The common diseases of fish can be divided into the following general categories: husbandry-related, bacterial, fungal, viral, and parasitic. The common husbandry-related disorders include trauma, environmental hypoxia, hypercarbia, temperature stress, acidic water, alkaline water, improper salinity, nutritional deficiencies, and toxicities.

Traumatic injury results in damaged or missing fins, traumatic wounds (especially on dorsum, “soreback”), opportunistic infections, and corneal edema or ulceration.

Hypoxia is caused by a low concentration of dissolved oxygen (DO) in the water. It is important to remember that the higher the temperature and higher the salinity, the lower the total amount of oxygen that will dissolve in the water. Common behavioral signs associated with hypoxia include lethargy and the congregating of fish gulping air near the air-water interface (piping behavior). A classical sign of asphyxiation is an agonal response, with the mouth open and flared opercula.

Hypercarbia is caused by increased concentration of carbon dioxide (CO$_2$) in the water. Carbon dioxide is very soluble in water and levels can far exceed the atmospheric concentration. In salmonids, chronically elevated CO$_2$ has been associated with nephrocalcinosis and systemic granuloma, a multifocal deposition of chalky, white mineral in the stomach, kidney, and epaxial muscles.

Temperature stress is caused by water temperatures outside the normal optimal temperature range for a fish. All fish are susceptible to rapid temperature changes. In general, most fish seem to tolerate a rapid drop in temperature better than an equivalent rise in temperature.

Water that is too acidic is stressful and potentially life-threatening to fish. A pH range of 6.5 – 9.0 is generally recommended for freshwater fish. Values outside this range are stressful and a pH of < 4.0 or > 11.0 is lethal. Gill tissue is the primary target of acid stress where a low pH stimulates increased mucus production, which interferes with gas and ion exchange.

Water that is too alkaline is also stressful and potentially lethal to fish. At high pH, gill mucus cells and epithelial cells become hypertrophic and corneal damage may occur.

Improper salinity results from the improper addition of salt to the water. Freshwater has less than 0.5 ppt salinity, while natural, full-strength seawater ranges from 30 to 40 ppt salinity. As much as 2 ppt salinity is probably safe for the great majority of freshwater fish. Marine aquarium fish are adapted to a narrow salinity range. It is important to note that aquarium salinity will rapidly increase because of evaporative water loss (salinity will often increase about 2 ppt (0.0005 – 0.001 specific gravity units) per week in a covered aquarium).

Signs of nutritional deficiencies in fish vary with specific deficiency, but in general one sees skeletal abnormalities, cataracts or other ophthalmic lesions, and hematopathologies (e.g.,
anemia). Although the exact nutritional requirement of aquarium fish is unknown, other signs include hepatic lipidosis, emaciation, loss of dorsal musculature, fish with big heads and small bodies, and gastrointestinal disorders. Goiters are seen occasionally in aquarium fish, especially marine fish and elasmobranchs. There is evidence that minimum iodine levels must be present in water to avoid hypothyroidism (goiter) in some marine fish. Goiter resulting from iodine deficiency has occurred in salmonids fed on all-meat diets.

Common toxicities include those caused by exposure to high levels of ammonia, nitrites, nitrates, heavy metals, and chlorine/chloramines. All of these can generally be avoided with proper attention to water quality.

Nitrogenous waste toxicities are common in aquaria with improper biofiltration. Ammonia poisoning is one of the most common water quality problems diagnosed in aquaculture. Ammonia is the primary nitrogenous waste product of fish and also originates from the decay of complex nitrogenous compounds (e.g., protein). Ammonia can cause acute mortality, but most often it presents as a sublethal stress. Chronic ammonia poisoning has been associated with hyperplasia and hypertrophy of gill tissue. Ammonia is present in two forms: unionized (\(\text{NH}_3\)) and ionized (\(\text{NH}_4^+\)). Unionized ammonia (UIA) is toxic to fish, while ammonium (\(\text{NH}_4^+\)) is much less toxic. The amount of UIA in water depends mainly upon the pH, and also on temperature and salinity. High pH and temperature and low salinity favor the presence of UIA. Most circumstances causing ammonia poisoning can also lead to nitrite poisoning. Nitrite is actively transported across the gill, where it enters the bloodstream and oxidizes hemoglobin (Hb) to methemoglobin (MetHb) resulting in clinical signs of hypoxia, pale tan or brown gills, and brown blood. Nitrate, the end product of nitrite oxidation, is less toxic; however high levels can add to stress.

