passing through several developmental stages that eventually develop into spores. The spores are enclosed in a visible pansporoblast or cyst formed of host tissue.

When post-spawned salmon decompose, the cysts rupture and release spores into the water where they are likely ingested by an invertebrate intermediate host such as a tubificid worm. Infectious stages (triactinomyxons) for juvenile salmon develop in the invertebrate host and are released into the water column.

V. Diagnosis

White cysts in the flesh are examined microscopically for the typical 2-tailed spores characteristic of *Henneguya*. The condition can also be diagnosed by histological examination of tissues to verify presence of the parasite.

VI. Prognosis for Host

Fish mortality due to *Henneguya* does not normally occur.

VII. Human Health Significance

Although the cysts in the flesh are visually unappealing when present in large numbers, there are no human health concerns associated with *Henneguya*.

Pansporoblast of *Henneguya* in muscle of a salmon

Henneguya spores showing two polar capsules and two tail-like processes

Hexamita

I. Causative Agent and Disease

Hexamita is a pyriform-shaped protozoan (6-12 um long by 3-5 um wide) with eight (6 anterior and 2 posterior) flagella. This is an intestinal parasite that can cause fatal systemic visceral infestations called hexamitosis in salmonids.

II. Host Species

Members of the genus *Hexamita* parasitize wild, farmed and aquarium freshwater fish and amphibians worldwide. In cold and temperate waters many fish families are potential hosts. *H. salmonis* most commonly parasitizes salmon species.

III. Clinical Signs

Fish parasitized with *Hexamita* may not have any clinical signs. However, when parasites are numerous fish may show signs of anorexia, emaciation, weakness, listlessness, pale gills, abdominal distention, fecal casts, a hemorrhagic vent, exophthalmia and/or dark body coloration.

IV. Transmission

Transmission occurs horizontally in the water by the fecal oral route where ingestion of cysts or vegetative stages (trophozoites) occurs by a host fish.

V. Diagnosis

Diagnosis is made by observation of the protozoan in fecal contents of the gastrointestinal tract (gut) or, if systemic, from visceral smears of parasitized fish. Confirmation is by morphological identification of the parasite based on body shape, size, number and location of flagella using phase contrast or bright field microscopy.

VI. Prognosis for Host

Prognosis for host is dependent upon degree of infestation. Mortalities are associated with heavy, systemic infestations of *Hexamita*. Damage to the intestinal epithelium, intestinal obstruction and anemia contribute to pathological changes in the host fish. Dietary administration of 3% magnesium sulfate has been an effective treatment in salmonids.

VII. Human Health Significance

Hexamita is not known to be a human health concern.
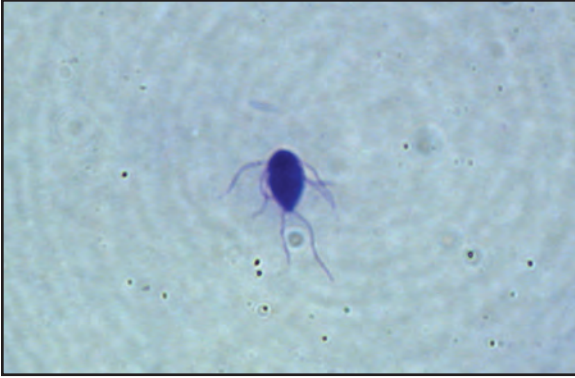
Single *Hexamita* stained with Giemsa
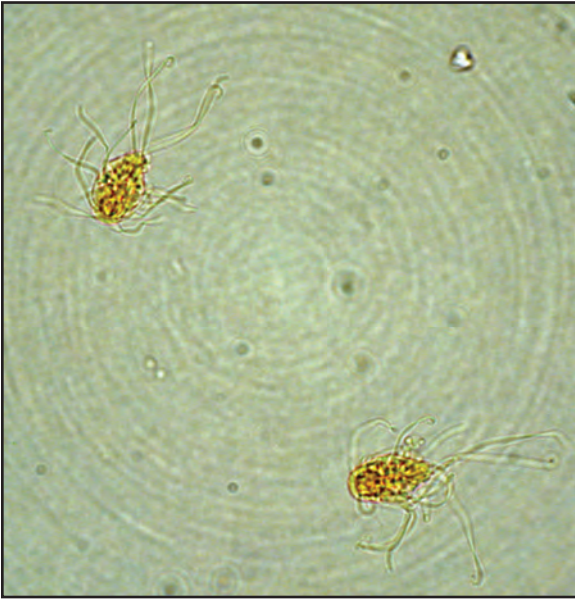
Two *Hexamita* stained with iodine showing posterior and anterior flagella

Ichthyobodiasis (Costiasis)

I. Causative Agent and Disease

Ichthyobodiasis is caused by a flagellated protozoan of the genus *Ichthyobodo*, formally known as *Costia*. These parasites are very small (5-10 um) with both free swimming and attached stages that can easily be overlooked in an examination. *I. necator* (*I. pyriformis* synonym) is an obligate parasite infesting the skin and/or gills of fishes including salmonids. When present on gills *Ichthyobodo* seriously reduces the ability of young salmon to adapt to seawater.

II. Host Species

This organism lacks host specificity and parasitizes a wide variety of warm and cold water fish species and amphibians worldwide. Although primarily in freshwater, there have been reports of marine strains. Fingerlings and fry are especially susceptible, although older fish also become parasitized.

III. Clinical Signs

Fish infested with *Ichthyobodo* are often anorexic and listless and will typically exhibit flashing behavior. In advanced cases a blue-gray film appears on the surface of fish caused by increased mucus production and general hyperplasia of epidermal epithelium. Gill hyperplasia and lamellar fusion (clubbing) can occur if gills are infested. Secondary bacterial and fungal infections are common.

IV. Transmission

This organism is horizontally transmitted from fish to fish. Subclinically parasitized fish are the reservoirs for the parasite in the environment. *Ichthyobodo* reproduces by asexual longitudinal fission where one cell produces 2 motile daughter cells, each with 2 flagella, that parasitize the same or different host. Motile forms attach by means of a flat disc with two small microtubules extending into the host cell, but retain flagella. Infestation of a host must occur within one hour after division or the parasite dies.

V. Diagnosis

Definitive diagnosis is made by wet mount preparations of skin and/or gills. The organisms exhibit a characteristic asymmetrical, oval, flat-bodied attached form with a smaller number of free-swimming forms that are more ellipsoidal in outline. Two unequal flagella are occasionally visible arising from the anterior end and lie along a funnel-shaped groove on the organism's ventral side. The parasites can also be observed as attached forms in stained histological sections.

VI. Prognosis for Host

Ichthyobodo is considered to be one of the most pathogenic flagellate protozoans of salmonid culture causing significant mortality, especially in smaller juvenile fish. In the hatchery environment, *Ichthyobodo* must be removed by chemical treatment, generally formalin. Seawater does not have any effect on the parasite and the severity of the disease may increase among lightly parasitized fish that survive seawater transition but are held for further rearing.

VII. Human Health Significance

There are no human health concerns associated with *Ichthyobodo*.
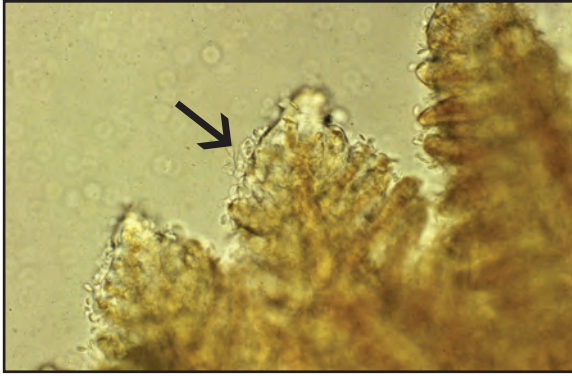
Wet mount of salmonid gills showing numerous *Ichthyobodo* (arrow) attached along the periphery
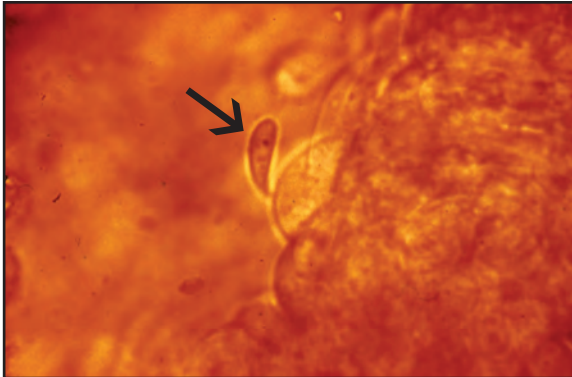
Higher magnification of attached *Ichthyobodo* (arrow)
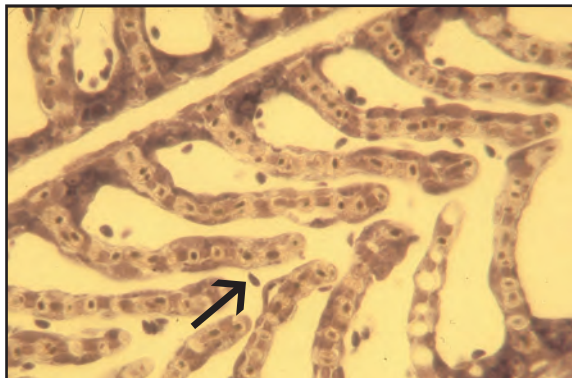
Ichthyobodo attached to gill lamellae (arrow), histological section

Ichthyophonus

I. Causative Agent and Disease

Ichthyophonus hoferi, the causative agent reported for the disease ichthyophoniasis, may actually comprise several different species yet to be identified. Although once considered a member of the fungi, *Ichthyophonus* was recently reclassified as a protozoan member of the class Mesomycetozoea, a highly diverse group of organisms having characteristics of both animals and fungi. The route of infection is probably through the intestinal tract. As with most diseases, the severity is dependent on the general stress and health of the fish host. Once within the body, *Ichthyophonus* is a systemic pathogen localizing in major organ systems including the heart.

II. Host Species

A wide range of marine and anadromous fish species in North America and Europe are susceptible to *Ichthyophonus*. The organism has also been reported from amphibians and reptiles.

III. Clinical Signs

The gross clinical signs of *Ichthyophonus* can be confused with other visually similar conditions. A strong inflammatory response against the parasite often results in visible granulomas encapsulating the macrospores of the organism. These granulomas contain host lymphocytes, macrophages, neutrophils, and fibrous connective tissue that appear as white, yellow or brown foci in infected tissues such as the spleen, liver, kidney, skeletal muscle and especially the heart.

IV. Transmission

Ichthyophonus is an obligate pathogen and considered to be of marine origin. The organism has been experimentally transmitted to Chinook salmon by feeding infected herring tissues. There also is speculation that herring become infected by feeding on copepods that are somehow infected with the organism.

V. Diagnosis

Microscopic diagnosis is made by wet mounts of infected tissues, usually lesions of the heart or muscle. Tissue explant cultures using a liquid *Ichthyophonus* medium can increase detection in lightly infected fish that are not clinically diseased. Microscopic or histological examination of infected tissues can demonstrate the various sized characteristic macrospores and hyphae of the organism. PCR is also available to diagnose *Ichthyophonus* and may be useful when the organisms are no longer viable for culture.

VI. Prognosis for Host

Some species, such as Atlantic herring, are more susceptible to *Ichthyophonus* infections and have sustained mass mortality from the disease. Other species and some stocks within a species have more resistance to exposure and may become infected with the parasite without serious consequences. In experimental studies with juvenile herring death from injection of *Ichthyophonus* macrospores can occur in 80% of the fish within 60 days. Other field studies of adult Pacific herring have suggested the pathogen can persist for long periods without initiating rapid disease or mortality.

VII. Human Health Significance

This parasite is a pathogen only for poikilothermic animals. Therefore, there are no human health concerns associated with *Ichthyophonus*.

Ichthyophonus granulomatous lesions in salmon muscle
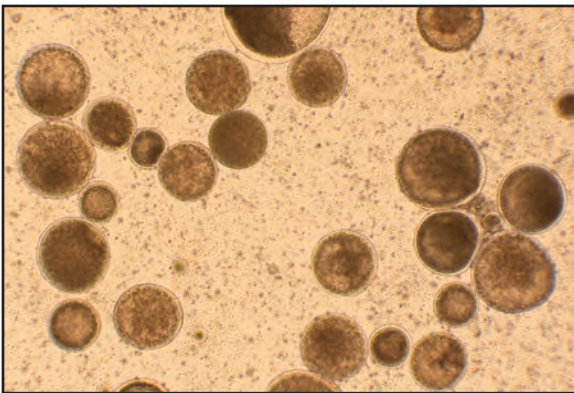
Ichthyophonus resting spores, phase contrast microscopy

Myxobolus squamalis

I. Causative Agent and Disease

Myxobolus squamalis is a protozoan parasite in the class Myxosporea that produces round spores having two polar capsules at one end.   The abnormal condition caused by this species is characterized by cyst-like pansporoblasts under the scales that contain developing spore stages of the parasite. The scales of the fish are pushed up and often look like bumps on the side of the fish.

II. Host Species

This parasite is found mostly affecting anadromous Pacific salmon within the Pacific Northwest. In Alaska, *M. squamalis* is observed most commonly in coho salmon.

III. Clinical Signs

Fish parasitized by *Myxobolus squamalis* have numerous white pansporoblasts under the scales. These cysts raise the scales and are filled with spores.

IV. Transmission

Transmission of *M. squamalis* most likely occurs in freshwater and is based on known life cycles of similar parasites in this class of organisms. Following the death of an infected fish, the cysts under the scales rupture releasing the spores into the bottom sediments where they are eaten by an intermediate host, probably an oligochaete worm. Infectious stages for fish (triactinomyxons) develop in the gut of the intermediate worm host. The triactinomyxons are released to ambient water in large numbers with the feces of the worm, and infect juvenile fish by entering through the skin. The parasite undergoes several divisions toward final development and travels to the specific target tissues, which in this case is under the scales.

V. Diagnosis

White cysts under the scales of parasitized fish are examined microscopically for spores characteristic of *Myxobolus squamalis*. The parasite can also be diagnosed by histological examination to verify presence of the spores.

VI. Prognosis for Host

The effects from *Myxobolus squamalis* are benign and mortality of the host does not usually occur.

VII. Human Health Significance

Although the cysts in the skin are visually unappealing if present in large numbers, there are no human health concerns associated with *Myxobolus squamalis.*

Skin infection by *Myxobolus squamalis* in a coho salmon

Wet mount of *Myxobolus* spore

Trichodiniasis

I. Causative Agent and Disease

Trichodiniasis is caused by ciliated protozoans of the family Trichodinidae in which the most common of 6 genera is *Trichodina* represented by over 30 species. This protozoan is probably the most frequently encountered e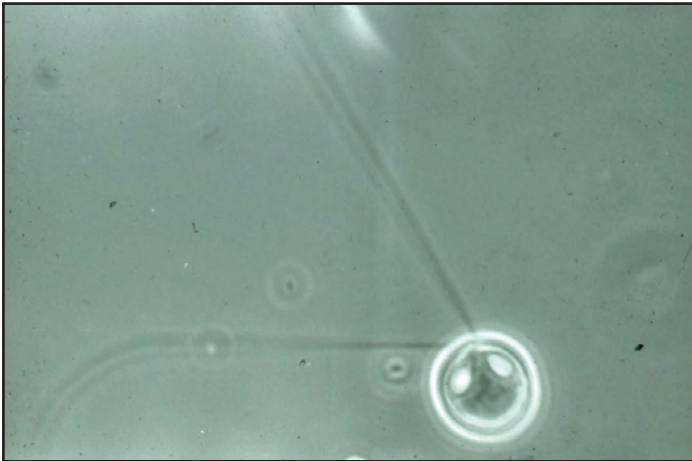xternal obligate parasite in cultured freshwater fishes worldwide. Some species in this family also parasitize fish and shellfish in the marine environment. *Trichodina* (40-60 um in diameter) is saucer-shaped and moves along the surface of the skin, fins and gills of fish by means of its cilia. It feeds on the detritus and other debris found on the surface of the fish using tooth-like structures called denticles. These denticles scrape the debris from the surface of the fish to the mouth of the parasite. When abundant, the scraping and movement of these organisms irritate the skin and gill surfaces causing hyperplasia of the epithelium. Extreme cases of hyperplasia can result in reduced gas exchange or reduced osmoregulation in the fish host. When environmental conditions are suboptimal or when fish tissues are mechanically damaged, more severe infestations may occur.

II. Host Species

Protozoa of this family are found parasitizing freshwater and marine fish species worldwide. Rainbow and steelhead trout, coho and Chinook salmon appear more susceptible than other species of salmonids. Young fish (yearlings or younger) are most susceptible. The parasite has also been reported from amphibian tadpoles.

III. Clinical Signs

Fish parasitized by *Trichodina* often have white patches and/or mottling of the skin and fins. Excessive mucus is produced causing a white to bluish sheen of the skin. Fins are generally frayed and fish exhibit flashing behavior by scraping their bodies against hard surfaces. If the gills are heavily infested opercular movements may be labored.

IV. Transmission

Fish are infested with *Trichodina* through direct transmission from fish to fish or from organisms in the water originating from a subclinically infested reservoir host. The organisms reproduce by binary fission whereby daughter organisms either attach immediately to the original host or seek a new host in the water column.

V. Diagnosis

Diagnosis is easily made by microscopic observation of the highly motile spinning protozoan in a wet mount preparation of skin scrapes or gill tissues. When abundant, the organisms may be visible gliding on the skin surface with the naked eye. Genus and species identification requires microscopic examination of the shape and arrangement of the denticles on the chitin disc surrounding the mouth of the parasite.

VI. Prognosis for Host

Trichodinid protozoa are relatively weak pathogens when compared to other external protozoans infesting fish. The prognosis for parasitized fish is good when parasite numbers are low and fish are not stressed. However, some of these protozoa are serious pathogens causing high fish mortality, especially in hatchery cultured species. Under these conditions external chemical treatment with formalin is necessary and effective in controlling the parasite.

VII. Human Health Significance

There are no human health concerns associated with trichodiniasis.
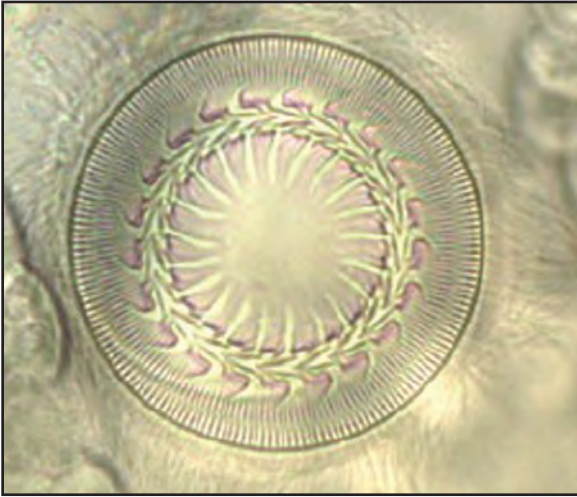
Trichodina ciliated protozoan showing cilia and denticles, phase contrast microscopy
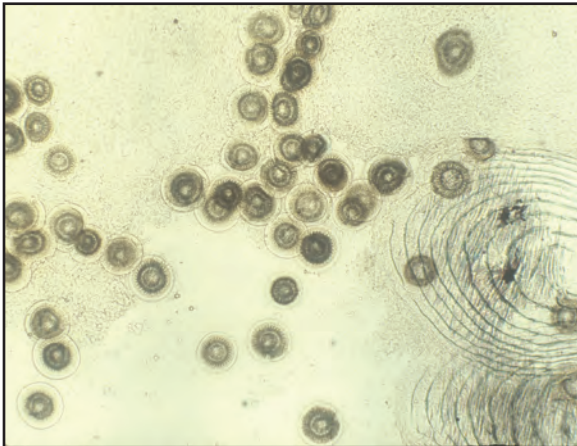
Many *Trichodina* from skin scrape

Trichophrya (Capriniana)

I. Causative Agent and Disease
Trichophrya is a protozoan (30-40 um) in the subclass Suctoria that attaches to the gills, skin or fins of a fish host. The protozoan has suctorial tentacles, which are used to feed on plankton and other ciliates in the water and on fish mucus and epithelial cells. When present in very large numbers, the ciliates can cause pathological changes in the gills including hyperplasia and necrosis that interfere with respiration.

II. Host Species
This protozoan is commonly found on the gills of many freshwater teleosts in North America and Eurasia.

III. Clinical Signs
Nonspecific gill hyperplasia is the principal clinical sign of infestation often accompanied by flashing behavior typical of any external parasite infestation. The parasite may be observed on the gill lamellae by microscopic examination. In Alaska, the occurrence of this parasite is generally incidental to other more significant etiologies.

IV. Transmission
This ciliate is horizontally transmitted from fish to fish. Water with high organic loads favors growth of this organism.

V. Diagnosis
Diagnosis is made by observing wet mounts of skin scrapes or gill tissues. The organism has an oval or irregularly elongated body (variable), which adheres to the gill lamella with a flattened broad attachment surface and the upper surface exhibits tentacles. The body of the parasite appears orange in color, is variably shaped as ovoid or elongated with prominent apical tentacles dorsally that can retract into the cell if disturbed. The broad base of the parasite is attached to gill tissues. Overall, *Trichophrya* resembles a pin-cushion.

VI. Prognosis for Host
Prognosis for the host is good when infestations are light and the fish are not otherwise stressed. When present in large numbers gill hyperplasia can interfere with respiration and predispose fish to infections by bacteria and fungi. Generally, this parasite is not a major cause of fish mortality.

VII. Human Health Significance
There are no human health concerns associated with *Trichophrya*.

Trichophrya protozoan showing typical suctorial tentacles some-times looking like a pin cushion, phase contrast microscopy

Trichophrya attached to gill tissue

Acanthocephalans
(Spiny-Headed Worms)

I. Causative Agent and Disease

Acanthocephalans are endoparasitic worms that are characterized by a retractable proboscis armed with rows of hooks. Adults of many genera can be found in the intestines of fish and some larval forms have also been identified in viscera. They use their proboscis to attach to the intestine of fish. Genera commonly found in Alaskan fishes are *Neoechinorhynchus*, *Acanthocephalus* and *Corynosoma*. Gut infestation by numerous acanthocephalans can cause fibrotic nodules on the surface of the intestine. The intestine may become inflamed with the destruction of intestinal villi and resulting necrotic and degenerative changes in mucosal epithelium. Absorptive efficiency of the fish intestine may be compromised leading to decreased growth and emaciation. Acanthocephalans occasionally perforate the intestinal wall which can lead to peritonitis and death of the host.

II. Host Species

Acanthocephalans have been found in both marine and freshwater fishes worldwide.

III. Clinical Signs

Parasitized fish may be emaciated with inflamed intestinal tracts and tissue necrosis in areas where worms are attached to the intestinal wall.

IV. Transmission

Acanthocephalans require a vertebrate animal as a definitive host and arthropods as an intermediate host. Fish usually are the final host for aquatic acanthocephalans and microcrustaceans (amphipod, copepod, isopod or ostracod) are generally the intermediate host. Intermediate hosts are infected by eating eggs eliminated in the feces of parasitized fish. An egg will hatch in the intermediate host releasing an acanthor that penetrates the gut and develops into an acanthella/cystocanth. The life cycle is complete when a fish eats a parasitized microcrustacean and the adult worm develops in the alimentary tract of the fish host. In some cases, fish are the second intermediate host as well as the final host.

V. Diagnosis

Diagnosis is made by the visual detection of adult acanthocephalans in the intestine or invasive larvae in the body cavity of a parasitized fish. The shape of the proboscis, the arrangement and the number of proboscis-hooks are important characteristics used to definitively identify the species of acanthocephalan.

VI. Prognosis for Host

The principal effects on the final host can include mechanical damage to the intestinal wall and emaciation. Significant fish mortality or emaciation due to infestation by acanthocephalans are rare unless the worms are present in large numbers.

VII. Human Health Significance

There are no human health concerns associated with these organisms.
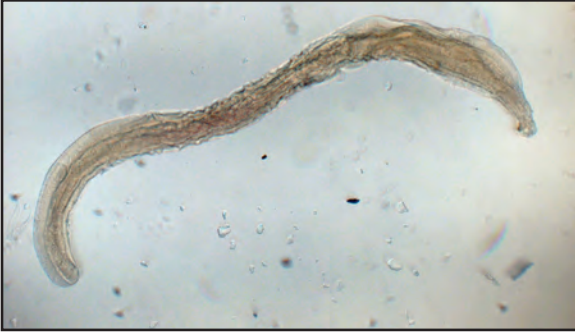
Neoechinorhynchus acanthocephalan worm

A higher magnification of the proboscis

Highly armed proboscis of *Echinorhynchus*

Anisakid Larvae

I Causative Agent and Disease

The larval form (third stage juvenile) of several nematode species within the subfamily Anisakinae are found coiled in the flesh and viscera of parasitized fish. Common genera include *Anisakis*, *Paranisakis*, *Porrocaecum* and *Contraceacum.* The larvae are relatively non-pathogenic to the fish host, although visceral adhesion due to migrating larval worms has been reported.