Metals are most toxic in low-alkalinity water, which allows a high concentration of metal to remain dissolved (and thus toxic). Lead, copper, or galvanized (zinc-coated) iron plumbing may leach metals. Signs of lead toxicity include sigmoid spinal curvature of the spine, caudal cutaneous melanosis, stippled basophilia of erythrocytes, and neurologic disorder. Iron toxicity results from precipitating iron (ferric hydroxide) on gills resulting in impairment of respiration. Zinc toxicity results in neurologic disorders.

Chlorine toxicity results in an acute to subacute mortality associated with fish added to a newly set-up tank or when fresh tap water is used for a water change. Chronic chlorine exposure may result in extensive mucous secretion and hypertrophy of the gill epithelium.

Lateral line depigmentation (LLD), previously referred to under a number of terms including hole-in-the-head syndrome and lateral line erosion (disease) is a chronic dermatopathy affecting mainly certain species of tropical freshwater and marine aquarium fish. This chronic, idiopathic syndrome presents as mild to severe, focally depigmented skin along the lateral line of the head and/or flank. Lesions usually begin on the head as shallow, pinpoint foci that expand in size, depth, and surface area. Advanced lesions may be deep ulcerations. The exact etiology of this condition is unknown and may be the result of a variety of factors. Suggested possible etiologies include inadequate nutrition, inadequate water quality, intestinal diplomonad flagellates.

Gas supersaturation in fish occurs when the total pressure of gases dissolved in water is higher than the ambient atmospheric pressure. This occurs with a rapid increase of water temperature
from water source to fish system; water intake pipe sucking in air; long pipe run; rapid decrease in pressure from water source to fish system; water falling over a deep spillway; ground water (borehole or spring) source; heated water; hydro power; ice formation; and heavy macrophyte growth in clear pond. Gas emboli forms in the blood vessels of virtually any organ, including skin, gills, eyes, viscera, and peritoneal cavity - exophthalmos caused by retrobulbar gas emboli and emphysema in dermis. Most gas emboli are produced by excess nitrogen because oxygen is assimilated metabolically and less likely to form persistent bubbles. Acute disease (high gas saturation) is associated with high mortality. Chronic disease (lower gas saturation) is associated with hyperinflation of the swim bladder and emboli in the intestines.

Bacterial diseases are common in aquarium fish. Most are opportunistic pathogens involving Gram-negative aerobes and facultative anaerobes. Clinical signs of Gram negative septicemia are nonspecific and include lethargy, anorexia, cutaneous hemorrhages and ulcers, fin rot, ascites (“Dropsey”), exophthalmia, and color changes. A post-mortem diagnosis of bacteremia or septicemia is based on kidney culture. Samples should be submitted to a laboratory that is familiar with culturing bacteria from aquatic species because many aquatic pathogens have special requirements. It is best to culture fish isolates at room temperature (22 – 25 °C), not 37 °C, as is routinely done in commercial microbiology labs, because some fish pathogens grow poorly or not at all at 37 °C. Samples from marine fish should be cultured on a medium that has a high salt content (e.g., trypticase soy agar with 2% NaCl) or on a nutrient-rich blood agar, such as Columbia agar with 5% defibrinated sheep blood (CBA). CBA or similar nutrient-rich blood agar is a good general-purpose medium for both freshwater and marine bacterial pathogens.

*Aeromonas salmonicida* (4 subspecies: *salmonicida, achromogenes, masoucida and smithia*) is a common fish pathogen as are several other species of *Aeromonas*, including: *A. hydrophila, A. formicans, A. liquefaciens, and A. hydrophila* complex that are capable of causing a disease known as “Motile Aeromonas Septicemia” or “Bacterial Hemorrhagic Septicemia”. The bacterial pathogens are opportunists that result in ulcerative dermatitis in freshwater fish (especially koi, goldfish and other cyprinids) and septicemia in cold water marine fish (predominantly salmonid fishes). “Furunculosis” is derived from the presence of “blisters” or furuncules on the surface of chronically infected fish. Significant resistance to both tetracycline and sulfamerazine occur with these bacteria.

*Vibrio* spp. are primarily pathogens of marine fish. They cause acute and chronic disorders with lesions such as cutaneous ulcers with muscle necrosis, septicemia, and enteritis. Important species of marine tropicals include *V. damsela, V. alglnolyticus, and V. anquillarum*.

*Edwardsiella* is another important bacterial pathogen of fish. Emphysematous putrefactive disease, EPD, is caused by *E. tarda* and Enteric septicemia of catfish, ESC, is caused by *E. ictaluri*.

*Pasteurella piscicida* causes a hemorrhagic septicemia in marine fish and lesions similar to those caused by vibrios.

*Renibacterium salmonarium* is an important pathogen of salmonids. Gross findings in affected fish include muscular cavitation; vesicular dermatitis (Spawning rash); necrotizing nephritis and splenitis. Histopathological findings include granulomatous and necrotizing nephritis, splenitis,
hepatitis, endocarditis and pancreatitis; occasionally with giant cell formation where histiocytes are expanded by intracytoplasmic gram-positive bacteria.

_Yersinia ruckeri_, the causative agent for Enteric Redmouth Disease, is an important pathogen of salmonids, but does affect other species as well. Clinical presentation includes blind fish with dark discoloration that cannot find food. It causes a typical hemorrhagic septicemia - especially dark coloration, exophthalmos, hemorrhage in mouth and eyes, depression, and swollen abdomen. The disease does vary from other bacterial septicemias in that the reddened skin is mainly on the head or mouth, especially the lower jaw. Histologically, there is bacterial colonization of well-vascularized tissues, causing hemorrhage and/or telangiectasis of gills, kidney, liver, spleen, and heart, as well as muscle. This leads to necrosis of the hematopoietic tissue, causing anemia. There is also necrosis and sloughing of the gastrointestinal tract.

Clinical findings associated with _Streptococcus_ spp. infections include ocular hemorrhage, cutaneous hemorrhage, exophthalmia, corneal opacity, dropsy and ulceration. Gross findings on necropsy reveal abdominal serosanguinous fluid, splenomegaly, hepatic pallor, endocarditis and nephritis. _Streptococcus_ spp. often infect the brain and nervous system of fish, explaining the erratic swimming frequently observed in infected fish. Isolated streptococcal pathogens in fish include _S. iniae_ _S. difficilis_ _S. parauberis_ _S. milleri_ _S. shiloi_.

Acid-fast bacterial infection in fish commonly involve _Mycobacterium marinum_ (marine fish), _Mycobacterium chelonae, Mycobacterium fortuitum_ (freshwater fish), and _Nocardia kampachi_. Mycobacterial infections are the most common chronic disease that affects aquarium fish - virtually all freshwater and marine aquarium fish- becoming a problem in research fish collections. Clinical signs include chronic wasting and granulomatous lesions. In the coelomic cavity, lesions common found include granulomatous, multifocal, severe peritonitis, hepatitis, splenitis. Histopathology reveals a chronic inflammatory response with epithelioid macrophages surrounding the bacteria. Lesions often have necrotic centers and may have melanomacrophages or melanocytes. Bacteria are typically located in the center of the inflammatory focus.