II. Host Species

Anisakid larvae are common in marine and anadromous fishes worldwide, and have also been reported in squid and cuttlefish. In Alaska, among other fish species, these worms are commonly found in Pacific salmon, Pacific cod, walleye pollock, Pacific halibut and Pacific herring (herring worm).

III. Clinical Signs

Parasitized fish contain white, tightly curled larval worms found most commonly in skeletal muscle and visceral organs. These areas may exhibit mild inflammation, encapsulation and/or granuloma formation. Visceral adhesion may occur in fish when many juvenile worms are present in the visceral cavity. This condition results in the production of fibrous connective tissue by the fish host in response to irritation from migrating worms.

IV. Transmission

Anisakid worms have a complex life cycle involving several hosts. Eggs eliminated in the feces from the final host hatch in the sea where the larvae are consumed by crustaceans (usually Euphausids), which in turn are eaten by fish. The larva burrows into the gut or flesh of the fish and encysts until its life cycle is completed when ingested by the final host, usually a bird, fish or marine mammal. Incidental parasitism of a human host usually results in re-encystment of the juvenile worm. The nematode matures in the gut of the marine mammal host and releases eggs into the sea to continue the cycle. Some anisakid larvae can also be transmitted from fish to fish through predation.

V. Diagnosis

Presumptive diagnosis is made by visual examination of the body cavity, organs and flesh of the fish for typical coiled worms. Examination under a dissecting microscope can verify the identity of the larval nematodes based on morphological characteristics.

VI. Prognosis for Host

Prognosis for the host fish is good. In most cases the worms are well tolerated and there have been few reported cases of fish mortality due to juvenile anisakid parasitism.

VII. Human Health Significance

Anisakiasis in humans can be acquired by eating viable worms in raw or partially cooked fish. The Center for Disease Control recommends cooking fish at 60°C for 5 minutes or freezing at -20°C for at least 60 hours before eating to kill juvenile anisakid worms.

Anisakis
Life Cycle

Humans contract by consumption of raw or undercooked fish (abnormal host)

Marine mammal final host for the adult nematode *Anisakis*

Eggs and second stage larva

Fish and Squid

Free swimming larva ingested by crustaceans (Euphausids)
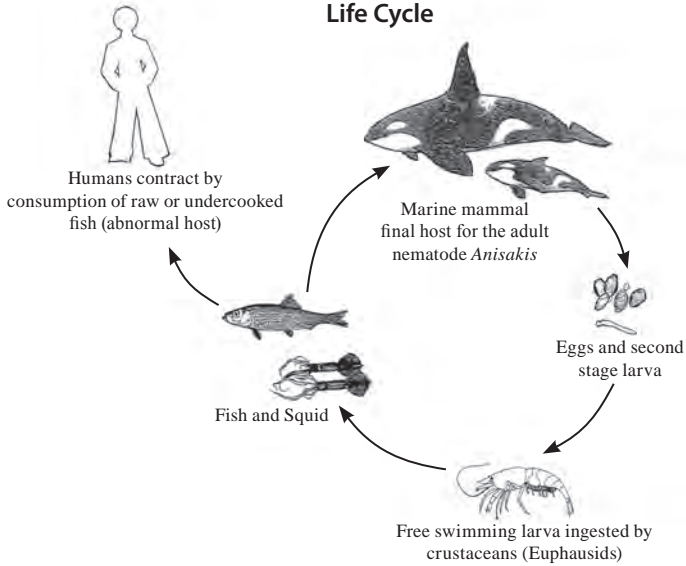
Anisakis third stage juvenile worms tightly coiled in liver
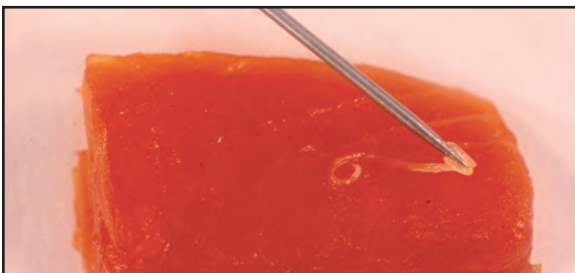
Anisakis third stage juvenile worm being pulled from salmonid muscle

Black Spot Disease *(Neascus)*

I. Causative Agent and Disease

Black spot is caused by dige-
netic trematodes (flukes) in the family
Diplostomatidae. The cercarial forms
of the trematodes penetrate the skin of
a fish, where they encyst and develop
into metacercariae. The fish surrounds
the cyst with black pigmented melanin
in response to the foreign organism.
The black spots are often visible to the
naked eye. These worms are present in
both freshwater (*Posthodiplostomum,
Uvulifer, Crassiphiala*) and seawater
(*Cryptocotyle*).

II. Host Species

Salmonids and other fresh water
and marine fish are second intermediate
hosts.

III. Clinical Signs

Infested fish exhibit black, raised
nodules in the skin which are often 1-2
mm in diameter.

IV. Transmission

Fish are parasitized by exposure to
surface water containing parasitized
snails. The actively swimming cercariae
from the snails penetrate the skin of the
fish where they develop into metacer-
cariae. The definitive hosts are fish eat-
ing birds that complete the life cycle by
releasing eggs into the water with feces.
The eggs hatch into miracidia which
parasitize the snails.

V. Diagnosis

Presumptive diagnosis is made by the
observation of small, multifocal, slightly
raised black spots in the fish skin.
Confirmation is obtained by observing
metacercariae in the cysts in wet mount
preparations or histological sections.

Tissue sections often reveal a thick,
fibrinous capsule around the encysted
metacercariae with the periphery of the
capsule containing numerous melano-
cytes.

VI. Prognosis for Host

Most metacercarial infestations of
the skin are relatively non-pathogenic,
although the aesthetic quality of the fish
is reduced.

VII. Human Health Significance

There are no human health concerns
associated with this parasite.

Typical black spots composed of melanin that surround encysted
metacercariae of the larval genus *Neascus* in an Arctic grayling
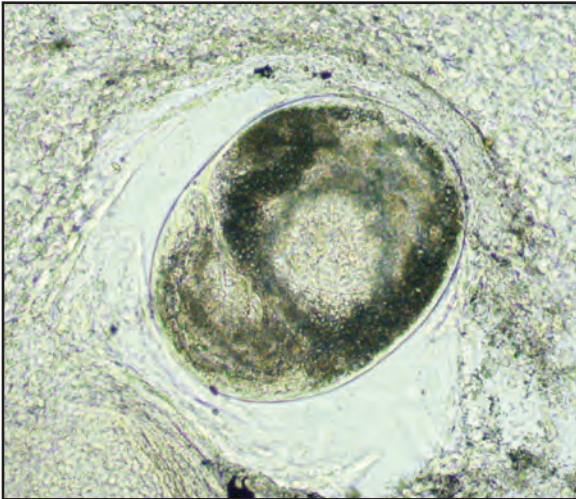
Encysted metacercaria of *Neascus*

Diphyllobothrium

I. Causative Agent and Disease

Six species of diphyllobothrid cestodes (tapeworm) occur in Alaska, all of which use fish as a second intermediate and/or as a paratenic host. Two species of larval *Diphyllobothrium* that most commonly occur in Alaskan salmonid fishes include *D. ditremum* and *D. dendriticum*. The cestode larvae can be found free in the visceral cavity or encysted in the viscera or muscle tissues.

II. Host Species

Plankton feeding and carnivorous fishes are potential hosts including salmonids, whitefish, perch, northern pike, sticklebacks, burbot, and blackfish.

III. Clinical Signs

The larval *Diphyllobothrium* can be found (sometimes encysted) in the muscles, viscera and connective tissues of the fish host causing adhesions, hemorrhaging (particularly the liver) and ascitic fluid resulting in abdominal distension. Severe infestations in juvenile fish can cause mortality.

IV. Transmission

Infestation of the fish host is part of a 3-host life cycle for this parasite. Adult worms are found in the small intestine of definitive hosts that are fish eating birds or mammals (including humans). Eggs from adult worms are released into the water with feces where they develop into a free swimming coracidium larval stage that is ingested by copepods, the first intermediate host. The procercoid develops in the copepod and, when eaten by the fish second intermediate host, develops into the plerocercoid stage. Plerocercoids re-encyst near the gut of predatory fish that become paratenic hosts when other infested fish are eaten. The life cycle is complete when the fish host is eaten by a mammal or bird definitive host where the worm becomes an egg-producing adult.

V. Diagnosis

Diagnosis is made by visual identification of the cestode during necropsy of a diseased fish. Plerocercoid stages of *Diphyllobothrium* have a compressed scolex with characteristic bothria or grooves. The body is usually slightly wrinkled, suggesting segmentation.

VI. Prognosis for Host

Prognosis for the host is good provided the infestation is low and there are not other stressors involved. Juvenile fish are more adversely affected than older fish and can die from severe plerocercoid infestations.

VII. Human Health Significance

Species of this cestode group can successfully parasitize humans. Most human infestations are accidental since the natural hosts are fish eating birds and mammals. Infestation in man occurs by ingestion of raw or lightly smoked fish that contain viable plerocercoid larva. The Center for Disease Control recommends cooking fish at 60ºC for 5 minutes or freezing fish at -20ºC for at least 60 hours to kill worm parasites before ingestion.

Diphyllobothrium
Life Cycle

Humans contract by consumption of raw or undercooked fish

Fish eating birds and mamals are final hosts for adult cestode *Diphyllobothrium*

Eggs

Ciliated larvae – coracidium

Piscivorous fish (paratenic host)

Crustacean ingested by second intermediate host (fish) where larvae develop into plerocercoids

Procercoid larvae in first intermediate host, a copepod crustacean

Brook trout liver with subsurface white cysts (center) containing plerocercoids of *Diphyllobothrium* sp.

Diphyllobothrium sp. plerocercoid with obvious segmentation

Diphyllobothrium sp. – two plerocercoids excised from liver cysts
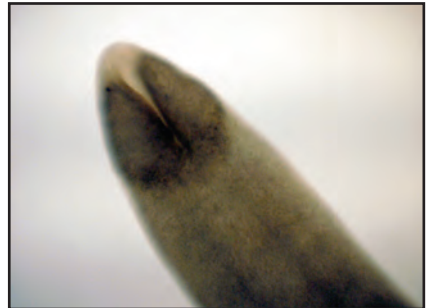
Bothria or grooves in plerocercoid scolex characteristic of *Diphyllobothrium* sp.

Larval *Diplostomulum* of the Eye

I. Causative Agent and Disease

This condition is caused by digenetic larval trematodes (fluke) of the genus *Diplostomulum* that parasitize the eye. One species is found in the lens (*D. spathaceum*) and others are found in the vitreous chamber of the eye. The parasites can remain in the eye for a long time often resulting in cataracts and blindness in the host fish.

II. Host Species

Many salmonids and other fresh water fish are susceptible.

III. Clinical Signs

The fish may have cataracts and the eye will look opaque.

IV. Transmission

As with other digenetic trematodes, the fish becomes parasitized horizontally through the water from infested snails. The invasive cercariae from a snail (first intermediate host) penetrate the fish (second intermediate host), usually through the skin, and migrate to the eye where they develop into the metacercarial form. The life cycle is completed when the fish host is eaten by a piscivorous bird where the adult fluke develops in the gut.

V. Diagnosis

This condition is diagnosed by wet mount observation of metacercariae in the lens or vitreous humor of the eye in a parasitized fish. Typical metacercariae can also be identified using histological methods.

VI. Prognosis for Host

If parasitized bilaterally, complete blindness may result and the host fish will probably die from predation or inability to find food. When only one eye is parasitized, the host fish may survive for a long time.

VII. Human Health Significance

There are no human health concerns associated with this parasite.