Columnaris infection results in necrotic ulcers or growths on skin, fins, or gills of freshwater and marine fish. A rapid, presumptive identification of the etiologic agent, _Flavobacterium columnare_ (formerly, _Flexibacter columnaris_), can be made by examining wet mounts of lesions revealing long, thin rods with a characteristic flexing or gliding motion, that tend to aggregate.

True fungi infections, like _Fusarium_, are rare in fish and are likely the result of chronic immunosuppression resulting from poor water quality, stress, and mis-use of antibiotics. _Saprolegniasis_ is caused by a water mold resulting in a white, fuzzy growth on the skin of affected fish.

_Ichthyophonus hoferi_ is a fungus-like agent that causes a chronic systemic, granulomatous disease. It is endemic in many feral, cold water marine fish populations and has been reported in over 80 species of marine fish. White or dark (pigmented) nodules occur in highly vascularized organs, such as heart, spleen, kidney, and liver. Thick - walled spores (flask-shaped, with a neck
that consists of a hypha that breaks through the outer wall) surrounded by granulomatous inflammation occur in internal organs.

There are hundreds of known viruses that affect fish. The DNA viruses include Herpesvirus, Iridovirus, Adenovirus and the RNA viruses include Picornavirus, Paramyxovirus, Reovirus, Togavirus, Birnavirus, Rhabdovirus, and Retrovirus.

Lymphocystis, a DNA Iridovirus, causes large wart-like tumourous growths in freshwater or marine fish. The markedly hypertrophic dermal fibroblasts (lymphocystis cells) may measure > 300 µm with vacuolated to granular pale basophilic cytoplasm surrounded by a 10-30µm amphophilic hyaline wall. Occasionally there is the presence of basophilic fibrillar inclusion material. This generally considered a self-limiting disease, but infected fish should be isolated from others to prevent spread of the virus.

Carp pox is caused by a cyprinid herpesvirus (CyHV-1). Lesions are typically smooth to rough, milky white to grey plaques up to 2 mm thick. The virus causes scarring, retarded growth, skeletal deformities, and hyperplastic epithelium (may be papillomatous). Intracytoplasmic and intranuclear (Cowdry type A) inclusions are found in the plaques that measure up to several cm along longest dimension. The lesions eventually slough but can last for months and may become dark pigmented; therefore, reducing the value of the fish. The lesions develop in low temperatures (winter/spring) and regress with high temperature (summer) but a latent infection remains with transmission from wounds and causing acute disease in young fish.

Koi herpesvirus (KHV, CyHV-3) is a highly contagious viral disease (via water, feces, or direct contact) that may cause significant morbidity and mortality in common carp (Cyprinus carpio). This is a serious emerging disease that causes major losses in koi and mass mortalities in common carp. Clinical signs include bleeding gills that are pale, swollen, and mottled, dyspnea, abnormal skin coloration (pale patches and blisters), and sunken eyes. The white patches are due to necrosis (death) of the gill tissue. Gill lesions caused by KHV disease are the most common clinical signs in affected koi. Histopathology reveals massive proliferation, degeneration, and necrosis of gill epithelium in the absence of another etiological agent (parasite or bacterium) which is strongly suggestive of KHVD.

The Channel catfish viral disease is caused by a species-specific herpesvirus, IcHV-1. It is the most important viral disease affecting channel catfish and occurs exclusively in young (< 1 year) and small (< 15 cm) fish. Most epidemics are in fish < 4 months old. Mortalities are most rapid and severe with higher temperatures, being highest at 25 –30 º C (77 – 86 º F). The virus attacks all major organ systems and results in yellowish coelomic fluid and punctate hemorrhage in the viscera.
Viral hemorrhagic septicemia (VHS) affects over 50 species of fish (freshwater and marine) and is caused by multiple strains of viruses in the Family *Rhabdoviridae*. Gross lesions include petechial hemorrhage (visceral, cutaneous, and muscle), exophthalmus, ascites (dropsy), and eccymoses around eyes, skin, gills and fins. Histopathology finding reveal variable necrosis of kidneys (hematopoietic), spleen, liver, and skeletal muscle because the virus infects endothelial cells, hematopoietic tissue, and leukocytes. Like other rhabdoviruses, there is impairment of osmoregulation, resulting in edema and hemorrhage. While hemorrhage is the major clinical feature of VHS, degeneration and necrosis are the most common histopathological findings with the kidney as the prime target where most of the damage is to the hematopoietic tissue. Liver necrosis and degeneration (vacuolation) is also common. The hemogram shows anemia, leukopenia, and thrombocytopenia. This disease is a major cause of mortality of freshwater salmonids (Rainbow and Brown trout).

Typical clinical presentation of Infectious Pancreatic Necrosis (caused by an Aquabirnavirus) is a sudden increase in mortality of fry or fingerling trout, with larger, more robust fish dying first. Older fingerling trout may exhibit many petechial hemorrhages in the viscera. Affected fish exhibit neurological signs, trailing white feces, dorsal darkening, coelomic distention, exophthalmia, and pale, hemorrhagic gills. A catarrhal exudate in the stomach and intestine produces the mucoid, cohesive fecal pseudocast.

Spring viremia of carp (SVC) is caused by *Rhabdovirus carpio*. The nonspecific clinical signs include darkened skin, dropsy, and exophthalmia. Gross necropsy reveals small hemorrhages throughout the body, ascites, coelomitis, enteritis, and edema of internal organs. The swim bladder is significantly affected, with the epithelial monolayer becoming multilayered and the submucosal blood vessels dilated and inflamed. There is also necrosis in the liver, hematopoietic tissue (kidney, spleen) and intestine. There is necrotic debris in the renal tubules and sloughing epithelium. Infected Purkinje cells in the brain cortex may have eosinophilic inclusions, which is especially diagnostic. The appropriate local fish health authority should be notified if SVC is suspected.

Protozoal infections are common fish diseases of fish. Most protozoa are harmless commensals with a direct life cycle. Captivity increases susceptibility to infection in fish by causing immunosuppression and high host population densities.

*Ichthyophthirius*, the agent that causes “Ich” in freshwater fish and its marine counterpart, *Cryptocaryon* are common ciliate pathogens of fish. Clinical signs include flashing, restlessness, respiratory distress, white spots (theront), and death. The infection is identified by the presence of round, ciliated trophozoites with a U-shaped nucleus and slow rolling motion on wet mount preparations. Pathogenesis occurs as the ectoparasites feed on host cells by burrowing into epidermis causing excessive mucus production. These organisms have resistant cyst that are difficult to remove. Gross lesions include up to 1 mm elevated nodules in a mililiary pattern along the body, gills and fins that may coalesce or progress to erosion and ulceration due to irritation. Histopathological findings reveal oval holotrich ciliated parasite which forms intraepithelial cysts composed of a 1-2 µm hyaline wall. Small circular oral opening, dark granules and a horseshoe/crescent shaped basophilic macronucleus. There may or may not be hyperplasia of neighboring epithelium.
*Trichodina* is another common ciliate protozoal parasite of both freshwater and marine fish. It is identified as a flattened, circular, ciliated protozoan with denticular rings. Clinical signs associated with this parasite include irregular swimming, flashing, respiratory distress, and skin disorders.

*Chilodonella*, a pathogen of freshwater fish and its marine counterpart, *Brooklynelia* are oval, flattened, heart-shaped protozoa with cilia lined in rows causing them to move in a characteristic slow circular movement. Clinical signs associated with these parasites include respiratory distress (caused by excessive mucus and gill hemorrhage), clamped fins, depression, and sudden death.

*Tetrahymena* is a pathogen of freshwater fish and its marine counterpart is *Uronema*. Both are identified on wet mount preparations as pear-shaped, actively motile ciliates. Clinical signs associated with these protozoans include discolored areas on skin surrounded by hemorrhage. These are free-living infusorians that become secondary pathogen. They are highly invasive, can be found in internal organs, and are difficult to treat.