Diplostomulum
Life Cycle

Fish eating birds
final host for adult trematode
Diplostomulum

Metacercaria
from lens

Mature egg

Fish with infected eye

Ciliated larvae – miracidium

Free swimming
cercaria

Infected snail
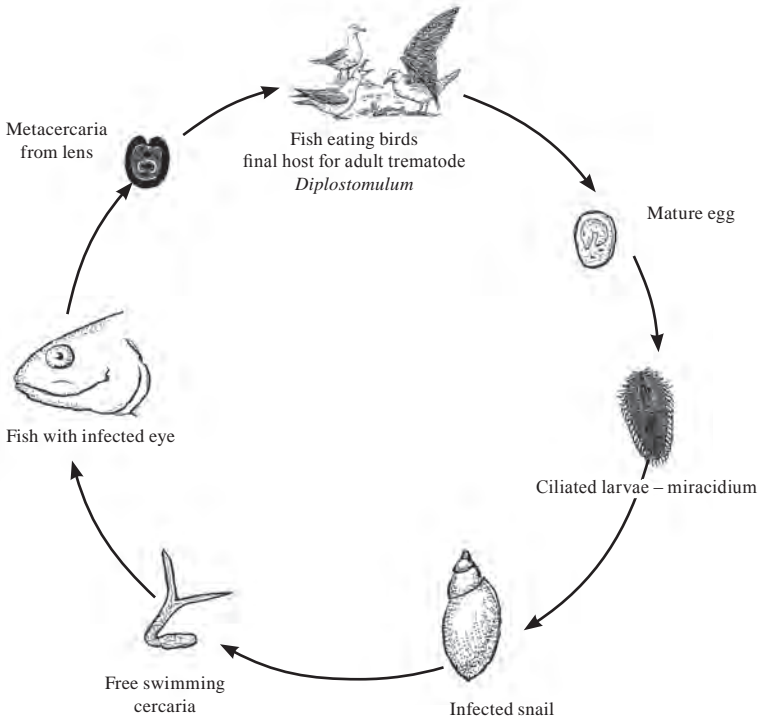
Metacercarial form of the eye fluke *Diplostomulum* from an Arctic grayling

Gyrodactylus and *Dactylogyrus*

I. Causative Agent and Disease

The genus *Gyrodactylus* contains many species but *G. salmonis* is a common parasite of salmonids in North America. This small (0.2 mm) monogenetic fluke attaches to gills, fins and skin epithelium using an attachment organ known as an opisthaptor armed with a pair of large hooks and 16 marginal hooklets. The head of the worm is bilobed, lacks eyespots and produces live young. Heavy infestations by the parasite can result in destruction of the gills or skin epithelium due to mechanical damage caused by the attachment organ. The genus *Dactylogyrus* is found on the gills of mostly cyprinid fishes and is also very small (0.3 mm). *Dactylogyrus* is recognized by a four-lobed head with four eyespots. The opisthaptor consists of one conspicuous pair of large hooks and up to 12 smaller hooklets. When the worm is present in large numbers, gill hyperplasia and necrosis can result.

II. Host Species

The genus *Gyrodactylus* has many species in Eurasia and North America that cause infestations in both marine and freshwater fish. In Alaska they are commonly seen as external parasites of salmonids in the wild and also in the hatchery environment. The genus *Dactylogyrus* is found worldwide parasitizing mostly cyprinids in freshwater.

III. Clinical Signs

The skin of fish infested with *Gyrodactylus* may become mottled, necrotic and dark with excess mucus production. Infestation of the gills often results in lamellar hyperplasia, also accompanied by excessive mucus production and rapid respiratory movements. Heavy body infestations cause fin erosion with flashing behavior and lethargy. Gill infestations of *Dactylogyrus* produce clinical signs very similar to *Gyrodactylus.*

IV. Transmission

Horizontal transmission occurs between fish by physical contact in crowded environments or when the flukes are present in the water seeking a host fish. Both genera are hermaphroditic. *Gyrodactylus* produces live young that attach to the same or different host. *Dactylogyrus* releases fertilized eggs that hatch in the water column producing juveniles that likely attach to a different host.

V. Diagnosis

Diagnosis is made by observing the parasites in wet mounts of skin scrapes or gill tissues. *Gyrodactylus* has no eyespots, is viviparous and embryos with well developed hooks may be seen inside the body of the adults. Dactylogyrid flukes have 4 eyespots and contain visible eggs.

VI. Prognosis for Host

Prognosis for the host is good if infestations are not excessive. If extensive mechanical damage occurs to the fins, skin and/or gills the fish become very susceptible to secondary infections with opportunistic pathogens. Formalin treatments are used in the hatchery environment to eliminate these external flukes from fish.

VII. Human Health Significance

There are no human health concerns associated with *Gyrodactylus* or *Dactylogyrus.*

Gyrodactylus attached to
gill lamellae by hooks

Adult *Gyrodactylus*

Gyrodactylus with visible internal embryos
(arrow)

Stained *Dactylogyrus* with 4 eye spots

Philometra

I. Causative Agent and Disease

Philometra is a nematode parasitizing the body cavities or tissues of fish. Larval stages of this worm migrate to the final resting sites in the subcutaneous tissues (fins, head, and body) or body cavities of predatory fish. The migration of the parasite within the host can result in inflammation of visceral organs, mechanical damage of blood vessels with hemorrhaging and destruction of skeletal joints resulting in poor growth and emaciation.

II. Host Species

Many species of marine and freshwater fish, including salmonids, are susceptible to this parasite that is found worldwide.

III. Clinical Signs

Nodules under the flesh containing juvenile or adult worms cause raised scales or are visible between the fin rays of the fish host. Larger nodules contain gravid females that eventually extrude through the skin to release larvae. After erupting through the skin the female worm disintegrates releasing live larvae followed by complete healing of the host flesh leaving little sign of previous infestation.

IV. Transmission

Philometra has a two-host lifecycle. Larval worms are transmitted through an intermediate host (copepods) to the final host fish. Predatory fish may acquire the parasite by eating infested copepods or forage fish that have preyed on infested copepods. In skin infestations the much larger female parasites break through and burst releasing larvae into the water to be ingested by copepods where the

larvae undergo a series of molts. When parasitizing a body cavity, larvae are released through the gut with the feces of the host or the female migrates to the skin surface to release larvae.

V. Diagnosis

Diagnosis is made by observation of typical *Philometra* worms in fish host body cavities or subcutaneous tissues, particularly the fins, snout, and head or areas of raised scales. Dissection of nodules expose the long, smooth, filiform worms characteristic of the genera. Worms are usually red in color and the immensely larger females contain live larvae and burst easily when placed in water.

VI. Prognosis for Host

Prognosis for the host is dependent on the degree of infestation and other environmental stressors that may be present. Generally, *Philometra* is well tolerated causing no significant harm to fish hosts.

VII. Human Health Significance

Philometra is not of human health concern.
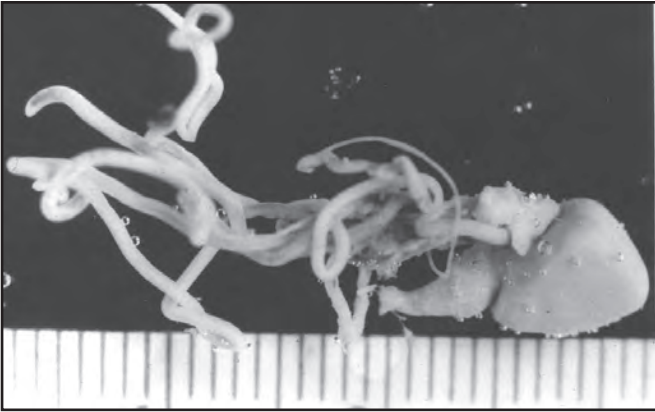
Philometra worms parasitizing the auricle chamber of the heart from a marine fish

Philonema

I. Causative Agent and Disease

Philonema (*oncorhynchi*) is a nematode (roundworm) found in the visceral cavity of fish and rarely migrates to the musculature. Larval, sub-adult and adult worms (17 mm to 86 mm) can be present. The worms generally do not cause significant pathology in the fish host but a condition known as visceral adhesion occasionally occurs in severely parasitized fish. Visceral adhesion is characterized by the production of fibrous connective tissue by the fish host in response to tissue irritation from migrating worms. In severe cases, internal organs are bound together by the scar tissue.

II. Host Species

The parasite occurs in all anadromous Pacific salmon.

III. Clinical Signs

Usually there are no clinical signs of nematode infestation. Highly parasitized fish may have extensive visceral adhesion discovered only by necropsy.

IV. Transmission

Juvenile fish acquire the parasites in freshwater but the adult worms may develop while the fish are at sea. The life cycle includes live larvae released from gravid female worms extruded with fish eggs from adult spawning fish. The larval worms infest a freshwater copepod where they develop into third stage larvae that are infectious for juvenile salmonids. Fish are infested by eating the parasitized copepods and the larvae migrate into the body cavity where molting occurs into sub-adults and eventually adults that produce more larvae.

V. Diagnosis

Diagnosis is made by necropsy of diseased fish and the visual identification of the nematode. *Philonema* is a filiform worm having a rounded anterior end and a posterior tail tapering into a sharp point.

VI. Prognosis for Host

Prognosis for the host is good unless infestation is severe or other stressors further debilitate the fish. Severe parasitism can cause visceral adhesions, interfering with spawning ability. Although adhesions can be serious, the literature indicates this condition is probably transitory and does not cause fish mortality.

VII. Human Health Significance

Philonema is not infectious for humans.
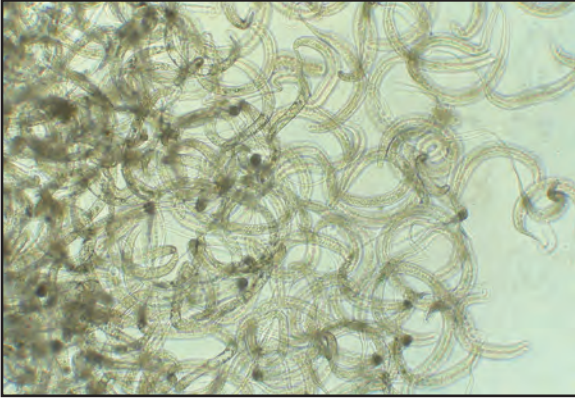
Many juvenile *Philonema* found in the visceral cavity of an adult coho salmon

Juvenile *Philonema* nematode with posterior tail tapering into a point

Piscicola

I. Causative Agent and Disease

Piscicola is a freshwater leech belonging to the phylum Annelida (segmented worms) and can be abundant in some freshwater lakes, ponds and streams. *Piscicola* attaches to the skin of freshwater fish and is nourished by sucking blood and other tissue fluids from the host. Members of the genus *Piscicola* usually remain attached to a fish for several days while feeding and then drop off and sink to the bottom where the food is digested. *Piscicola* has well developed oral and caudal suckers with a sub-cylindrical and elongate body. Leeches usually do not cause serious harm to their hosts since any tissue damage usually is localized at the sites of attachment. However, when present in large numbers parasitic leeches can cause extensive tissue damage to fishes including epidermal erosion and ulceration, hemorrhaging, necrosis and anemia. External epidermal erosions may serve as portals of entry for secondary bacterial or fungal pathogens.

II. Host Species

The parasite occurs on many species of freshwater fishes in Europe and North America. Salmonids are most commonly parasitized (*P. salmositica*) in Alaska.

III. Clinical Signs

Piscicola leeches are visible with the naked eye. Attachment of leeches may occur anywhere on the host body, and are often found on or under the opercula, in the mouth, along the jaw and at the bases of fins.

IV. Transmission

The life cycle of leeches is relatively simple, consisting of an egg, a juvenile stage and a mature hermaphroditic adult that produces eggs. After digestion of a blood meal, a leech either attaches to a fish for another feeding cycle or it produces eggs. Eggs are encased in oval "cocoons" that are attached to the substrate at the bottom of the lake or river. Juvenile leeches hatch from the eggs and enter the water column to find a fish host. Parasitic juvenile leeches usually require several blood meals before becoming mature adults. Leeches of this genus have been implicated as possible vectors of IHNV.

V. Diagnosis

Leeches are obvious by visual examination of the host. Observation of the worm under a dissecting microscope for various morphological characteristics including color and pattern of pigmentation, number and arrangement of eye spots on the oral sucker and other external features help identify the genus *Piscicola*.

VI. Prognosis for Host

Leeches usually do not cause significant harm to their hosts unless present in large numbers. Prognosis for a host is good when infestations are low to moderate but a host inflammation may occur locally at the site of attachment.

VII. Human Health Significance

There are no human health concerns associated with *Piscicola*.