*Epistylis* is a bell-shaped ciliate protozoal parasite of freshwater fish that has apical cilia and a body that contracts on a coiled stalk. Affected fish present with white tufts on the surface of the skin or fins that resemble a fungal lesion. The organism is normally found attached to vegetation or crustaceans associated with water with high organic content.

*Ichthyobodo* (formerly known as *Costia*) is a flagellate parasite of freshwater and marine fish. On wet mount preparations, it appears as small, actively motile, comma-shaped flagellates that move in a circular motion when attached. Clinical signs associated with this parasite include irregular swimming, flashing, inappetence, and death. The organism attaches to the skin and gills and feeds on cells causing hyperplasia of epithelium and destruction of goblet cells. It is one of the smallest ectoparasites that infest fish (about the size of a red blood cell). *Ichthyobodo* is especially dangerous to young fish and can attack healthy fry and even eggs. In older fish it is associated with some type of predisposing stress.

Amyloodiniosis is one of the most important diseases of warm water marine fish, infesting both food fish and aquarium fish worldwide. *Amyloodinium ocellatum* can infest both elasmobranchs and teleosts. The trophont bears little resemblance to free-living dinoflagellates. Typical dinoflagellate morphology is apparent only during the disseminative (dinospore) stage. The life cycle is virtually identical to that of *Ichthyophtherus* where the trophont attaches to and feeds on the host’s epithelium – after several days, it detaches from the host, retracts its rhizoids (root-like structures used to attach to the epithelium), and becomes a tomont. The tomont divides, producing numerous motile, infective dinospores (8–13.5 µm long by 10–12.5 µm wide). The dinospores attach to a host, differentiate into a trophont, and continue the cycle. *Piscinoodinium* is the freshwater analogue of *Amyloodinium*. Clinically, this organism causes “velvet disease” or “rust disease,” because of the sheen produced by its chlorophyll. It is known to destroy gill lamellae.
Hexamita and Spironucleus are gastrointestinal flagellate protozoans of freshwater and marine fish. Massive infestations are likely caused by Spironucleus. Clinical signs include anorexia, lethargy, and death.

Trypanosomes are flagellate hemoparasites with unknown pathogenesis as most fish harboring this parasite are asymptomatic. They appear as incidental finding in blood films and tissue imprints (kidneys).

Apicomplexa are intracellular parasites that produce spores. The hemogregarines and piroplasms have indirect life cycles and are found in blood. Coccidia have direct life cycles. The most important fish coccida pathogens affect solid tissues and virtually all tissue coccidians that infect fish belong to the family Eimeriidae (over 200 species). Species of Cryptosporidium are not serious fish pathogens. Piscine coccidia are less species-specific compared to those of mammals and often infect several closely related fish species of the same genus. They generally have low pathogenicity in intestines and affected fish exhibit weight loss and yellow fluid feces.

Micromonogamia parasites of fish include Glugea, Ichthyosporidium, Loma, Pseudoloma, Microgemma, Microsporidium Mrazekia, Nosema, Pleistophora, Spraguea, Tetramicra, and Theragra. Microsporidians (class Microsporidia) are not as common as myxozoans, but they are responsible for a number of serious diseases in cultured fish resulting in high morbidity/mortality or reduced market value from damaged muscle, such as “Neon tetra disease” and “Skinny disease of Zebra fish.” They are often host-specific (infecting only one fish species or a closely related group); however, some species, such as Pleistophora hyphessobryconis, Ovipleistophora mirandellae, Glugea stephani, Glugea anomala, Loma salmonae can infect a broad range of fish. These are small (<20um), unicellular, intracellular, obligate parasites with one spindle-shaped polar capsule within their spores. Glugea hiraldi causes “White spot disease” of sea horses.

Myxosporidia infections in fish are pathogenic with the intracellular organisms involving many organs. There are many genera that cause nodules in skin and gills and resemble microsporidia, except they have two spindle-shaped polar capsules within their spores. All require an intermediate host. Virtually all Myxozoa that infect fish are members of the class Myxosporea, with two (one in carp and one causing Proliferative Kidney Disease (PKD)) being members of the class Malacosporea. Myxozoans are obligate parasites of tissues (histozoic forms that reside in intercellular spaces or blood vessels or reside intracellularly) or organ cavities (coelozoic forms that live primarily in the gall bladder, swim bladder, or urinary bladder). Hoferellus carassii, formerly known as Mitraspora cyprinid, of goldfish and koi may result in severely bloated fish that live for months and enlarged kidneys at necropsy. Most are intercellular parasites that are typically site specific, infecting only certain target organs, and taxonomically specific, usually infecting only one species or a closely related group. However, some have a broad host range. They are characterized by the development of a multicellular spore, presence of polar capsules in their spores, and endogenous cell cleavage in both the trophozoite and sporogony stages. Most myxozoan infections of fish are relatively innocuous, eliciting only moderate host reactions, but heavy infections can be quite serious. Histological diagnosis is made by identifying the spores, which are refractile and difficult to see in hematoxylin and eosin sections, but polar capsules stain intensely with Giemsa or toluidine blue. Proliferative gill disease (PGD), due to infection by Henneguya ictaluri, causes acute branchitis and low-to-high mortality (1% to 95%) in all ages of (but primarily fingerling) channel catfish. Also known as “Hamburger gill
disease” because the gill lamellae become pale and swollen gill lamellae that later become thickened, blunted, and bleed easily. Histopathology: swollen, clubbed, and broken lamellae with inflammatory response and trophozoite stage in the gill parenchyma.

Myxobus cerebralis causes whirling disease of salmonids characterized by whirling behavior, mis-shaped head, jaws, and spines, black tail, and death. Gross findings include severe scoliosis, frequently with black discoloration of the tail, and with chronicity, misshapen head and jaws. Histopathological findings reveal cartilage degeneration and necrosis, granulomatous chondritis with intralesional ovoid to ellipsoidal spores (5-15µm) with 2 piriform-shaped polar capsules at the anterior end. Because the spores are trapped in cartilage, it is difficult to make a diagnosis via wet mounts of fresh tissues. Therefore, a histopathologic diagnosis is usually from head, gill, or vertebral cartilage. To sample for Myxobolus cerebralis, a cross-section should be taken just behind the eye (approximately 5 mm posterior), so that the cartilage around the auditory capsule is included (a common site for M. cerebralis infection).

Trematodes can infest both freshwater or marine fish. Monogeneans have haptor or attachment organs, a direct life cycle, and live on skin, gills, and fins. Digeneans are endoparasites with an indirect life cycle where the adults with two suckers and y-shaped gut live in gastrointestinal tract of a piscivorous bird or mammal.

Monogeneans are common parasites of the skin and gills of both marine and freshwater fish and are often called skin fluke, gill fluke, or eye fluke. The two types of monogeneans, differ based upon their opisthohaptor morphology. Monopisthocotylea (e.g. dactylogyrids, gyrodactylids, capsalids) are the most common. They have a single unit comprising several, large, centrally located, sclerotized anchors (hooks) and often small marginal hooklets that they use to pierce tissue when they attach. They gave a direct lifecycle and parasite numbers have been correlated with mortality. The most economically important monogeneans in cultured and aquarium fish are in the monopisthocotylean Superfamilies Gyrodactyloidea and Dactylogyroidea. The oviparous dactylogyrids are primarily gill parasites of freshwater fish. Gyrodactylus when viewed under the microscope has 2 points at anterior end, anterior sucker, no eyespots, a haptor with 2 large hooks surrounded by small hooklets, and are viviparous, often revealing embryos with hooks. Dactylogyrus is identified by 4 points at anterior end, anterior sucker, 4 eyespots, and a haptor with 2 hooks surrounded by small hooklets.