Piscicola
Life Cycle

Adult *Piscicola* leeches
attach to fish host

Hatched, leeches seek
fish host

Engorged leech falls off
host producing eggs

Eggs encased in cocoons
attach to vegetation or
rocks in the substrate

Eggs
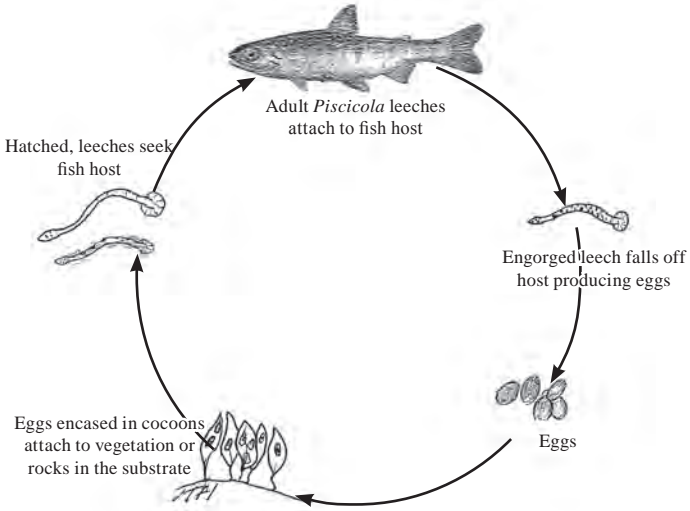
Adult and juvenile *Piscicola* on rainbow trout and typical epidermal
attachment lesions

Fresh water leech of genus *Piscicola* (cm)

Schistocephalus

I. Causative Agent and Disease

Schistocephalus is a cestode (tapeworm) within the family Diphyllobothridae parasitizing fish hosts as a plerocercoid larva transmitted by ingestion of parasitized copepods. The worm in the fish host occurs in the body cavity often causing abdominal distention due to multiple infestations and the large size of the plerocercoids.

II. Host Species

Several freshwater fish species are susceptible to this parasite in North America and Eurasia. In Alaska, this cestode is most often found in sticklebacks.

III. Clinical Signs

Fish with heavy infestations of this parasite are often bloated and misshapen since the parasite is quite large. Normal fish swimming behavior may be impaired.

IV. Transmission

Transmission occurs through a complex lifecycle utilizing two intermediate hosts. The first intermediate host is a copepod that is parasitized by a coracidium hatched from a cestode egg deposited in the water column. A procercoid stage develops in the gut of the copepod that is eaten by the second intermediate host, a freshwater fish. The plerocercoid develops in the second intermediate fish host that is eaten by the final bird host where the adult worm develops and produces eggs in the intestinal tract.

V. Diagnosis

Diagnosis is made by internal observation of the plerocercoid larvae. The white larvae are 2-7 cm long, broader at one end and segmented with shallow bothria (grooves) on the scolex. Fish will often contain multiple plerocercoids.

VI. Prognosis for Host

Prognosis for the host is dependent on the degree of infestation. Pathology caused by the plerocercoids includes growth retardation, abdominal distension, and physiological dysfunction of internal organs. The debilitation caused by the parasite increases the vulnerability of the fish host to predation by the final host.

VII. Human Health Significance

There are no known human health concerns associated with *Schistocephalus*.

Plerocercoid stage of *Schistocephalus* from abdominal cavity of parasitized stickleback

Two plerocercoids of *Schistocephalus* removed from the body cavity of a stickleback

Triaenophorus

I. Causative Agent and Disease

Triaenophorus crassus is a cestode (tapeworm) belonging to the family Triaenophoridae that parasitizes fish as a plerocercoid larva (1 mm X 30 cm) found encysted in the musculature. These cestodes can also be found as adults living in the guts of predatory fish.

II. Host Species

There are many fish intermediate hosts for the plerocercoid (larval) stage of *Triaenophorus* in North America and Europe. The definitive hosts are piscivorous fishes such as northern pike and whitefish.

III. Clinical Signs

Triaenophorus often stimulates formation of yellow to white cysts of host connective tissue that surround the plerocercoids in the muscle. Encysted or unencysted larvae can cause localized muscle discoloration and necrosis. Liver dysfunction and blood loss can occur from larval migration through the viscera and may be associated with hemorrhaging, necrosis, fibrosis, edema and tissue discoloration. Severe adult tapeworm infestations in the gut can cause perforations, mechanical blockage and distension and prevent nutrient uptake causing emaciation and anemia.

IV. Transmission

The life cycle of this tapeworm occurs in freshwater where eggs are shed from adult worms living in the intestinal tracts of the final hosts (usually predatory fish). The cestode eggs are eaten by copepods and develop into procercoids. The copepods are eaten by the second intermediate fish host where the procercoids migrate from the intestinal tract to the muscle and develop into the plerocercoid stage. The life cycle of the worm is completed when the parasitized fish is eaten by the final fish host, commonly a northern pike. Eggs are produced after the worm develops into an adult in the intestinal tract of the final fish host.

V. Diagnosis

Diagnosis is made by observation of encysted or unencysted white plerocercoids in the skeletal musculature of a parasitized fish. Identifying microscopic features of the plerocercoid scolex (head) include dorsal and ventral pairs of trident-shaped hooks on an apical disc. Adult worms are larger and found in the intestinal tract.

VI. Prognosis for Host

Prognosis for the fish host depends on the degree of cestode infestation, the age and size of the fish and exposure to other stressors. Generally, these parasites have caused health problems with juvenile cultured fish but are well tolerated when occurring as natural infestations of larger healthy fish.

VII. Human Health Significance

Although this tapeworm is not known to occur in man or other warm-blooded animals, infested fish flesh is unsightly.

Characteristic trident shaped hooks on scolex of *Triaenophorus crassus* plerocercoid found in fish muscle

Triaenophorus Life Cycle

Adult pike, a common final host for the adult cestode *Triaenophorus*

This fish eaten by piscivorous fish, the final host – with development of adult tapeworm

Eggs

Ciliated larvae – coraciium

Crustacean ingested by second intermediate host (fish) where larvae develop into plerocercoids

Procercoid larvae in first intermediate host, a copepod crustacean
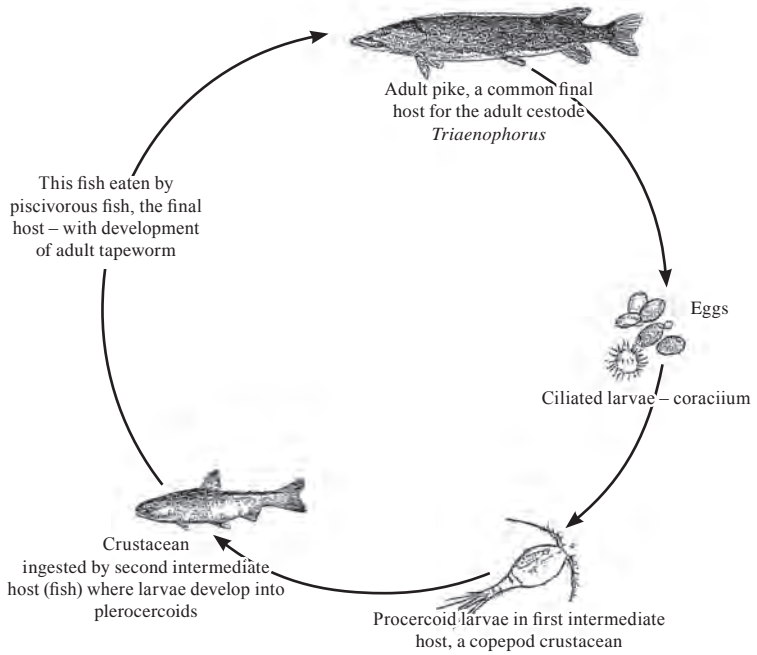
External Parasitic Arthropods

I. Causative Agent and Disease

A variety of different parasitic arthropods can cause external infestations of freshwater and marine fish. Some members of the group are commonly referred to as fish lice. They are commonly found on the body, around the mouth, and on the gills. Members of the class Copepoda commonly found in Alaska include the genera *Lernaea* (anchor worm) in both fresh and marine waters, *Salmincola* (discussed in other section) in fresh water and *Lepeophtheirus* (sea lice) in marine waters. The most common member of the class Branchiura in Alaska is the genus *Argulus* (fish louse). Fish infested with external parasitic arthropods are often lethargic and may flash or rub against substrate. In heavy infestations the skin may look opaque due to the production of mucus and the fins may be frayed. If epidermal or gill tissues become necrotic, secondary infections by fungi and bacteria can occur. These parasites are found worldwide.

II. Host Species

A variety of different freshwater and marine fishes are susceptible to infestations with these arthropods.

III. Clinical Signs

Parasitized fish may act listless and lethargic. Mechanical abrasion due to the attachment and/or feeding by the arthropods is common resulting in frayed fins, gill hyperplasia, and patchy epidermal damage and necrosis. Infections with secondary pathogens often occur.

IV. Transmission

Most of these organisms have a direct life cycle involving a number of free-living and larval stages. Transmission is through contact with an infective free-swimming stage of the organism in the water column. The infective stage attaches to the fish where it goes through a number of larval stages before becoming an adult.

V. Diagnosis

The larger parasites can be seen with the naked eye. Definitive identification is based on microscopic morphologies of body parts and structures.

VI. Prognosis for Host

The prognosis for the host depends on the type, location and number of parasites present. If parasite numbers are small, fish normally survive with little deleterious effects. When present in large numbers, such as *Lepeophtheirus* in seawater netpens, they can be serious pathogens causing significant fish mortality.

VII. Human Health Significance

There are no human health concerns associated with these organisms.

Argulus or fish louse

Lepeophtheirus salmonis copepod from the surface of a salmonid fish

Salmincola

I. Causative Agent and Disease

Parasitic copepods of the genus *Salmincola* are most often found attached to gill filaments, opercula, tissues within the mouth cavity, and fins of salmonid fishes. The parasites feed on blood and epithelial tissues of their hosts. *Salmincola* species are restricted largely to fresh water, but may survive on salmonids while at sea. The adult female copepods are larger than the males and attach permanently to the fish host with a modified mouth part known as a bulla that is inserted into the host tissues. Host damage by parasitic copepods depends on the location of the attachment site, the species of parasite, and the size and type of bulla. Gill attachment by *Salmincola* can damage delicate epidermal tissues resulting in necrosis and loss of surface area for respiration. Attachment may also provide portals of entry for secondary invaders such as bacteria and fungi.

II. Host Species

Salmincola has been reported more commonly from salmonid species in North America and Europe.

III. Clinical Signs

Salmincola copepods are visible to the naked eye when attached to fins, bases of fins, skin, opercula, gills and branchial chamber. Gill damage caused by displacement from *Salmincola* can be extensive resulting in retarded filament growth and tissue necrosis. Gill hyperplasia and hypertrophy may also lead to fusion of the filaments thus reducing surface area for necessary gas exchange and respiration.

IV. Transmission

Salmincola have a direct, but complicated life cycle. Females produce two clusters of eggs twice during a 3-month life span. Eggs hatch into a larval form that can survive free-swimming for several days. The larvae attach to gills or fins of a host fish and molt into 4 successive larval stages and degenerate into grub-like parasites. Males then detach and copulate with the females, after which the males die. Females molt into the adult stage and produce two pairs of egg clusters. The female *Salmincola* dies shortly after the second group of eggs hatch.

V. Diagnosis

Salmincola are large enough to observe grossly. Visual examination of fish skin, fins, gills and mouth can reveal the extent of copepod infestation. Microscopic examination of various morphological characteristics aid in identifying the parasite to the genus and species.

VI. Prognosis for Host

Prognosis for the host is good when infestations are not severe and damage to gill tissue is minimal. Generally, infestations with this parasite do not cause significant fish mortality.

VII. Human Health Significance

There are no human health concerns associated with *Salmincola*.

Severe *Salmincola* infestation of rainbow trout gills; note necrotic areas at tips of gill lamellae

Salmincola Life Cycle

Adult *Salmincola* attaches to fish host

← Egg sacs

Copepodids are released from egg sacs

Free swimming copepodids

Copepodids molt several times while attached to fish host

Adult female copepod with button shaped bulla and male attached

—male
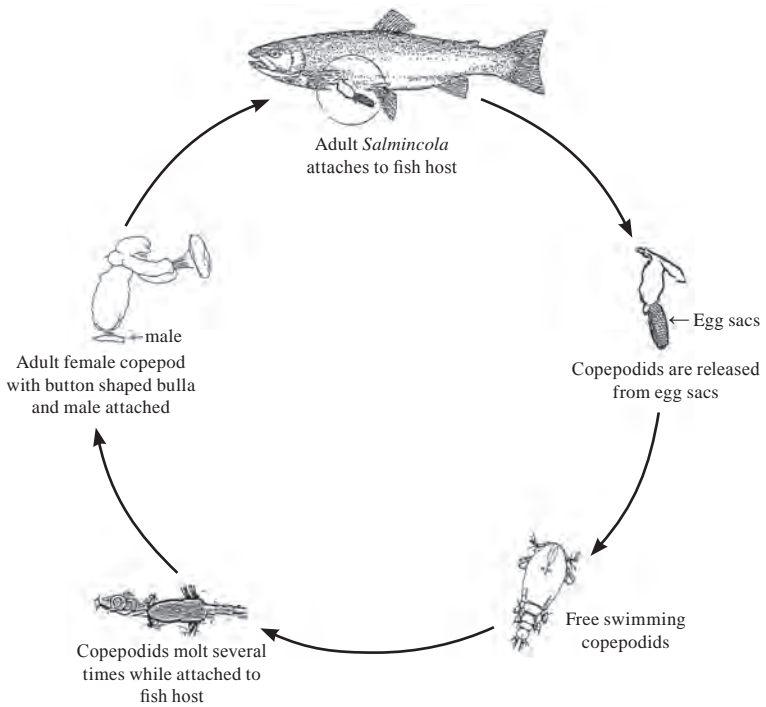
Sarcotaces

I. Causative Agent and Disease

Sarcotaces arcticus is an endoparasitic copepod several centimeters long found encysted under the skin and in the muscle tissue of marine fish. The copepod inserts its head into the flesh, and is eventually covered by the host skin except for the last pointed body segment that maintains connection with the outside seawater. When the copepod dies, the tissue forms a closed cyst around the parasite. In Alaska, this parasite is most commonly found in rockfish (*Sebastes* spp) encysted near the anus where surrounding intestinal tissue forms a sac like process. When fish are filleted the ruptured cysts release a black fluid from the breakdown of blood that the parasite has engorged.

II. Host Species

This parasitic copepod is found most commonly in species of *Sebastes* spp. in the northern Pacific Ocean and in several other genera of teleosts in European waters.

III. Clinical Signs

Sarcotaces parasites are difficult to detect externally because of the subdermal location. If the encysted copepod (the female) lies over a bony surface, swelling of the skin may occur.

IV. Transmission

The life cycle is direct having only one host. Transmission is horizontal by release of larval copepodids into ambient seawater from the encysted adult female. The juveniles seek out a new host to continue the cycle.

V. Diagnosis

Diagnosis is made by internal examination of the fish for characteristic pear shaped cysts exuding black fluid and microscopic identification of the larger female parasite. The body is oval with ill-defined transverse bands corresponding to segments and a double rosette is often visible around the mouthparts.

VI. Prognosis for Host

Prognosis for the host is good if infestation is minimal and there are no significant environmental stressors present. Infestations by *Sarcotaces* are associated with lower fecundity in rockfish and fish flesh becomes unappealing when fillets are tainted with the fluid leaking from cysts.

VII. Human Health Significance

There are no human health concerns with *Sarcotaces*.

Sarcotaces encysted in intestinal tissue near anus of rockfish
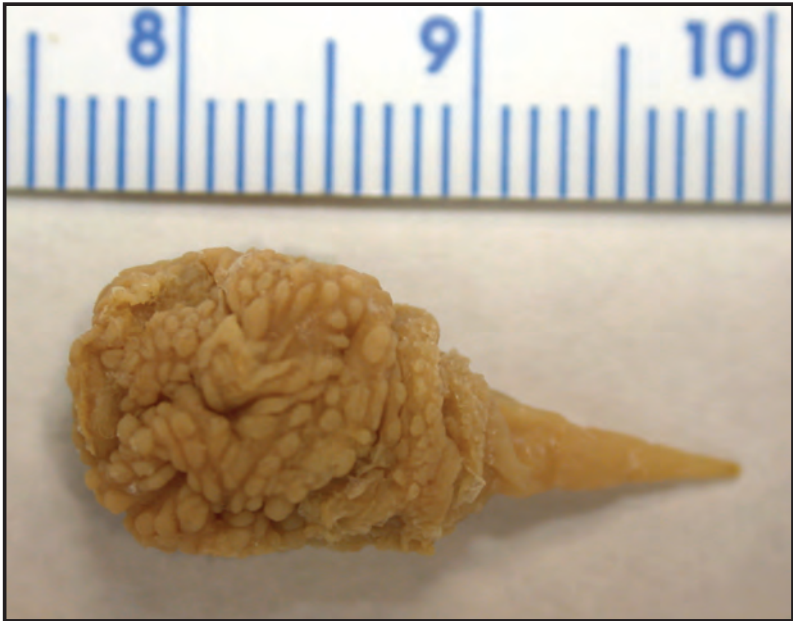
Adult *Sarcotaces* dissected from a cyst (mm)

Bloat (Water Belly)

I. Causative Agent and Disease

This is a non-infectious condition where the abdomen of salmonids is abnormally distended by an enlarged, water-filled stomach. The condition is most often seen in salmonids reared in seawater. The cause of this condition is not well understood, but potential causes may include: a combined failure of osmotic regulation; increased lipids, temperature and stress; increased drinking of seawater and nutrient overloading due to excessive feeding.

II. Host Species

This condition is observed frequently in Chinook, coho, chum and Atlantic salmon and also in rainbow trout. In Alaska, this condition is most common in chum and Chinook salmon.

III. Clinical Signs

Fish with bloat exhibit severe distention of the abdominal wall. Necropsy reveals a massively enlarged stomach with a very thin wall. The stomach is filled with a clear, watery fluid mixed with feed.

IV. Transmission

The disease is non-infectious and cannot be transmitted from fish to fish.

V. Diagnosis

Bloat is usually diagnosed by the presence of excessive amounts of clear, watery fluid in the stomach. The stomach wall is thinned from distension, but other significant histological changes are not present.

VI. Prognosis for Host

Although this condition can cause mortality, affected fish often survive for weeks with the condition. A reduced feeding regime after fish have been starved for several days or changing the composition of the food will reduce the problem in captive fish.

VII. Human Health Significance

There are no human health concerns associated with this condition.
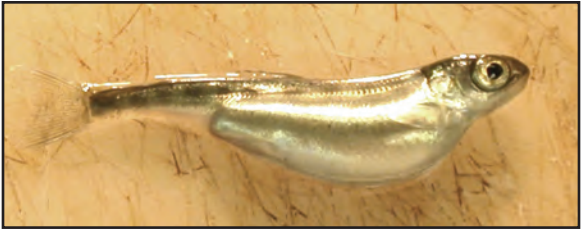
Chum salmon fry with characteristic signs of bloat

Chinook salmon smolt with characteristic signs of bloat

Blue Sac Disease of Fry

I. Causative Agent and Disease

Blue sac disease of fry is a non-infectious disease that is caused by the accumulation of metabolic wastes and reduced dissolved oxygen resulting in excessive buildup of ammonia nitrogen.

II. Host Species

This condition has been reported primarily in salmonid fishes, especially brook trout and other char that tend to be the most susceptible species.

III. Clinical Signs

The alevin/fry exhibit an abnormal accumulation of fluid, often bluish in color, at the posterior of the yolk sac often progressing to surround the entire yolk. Due to the increased fluid, fry cannot swim normally. Fry may have ex-ophthalmia, coagulated yolk, and appear smaller and pale. Petechial hemorrhages of the head, thoracic and vitelline blood vessels can occur in severe cases with hemorrhaging into the blue-sac fluid and severe anemia.

IV. Transmission

Due to the environmental nature of this disease, transmission between fish does not occur.

V. Diagnosis

Diagnosis is based on the observation of typical clinical signs of the condition.

VI. Prognosis for Host

The condition is usually fatal due to improper organogenesis and body development.

VII. Human Health Significance

There are no human health concerns associated with this condition.

Swollen yolk sacs of cultured lake trout caused by Blue Sac Disease

Coagulated Yolk Disease
(White Spot Disease)

I. Causative Agent and Disease

Coagulated yolk disease is a non-infectious condition resulting from unsatisfactory environmental conditions during incubation. A wide variety of factors probably contribute towards the disease including gas supersaturation, unfavorably high water temperatures, heavy metals in the water supply (Cu, Al, Zn), soft water, low water flows, low dissolved oxygen, exposure to chemicals or contaminants, excessive handling and otherwise inadequate or stressful incubation conditions. Yolk proteins become denatured and coagulate as manifested by the appearance of white-spots in incubating eggs and the yolk sacs of hatched alevins. Yolk resorption is disrupted, resulting in defective development of vital organs. This causes physiological alterations in organ functions resulting in death of eggs during incubation or of alevins and larger juveniles.

II. Host Species

All fish eggs and alevins are susceptible.

III. Clinical Signs

White spots or flecks in eggs are typically at the surface of the yolk and randomly distributed. In alevins, the coagulated yolk appears a few days after hatching and may enlarge and coalesce with time. In fry that have completely absorbed the egg sac the coagulated yolk appears as a white mass in the visceral cavity, sometimes associated with clotted blood. Pinheading, anemic gills, and white or frayed fins are sometimes observed in affected fry. Noninfectious tail and fin erosion (especially pectoral fins) can be caused by unabsorbed coagulated yolk that remains in the body cavity and interferes with the ability of the fish to maintain the replacement of epithelium covering the extremities; the fins are the first to be affected but is self-limiting, commonly observed in young chinook salmon shortly after transfer from incubators and troughs to rearing ponds.

IV. Transmission

This disease is not infectious and cannot be transmitted from fish to fish.

V. Diagnosis

Diagnosis is made by observing the abnormal white flecks or masses of coagulated yolk in eggs, alevins or fry.

VI. Prognosis for Host

Most fish with coagulated yolk will eventually die before reaching 1 gram in size due to improper organ development. Juveniles appear normal then suddenly drop-out.

VII. Human Health Significance

There are no human health concerns associated with this condition.
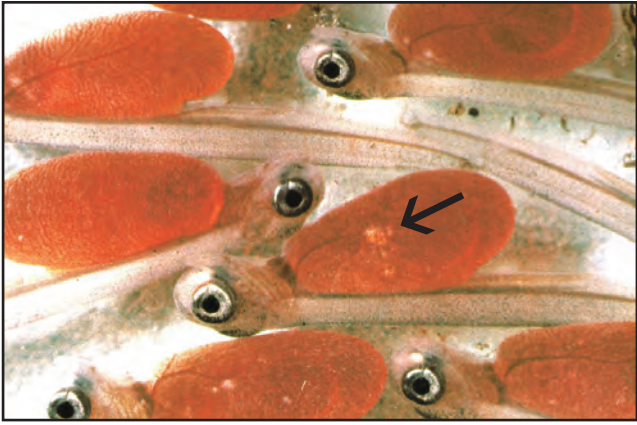
Coagulated yolk (arrow) or white spot in salmonid alevins

Drop-out Disease

I. Causative Agent and Disease

Drop-out disease is commonly observed in hatchery reared juvenile salmonids but is not caused by an infectious agent or a deficiency in the diet. Affected fish may exhibit gill hyperplasia and severely clubbed gills, usually stop feeding and become emaciated or pinheaded. Other forms of drop-out are not associated with gill hyperplasia. Secondary bacterial, fungal and protozoan infections often develop in affected fish.

Drop-out associated gill hyperplasia-causes include:

1. The fine particles in starter feeds irritate delicate gill epithelium.
2. Diatom blooms of *Chaetoceros convolutus* can cause severe gill abrasion in fish that are held in seawater netpens.
3. Repeated therapeutic chemical treatments for external parasites and bacteria can irritate gill epithelium.

Drop-out not associated with gill hyperplasia – causes include:

1. An increase in feed pellet size may prevent a proportion of the smaller fish from eating enough to maintain good body weight and they become pinheaded.
2. White spot or coagulated yolk causing incomplete organ development can result in mortality of seemingly healthy fish during early or later juvenile stages. Fish are not pinheaded.
3. Not enough yolk (minimum 3-5% body weight) remaining when alevins emerge from incubators. Mechanical removal may be necessary, especially with chum salmon.

II. Host Species

All salmonids are susceptible but the condition is seen most frequently in Chinook, coho and chum salmon in Alaska.

III. Clinical Signs

Fish may stop feeding and become emaciated resulting in thin bodies and large heads referred to as pinheading. Gill hyperplasia, sometimes very severe, occurs in many instances but not all as indicated previously. Secondary infections commonly occur adding confusion to the primary diagnosis.

IV. Transmission

Since this is not an infectious disease, transmission between fish does not occur.

V. Diagnosis

Diagnosis of drop-out and its cause depends on whether the fish are pinheaded and have or do not have gill hyperplasia and have been exposed to one or more of the conditions listed.

VI. Prognosis for Host

Mortality can be up to 20-30% of the population or higher in the case of an algal bloom. Drop-out from gill hyperplasia can be corrected by removal or avoidance of the gill irritant(s). Drop-out from coagulated yolk is a sequella from preexisting conditions that cannot be changed in the current cohort of fish but could be prevented in the next production cycle by improving environmental quality during incubation.

VII. Human Health Significance

There are no human health concerns associated with this condition.