Digenean trematode infections are common, usually asymptomatic infections in wild fish. About 1,700 species of adult digeneans infect fish. Metacercariae are more common than adults. Adult digeneans mostly inhabit the gastrointestinal tract (rarely infecting the swim bladder, ovary, peritoneal cavity, urinary bladder, or circulatory system). All but the hemoparasites and gill digeneans are an incidental finding. Metacercariae can be found in virtually any tissue, depending on the infecting digenean species. Metacercariae are usually innocuous and fish can carry amazingly high worm burdens without any apparent ill effects, except for the gill digeneans which can cause significant morbidity and mortality in wild and cultured fish. Cryptocotyle, the causative agent for black spot in marine fish, has cercariae that shed from a
snail and penetrate the skin of fish where black spots occur around the encysted metacercaria because the host produces melanin pigment around cyst.

Turbellarians are free-living worms related to trematodes. The best known is the tang turbellarian that causes black ich as a result of a hypermelanization reaction resulting in dark foci on the skin. The lesions are similar to those of digenean metacercariae.

Cestodes (tapeworms) infestations in fish are generally nonpathogenic. Adult worms are found in intestine or larvae in peritoneal cavity, liver, or muscle. Clinical signs of emaciation occur with heavy worm burdens; otherwise, usually asymptomatic. One of the most serious adult cestodes that affect fish is the Asian tapeworm, *Bothriocephalus acheilognathi* (formerly known as *B. gowkongensis*), has an unusually wide and currently expanding host range (including minnows, golden shiner, various carp species, channel catfish, and possibly aquarium fish).

Fish are either intermediate or definitive hosts for nematodes. About 650 species of nematodes parasitize fish as adults and many others use fish as intermediate hosts. Nematodes are common in wild fish, but neither adult nor larval forms are usually a problem in cultured fish because of the absence of intermediate hosts in the life cycle. Nematodes infecting aquarium fish might have a direct life cycle. Adults are almost always found in the digestive tract and when they occur in high numbers (e.g., *Capillaria*), they can cause chronic wasting.

Acanthocephala or Thorny headed worms are rare in cultured and aquarium fish. Acanthocephalans (around 400 species affecting fish) have a complex life cycle that requires one or two intermediate hosts. Fish may be intermediate or final hosts, depending on the acanthocephalan species.

The common ectoparasitic crustaceans of fish are either branchiurans, those with dorsoventrally flattened body and a prehensile sucker to attach to fish, or copepods, those with a diversity of body form, fused body segments, and variable appendages. Parasitic copepods are increasingly serious problems in cultured fish and can also impact wild populations. Sea lice have become a serious problem in all major salmon-producing regions of the northern hemisphere. Most affect marine fish where they are found on the skin and gills. Ergasilids (family Ergasilidae) primarily are found in the gills of freshwater fish. Lernaeopodids (family Lernaeopodidae) are mainly parasites of marine fish. Anchor worm is a general term for species of highly modified copepods, Lernaeids (*Lernaea* is the most important genus), that possess anchor-like processes for securing themselves to freshwater fish. Treatment in aquarium fish involves either manual removal of the parasite or use of an organophosphate.

Accepted methods for humane euthanasia of fish include use of tricaine, benzocaine, 2–phenoxyethanol, barbiturates – sodium pentobarbital, inhalant anesthetics, and carbon dioxide.

Neoplasia is occasionally seen in aquarium species, especially goldfish and geriatric fish. Any tissue can be affected. Histopathologic criteria for malignancy in mammals does not always apply to fish and metastasis is rare. Common neoplasia of carp, goldfish, koi include lymphoma, neurilemmoma/ neurofibroma, schwannoma, fibrosarcoma, pigment cell tumors, especially melanoma, thyroid adenoma/ adenocarcinomas, hepatoma/ hepatocarcinoma, and testicular adenoma.
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