Gill hyperplasia commonly seen in dropout disease due to irritation caused by feeding starter diets
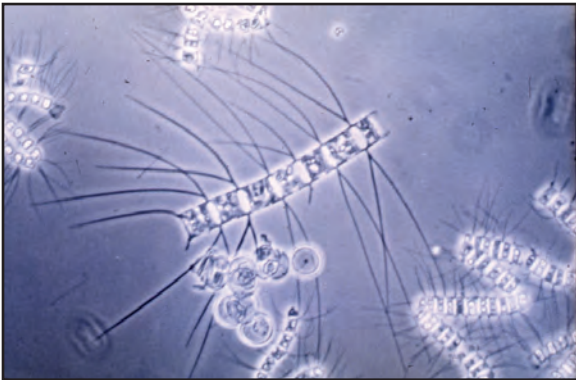
Wet mount of *Chaetoceros convolutus* diatoms

Gas Bubble Disease (GBD)

I. Causative Agent and Disease

Gas bubble disease is not infectious and is caused by supersaturated levels of total dissolved gas in the water. Lesions in the fish are caused by the accumulation of gas bubbles in blood vasculature and tissues. Either supersaturation of oxygen or nitrogen can result in the disease, however, the total dissolved gas (TDG) is more important than individual gases or varying combined gas ratios. Supersaturation occurs when water contains more dissolved gas than it can normally hold in solution at a given temperature and atmospheric pressure. Under high pressure or at low temperatures water can contain more gas. Gas supersaturation in water can occur from both natural and artificial causes. In nature, supersaturation occurs in plunge pools at the base of waterfalls, in natural springs and wells where water is under pressure at depth, and in water that has melted from glaciers or snow. During the photosynthetic process water bodies containing heavy aquatic plant growth can be saturated with oxygen and can become supersaturated upon warming. Artificially supersaturated water occurs in plunge pools from dams, when water is heated such as power plant effluent, and if air is entrained in pipes or pumps where pump pressure or gravity head forces gas into solution.

II. Host Species

The disease can affect any fish or invertebrate anywhere when in supersaturated waters. Levels of gas supersaturation causing pathological changes or mortality vary for different fish species and age of fish. Fry become susceptible post-hatch when they begin swimming up for food (at about 16 days). Steelhead trout are most sensitive (average threshold of 102-103% TDG before chronic problems develop) while coho salmon are least sensitive (average threshold 115.7% TDG)

III. Clinical Signs

Fish with GBD often exhibit loss of equilibrium, abnormal buoyancy and may float at the water surface. Fish may also exhibit violent head shaking, convulsions, flared opercula, release of excessive gas from buccal cavity, blindness and may die with the mouth open. Alevins may show hemorrhage of vitelline vessels, rupture of yolk-sac membranes, and coagulated yolk. Subcutaneous bubbles can accumulate in tissues of the head, mouth, fin rays, and gill arches. Air bubbles are often visible in gill lamellar capillaries. Hemorrhage of gills, fins, skin, muscle, gonads, and intestinal epithelium can also occur. Fins may be eroded with whitened fin tips and exophthalmia may occur with blood present in the anterior chamber of eye.

TDG

100-106%	Embolic lesions will appear with hemostasis
$\geq 103\%$	Certain species of salmonid fry are stressed and may later develop conditions leading to death (i.e., coagulated yolk, fin erosion, tail erosion, etc.)
> 120%	Acute levels, fry will die before signs or lesions indicate a problem

IV. Transmission

Due to the environmental nature of this disease, transmission between fish does not occur.

V. Diagnosis

Diagnosis is made by the observation of typical clinical signs and lesions. The presence of gas emboli in capillaries of the gills, fin rays, mouth and eyes are diagnostic.

VI. Prognosis for Host

Gas bubble disease often results in chronic low-level fish mortality, especially in a hatchery environment. In natural waters, fish exposed to high TDG's will seek greater depth or cooler waters where gas saturation is lower.   There is no evidence that gas supersaturation adversely affects hatching success of salmonid embryos. The great mimic, GBD often predisposes fish to other secondary bacterial, viral or protozoan diseases that must be differentiated first before determining the primary problem.

VII. Human Health Significance

There are no human health concerns associated with GBD in fish.
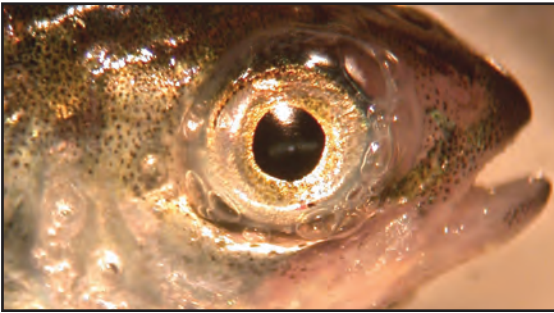
Visible gas bubbles in vasculature of operculum and in eye as seen in acute gas bubble disease
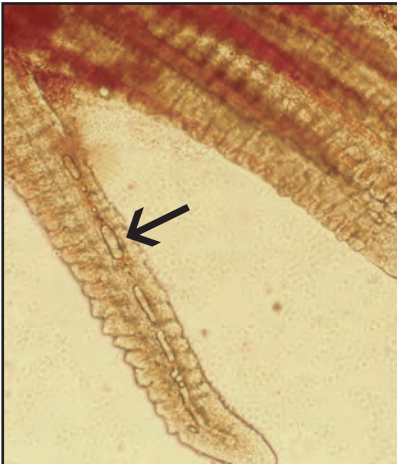
Gas bubbles (arrow) trapped in capillaries of gill lamellae as typically seen in gas bubble disease

Gas bubbles in and around mouth.

Mushy Halibut Syndrome

I. Causative Agent and Disease

Smaller halibut of 15-20 lbs. caught by sportfishing charters near Homer and Soldotna, AK have had a condition locally known as "mushy halibut". Typically, this condition consists of fish having large areas of body muscle that is abnormally opaque and flaccid or jelly-like. The overall body condition of these fish is usually poor and often they are released because of the potential inferior meat quality.

II. Host Species

Smaller Pacific halibut in the Cook Inlet and Homer/Seward areas of Alaska.

III. Clinical Signs

Fish are asymptomatic except for poor body condition. Large areas of the fillets are abnormally opaque and flaccid in texture.

IV. Transmission

No infectious agents or parasites have been detected in affected fish, therefore, transmission from fish to fish is not likely. A nutritional deficiency is suspected.

V. Diagnosis

Diagnosis is by gross observation of flaccid, opaque musculature with confirmation of a noninfectious degenerative myopathy by histological examination. Microscopically, there is severe muscle fiber atrophy, fragmentation and necrosis with a loss of muscle mass. In some cases there is accompanying inflammatory cells, fibrosis and calcification of atrophied fibers.

VI. Prognosis for Host

Reportedly, the Cook Inlet and Homer/Seward areas are nursery grounds for large numbers of young halibut that feed primarily on forage fish that have recently declined in numbers. Stomach contents of smaller halibut now contain mostly small crab species. Whether this forage is deficient, either in quantity or in essential nutrients is not known. However, mushy halibut syndrome is similar to that described for higher animals with nutritional deficiencies in vitamin E and selenium. This muscle atrophy would further limit the ability of halibut to capture prey possibly leading to further malnutrition and increased severity of the primary nutritional deficiency.

VII. Human Health Significance

Although aesthetically displeasing, there are no known human health concerns with mushy halibut syndrome.

Flaccid, glistening halibut flesh typical of mushy halibut syndrome
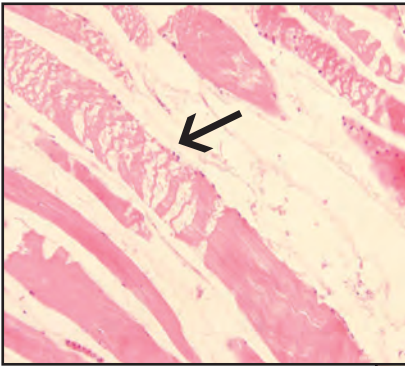
Skeletal muscle fiber atrophy with fragmentation (arrow) necrosis and loss of muscle mass (empty spaces)
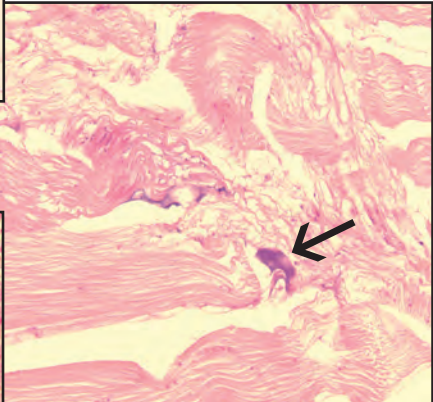
Early calcification (arrow) of atrophied muscle fibers
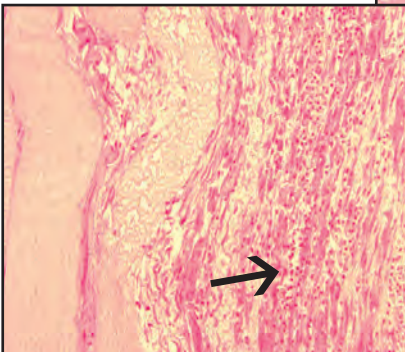
Atrophied muscle fibers with fibrosis and infiltration of inflammatory cells (arrow)

Neoplasia (Tumors)

I. Causative Agent and Disease

Tumors or neoplasms are tissue growths of abnormal cells that proliferate uncontrollably. In bony fishes, neoplasms of the connective tissues, such as fibroma and fibrosarcoma, are most common. Fish develop neoplasia or cancer in much the same way as do higher animals. Known and suspected factors contributing to neoplasia in fish include viruses, environmental chemicals (carcinogens), repeated physical trauma, hormones, age, sex, genetic predisposition and immunological competence of the host.

II. Host Species

All teleost fishes in any part of the world could potentially develop neoplasia. For unknown reasons cancer has been rare in cartilaginous fishes such as sharks and rays.

III. Clinical Signs

Neoplasms usually become apparent by gross observation of an external or internal swelling, lump, or formation of an abnormal tissue growth.

IV. Transmission

Except for neoplasia caused by infectious viruses, horizontal fish to fish transmission does not occur. Generally, neoplastic growths are spontaneous within an individual due to congenital malformation, age or genetic predisposition but could also be caused by environmental conditions.

V. Diagnosis/Classification

Definitive diagnosis is made by observing the abnormal cells using histopathological methods. Neoplasms are classified according to the cell or tissue of origin and are further grouped based on benign or malignant characteristics. Benign tumors are often well-differentiated, grow slowly, are well circumscribed without invading surrounding normal tissue and do not metastasize. Most benign neoplasms are not usually life threatening and often end in the suffix "oma". Exceptions are benign neoplasms of the brain and some endocrine organs that can be life threatening due to their location and deleterious physiological effects on the host. Malignant tumors are often not well differentiated, may grow rapidly, infiltrate normal tissues and tend to metastasize. The names of these neoplasms are often preceded by the word "malignant" or with the suffixes "sarcoma" or "carcinoma". Types of cancer in fish include the following:

TISSUE TYPE	BENIGN TUMORS	MALIGNANT TUMORS
epithelial	papilloma	epithelial carcinoma
	adenoma	adenocarcinoma
mesenchymal	fibroma – connective tissue	fibrosarcoma
	leiomyoma – smooth muscle	leiomyosarcoma
	rhabdomyoma – striated muscle	rhabdomyosarcoma
	lipoma - fat	liposarcoma
	chondroma - cartilage	chondrosarcoma
	osteoma - bone	osteosarcoma
hematopoietic	lymphoma	lymphosarcoma
blood vessels	hemangioma	hemangiosarcoma
neural – nerve cell	schwannoma	glioma, astrocytoma
pigment	erythrophoroma	malignant melanoma
embryonal	nephroblastoma	-

VI. Prognosis for Host

Prognosis for fish having neoplasms depends on the type of tumor and whether the lesion is benign or malignant. Benign tumors are usually not life threatening. Malignant tumors can cause mortality if growth is rapid and interferes with normal organ functions.

VII. Human Health Significance

Although aesthetically disturbing, there are no direct human health concerns associated with neoplasia in fish. Neoplasia is generally a rare event affecting one fish in several thousand. Should tumors occur more frequently in a population of fish, an indirect human health concern would be whether the cause is linked to environmental contamination.

Fibroma, connective tissue tumor

Liposarcoma on a whitefish

Neoplasia (Tumors)
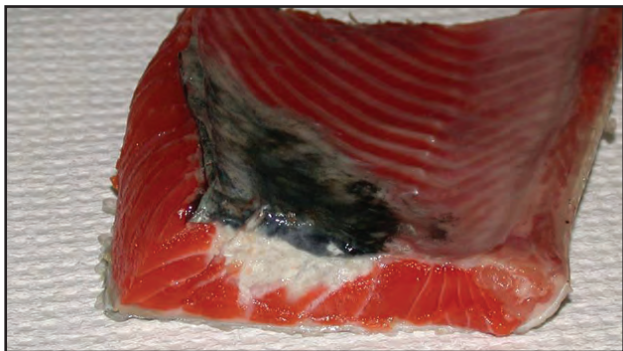

Liposarcoma in a quillback rockfish

Thymic lymphosarcoma in a sockeye salmon

Fibrosarcoma, connective tissue tumor invading muscle

Melanoma, on skin of rockfish

Papilloma in a coho salmon

Rhabdomyosarcoma in Pacific halibut

Sunburn (Back-Peel)

I. Causative Agent and Disease

Sunburn is a non-infectious disease in cultured fish caused by overexposure to ultraviolet radiation (UV) from sunlight. Certain diet ingredients causing photosensitization can be predisposing factors. Sunburn is most commonly observed during the summer months in the northern latitudes when hatchery fish are moved from an indoor rearing container to shallow outside units with very clear water. Wild fish in shallow lakes and rivers could be potentially susceptible except they rarely remain in direct sunlight long enough for overexposure.

II. Host Species

Sunburn is observed almost exclusively in cultured salmonids exposed for long periods to direct sunlight. Other fish species with small delicate scales, partial scaling or no scales at all would also be particularly susceptible.

III. Clinical Signs

Lesions from sunburn are first recognized by a darkening of the skin between the head and the dorsal fin. The epidermal layer turns white and eventually sloughs off. The underlying dermal layer of skin becomes exposed and eventually a white, craterous lesion forms. This lesion can begin with the dorsal fin that first becomes whitened and then erodes to the body surface. Any lesion from sunburn is very likely to become infected with opportunistic bacteria and/ or fungi.

IV. Transmission

Since sunburn is an environmentally mediated disease, transmission between fish does not occur.

V. Diagnosis

Sunburn is diagnosed by the observation of typical lesions with a history of lengthy exposure to sunlight.

VI. Prognosis for Host

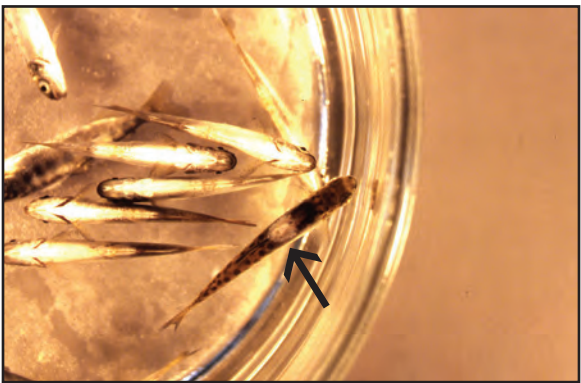When the lesions are uncomplicated by secondary infections of bacteria or fungi, the mortality is generally quite low. If shade is provided, healing of the lesions is rapid with complete recovery.

VII. Human Health Significance

There are no human health concerns associated with sunburn in fish.

Sunburn lesion on dorsal surface of coho salmon

Sunburn lesion (arrow) eroding dorsal fin of cultured Chinook salmon

Glossary of Terms

Acanthocephalans – a phylum of spiny headed worms, these parasites require two hosts for completion of the life cycle and are most commonly found in the intestines of fish.

Acid-fast – a physical property of some bacteria that are resistant to de-colorization by acids during the staining procedure.

Alevin – a newly hatched fish still having yolk sac attached.

Anadromous – relating to fish, such as salmon, that migrate up rivers from the sea to spawn in fresh water.

Anemia – deficiency of red blood cells and/or hemoglobin.

Arthropod – belonging to the phylum Arthropoda, an insect or crustacean that has a cuticle made of chitin forming an exoskeleton with segments and jointed appendages.

Ascites – the presence of fluid in the abdominal cavity.

Bacteria – any of a large group of unicellular prokaryotic organisms that lack a cell nucleus, reproduce by fission or by forming spores, and in some cases cause disease.

Basophilic – tissue components having an affinity for dye under basic pH conditions (as in histology).

Buccal cavity – cavity inside the mouth above the gill arches.

Caudal peduncle – the region of the fish body between the end of the anal fin and the base of the caudal fin.

Cercariae – free swimming larvae of digeneans; stage usually released from the gastropod first intermediate host.

Cestode – a tapeworm possessing a modified end segment called a scolex that is used for attachment. Tapeworms generally require two hosts for development.

Copepod – small planktonic crustaceans which are an important part of the aquatic food chain.

Crustacean – an arthropod having a segmented body and jointed appendages, with two pairs of antenna at some stage in their life cycle.

CPE (Cytopathic Effect) – damage to cultured cells caused by virus infection.

Cyprinid – a fish of the Cyprinidae family consisting of carps, shiners and minnows.

Cyst – a capsule of connective tissue formed by the host around a foreign body, such as a parasite, that acts as an irritant.

Cytoplasm – the fluid like substance that fills the cell, consisting of cytosol and organelles excluding the nucleus.

Diplobacilli – paired rod shaped bacterial cells.

DNA - deoxyribonucleic acid that contains genetic information for the reproduction, development and function of living organisms including some viruses.

Electron microscopy – use of an electron microscope that generates an electron beam focused through a series of objectives and lenses to create an image for

observing ultrastructural details at a much higher magnification than a traditional light microscope.

ELISA – enzyme-linked immunosorbant assay is an antigen/antibody reaction coupled with an enzyme substrate that produces a color change measured in a spectrophotometer. The test is used to detect the presence of a target organism antigen or antibody directed towards a target organism.

Endoparasitic – a parasite that lives within the body of another organism rather than on the surface.

Encyst – to enclose in a cyst.

Epizootics – an outbreak of a disease in an animal population or an unusually large increase in prevalence and/or intensity of a parasite.

Eosinophilic – a red color of cells or tissues in histological sections or stained smears that have been stained with the dye eosin.

Erythrocyte – red blood cell.

Exophthalmia – or popeye is a condition characterized by protrusion of the eyeball from the orbit.

Extracellular – outside the cell.

Epibiont – an organism that uses the body surface of another as a substrate but takes no nourishment or other benefit.

Epithelium – one or more layers of specialized cells forming the covering of most internal and external surfaces of the body and its organs.

Erythema – an abnormal red color of the skin or other tissues caused by capillary congestion.

Explant culture – tissue that is placed in a culture medium for either growth of the tissue cells or growth of an organism contained in the tissues.

Fibrotic nodules – focal areas of excess fibrous tissue formed as a reparative process or as a reaction to a foreign body.

Final host – (definitive host) the host in which a parasite develops to an adult form and reproduces.

Fluorescent antibody test (FAT) – a test using antibody against a specific pathogen that is conjugated with a fluorescein dye. The conjugated antibody sticks to the target organism causing fluorescence when viewed with a fluorescent microscope.

Fungi – heterotrophic organisms that may exist in a symbiotic, saprophytic or parasitic relationship to obtain their nutrients.

Furuncle – boil like lesion in the musculature.

Gill Lamellae – gill filaments bear many branches known as lamellae covered by a single layer of epithelium and each containing a blood capillary. Lamellae increase the surface area of the gill filaments to enhance respiration and gas exchange from ambient water.

Gram-negative rod – a rod shaped bacterium that does not retain the violet stain in a Gram stain process, but retains the counter stain and is pink in color.

Gram-positive rod – a rod-shaped bacterium that retains the crystal violet from the Gram stain process and is dark purple in color.

Granuloma – a chronic focal inflammatory lesion that walls off a foreign body

and may consist of several elements including different types of host inflammatory cells and fibroblastic connective tissue.

Hemagglutinate – the clumping together of red blood cells.

Hemorrhage – occurrence of blood within the tissues outside the normal vascular channels.

Hemostasis – the ability of an organism or cell to maintain internal equilibrium by adjusting its physiological processes.

Hemotocrits – the packed cell volume of erythrocytes in whole blood expressed as a percentage.

Histological (histology) – the microscopic anatomy of cells and tissues as viewed in thin stained sections on glass slides.

Hyphae – long branching vegetative filaments of a fungus.

Hyperplasia – an increase in the growth of cell numbers of a tissue or organ that may or may not increase in overall size; usually stimulated by an irritant.

Inflammation – a host response to tissue damage or irritation comprised of swelling, redness, heat (in warm blooded animals), pain, sometimes causing dysfunction of the tissues and organs involved.

Intracellular – inside the cell.

Intermediate host – a host in which there is development of the asexual or immature stage of a parasite.

Lethargy – a state of sluggishness or inactivity.

Macrophage – a large host white blood cell occurring in tissues and in peripheral blood that ingests foreign particles and infectious microorganisms by phagocytosis.

Melanocytes – an epidermal cell of neural crest origin capable of synthesizing the black pigment melanin.

Metacercariae – a developmental stage from encysted cercariae of digenetic trematodes generally occurring in an intermediate host.

Micropyle – the tiny opening in an egg through which a spermatozoon can enter for fertilization.

Miracidium – the ciliated larval stage of a digenetic trematode hatching from the egg which infests the first intermediate snail host.

Mycosis – fungal infection.

Myopathy – a degenerative disease of muscle.

Necropsy – a postmortem examination of an animal.

Nematode – unsegmented worm of the phylum Nematoda, having an elongated, cylindrical body; a roundworm.

Neoplasms/ neoplasia – cancer caused by uncontrolled abnormal growth of tissue cells.

Operculum – the flap on either side covering the gill chamber in bony fishes.

Opisthaptor – a posterior attachment organ in monogenetic flukes.

Organogenesis – the process in which the embryonal ectoderm, mesoderm and endoderm differentiate and develop into the in-

ternal organs of the juvenile and adult fish.

Parasite – an organism that lives on or in another organism at whose expense it obtains some advantage.

Paratenic host – an additional or optional intermediate host in which no development of the parasite occurs but a host which may serve as an essential link in the completion of the parasite's life cycle.

Pathogen – an infectious agent that can cause disease.

PCR – polymerase chain reaction is a test that amplifies targeted RNA or DNA.

Pericardium – the membrane surrounding the heart.

Peritonitis – inflammation of the lining of the abdominal cavity (peritoneum).

Petechial hemorrhage – a small focal or pinpoint hemorrhage.

Pinheading – young fish that exhibit an emaciated body due to poor feeding giving the appearance of an enlarged head.

Piscivorous – fish eating.

Plaque Forming Unit (PFU) – number of infectious virus particles per unit volume based on the number of holes or plaques in the monolayer of the infected cell culture.

Plerocercoid – the third larval stage of cestodes parasitizing fish that have an obvious scolex. Generally found in the second intermediate fish host.

Poikilothermic – animals with internal body temperatures that cannot be self regulated, often determined by the ambient temperature of the environment; cold blooded.

Proboscis – any of various elongate feeding, defensive, attachment or sensory organs of the oral region, found in certain leeches and worms.

Prognosis - a prediction of how a disease will progress, and the chance for recovery.

Protozoan – any of a large group of single-celled, usually microscopic, eukaryotic organisms, such as amoebas, ciliates, flagellates, and sporozoans.

Pycnidia – an asexual structure containing conidia, found in certain fungi.

RNA – ribonucleic acid; the nucleic acid that is used in key metabolic processes for all steps of protein synthesis in all living cells and carries the genetic information for many viruses.

Salmonid – belonging or pertaining to the family Salmonidae including salmon, trout, char, and whitefishes.

Scolex – the head segment of a cestode that attaches to its host.

Scoliosis – lateral deviation in the normally straight line of the spine.

Septicemia – presence of bacteria in the blood.

Serotype – a unique antigenic property of a bacterial cell or virus identified by serological methods.

Spore – a reproductive structure that is adapted for dispersion and survival for extended periods of time in unfavorable conditions.

Septate –divided by crosswalls or septa.

Sporangia – a single or many celled

structure from which spores or zoospores are produced in a fungus.

Teleost –the group of fishes with a bony skeleton.

Trematode – a worm with a characteristic flattened shape; these worms have complex (digenetic) life cycles involving two or three hosts.

Virulence – the pathogenicity or ability of an infectious agent to produce disease.

Viscera – internal organs of an animal.

Virus – a very small infectious agent composed of a nucleic acid core (RNA or DNA) surrounded by a protein coat that replicates only within living host cells.

Vitelline – relating to or associated with the yolk of an egg.

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Notes