3.2.10 Monogenean Diseases

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Monogenea are ubiquitous parasites of the body surface (skin, fins, and gills) of many freshwater fishes. Three groups are known to have the potential to cause disease in fishes of economic importance in North America: (1) *Gyrodactylus* spp., (2) ancyrocephalids including *Ligicetaluridus* spp. and *Cleidodiscus* spp., and (3) dactylogyrids including *Dactylogyrus* spp. All of these worms are small (generally 1 mm or less) and host specific. Each species of parasite will generally only parasitize one host species (or closely related group of hosts).

I. *Gyrodactylus* spp.

A. Name of Disease and Etiological Agent

Gyrodactyliasis is caused by *Gyrodactylus* spp. (*Platyhelminthes:Monogenea*).

B. Known Geographical Range and Host Species of the Disease

1. Geographical Range

*Gyrodactylus* spp. occurs on freshwater fishes throughout North America. These worms have, in fact, been reported from every continent except Antarctica.

Although early reports of *Gyrodactylus* spp. in salmonid culture facilities in North America implicated these parasites as the cause of considerable mortality, and gyrodactyliasis has been listed as a "threatening" disease of the salmonids in the Pacific region. There have been almost no recent records of gyrodactyliasis, apart from brief mention of "problems" on Vancouver Island and in Washington State. This situation probably reflects the efficacy of currently used treatments rather than an absence of these worms. The closely related *Laminiscus strelkowi* (Family *Gyrodactylidae*) has been detected in large numbers on the gills of salmonids reared in Pacific netpens. However, it is still unclear as to the precise role that this parasite may play in disease.
2. **Host Species**

Most salmonid species are susceptible to infection by one or more species of *Gyrodactylus*. The following are of interest in the context of disease: coho salmon *Oncorhynchus kisutch*, sockeye salmon *Oncorhynchus nerka*, rainbow trout *Oncorhynchus mykiss*, Atlantic salmon *Salmo salar*, brown trout *Salmo trutta*, and salmonid hybrids e.g. splake *Salvelinus fontinalis* (brook trout) x *Salvelinus namaycush* (lake trout).

Ictalurid (catfish) species including channel catfish *Ictalurus punctatus* are also susceptible.

Cyprinids including goldfish *Carassius auratus*, common carp *Cyprinus carpio*, grass carp *Ctenopharyngodon idella*, and golden shiner *Notemigonus crysoleucas* are also susceptible.

C. **Epizootiology**

The only documented, overt disease in wild salmonids involves *Gyrodactylus salaris* on *Salmo salar* in Norwegian rivers. It is of interest that in brook trout fry, experimentally infected with *Gyrodactylus salmonis*, a mortality of 44% was observed over 22 days. Larger fish were less susceptible. Both channel catfish and golden shiner in the southeastern U. S. and in Illinois, respectively, have been adversely affected by *Gyrodactylus* spp.

Carp fry and fingerlings can be killed by severe outbreaks of gyrodactyliasis. While most information comes from Eastern Europe and the Commonwealth of Independent States (C. I. S.), there are reports of "problems" in the southeastern U. S. A.

1. **Conditions Under Which Disease Occurs**

*Gyrodactylus* spp. is viviparous (i.e. producing live young) and hermaphroditic (i.e. have both female and male reproductive organs). Thus, in a population all adult worms may carry a fully developed embryo which in turn, while still in the parental uterus, carry young of the next generation. Thus, *Gyrodactylus* populations can multiply extremely quickly. The finding of *Gyrodactylus* on cultured fishes (especially salmonids) should always be a reminder of their disease potential. This is particularly true under conditions where fish are stressed.

Although fish of all ages may be parasitized, young fish appear to be the most affected. The number of worms infecting an individual (i.e. intensity) generally decreases with increased host age. Overcrowding, especially of young fish, often leads to increased intensities of infection, which may or may not produce signs of disease.

Secondary infections by bacteria and/or fungi have often been considered part of the cause of mortality associated with gyrodactyliasis. However, there is no experimental evidence from observations in North America to support these hypotheses, although in Norway, the fungus *Saprolegnia* seems to be an established secondary pathogen.

With the scanty evidence available, it appears that on salmonid fishes, *Gyrodactylus* spp. is most abundant at cooler times of the year (winter and early spring) when water temperatures are 8°C or less. Warm-water species have higher optimum temperatures, and the parasites are usually well adapted to the temperature ranges tolerated by their hosts.

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D. Disease Signs

1. Behavioral Changes Associated with the Disease
   Some fish (e.g. rainbow trout) with moderate or heavy infections of skin-dwelling *Gyrodactylus* spp. may flash, turning sideways as if attempting to rub away the irritation. Under experimental conditions, intensely infected brook trout fry were observed to be weakened and lethargic before becoming moribund.

2. External Gross Signs (in heavy infections)
   *Gyrodactylus* spp. are site specific as well as host specific parasites. In salmonids and cyprinids, the general body surface (skin, fins, and/or tail) is affected while in catfish the barbels, underside of the head, and fins are particularly prone to attack. In heavily infected cyprinids, the gills may become affected as well as the body surface.

External gross signs in heavy infections include overall darkening in color in fry, erosion of the fins (particularly the dorsal fin), pale discolored flanks, and thickened cuticle, obvious secretions of mucus sometimes described as a blue/grey slime, and emaciation (especially in young fish). In catfish detachment of the barbels due to necrosis may occur.

3. Internal Gross Signs
   None reported.

4. Histopathological Changes Associated with the Disease
   *Gyrodactylus* spp. are tissue browsers, feeding on the epidermis (skin) of the body surface, fins, or gills. Mechanical damage may also be inflicted by the attachment apparatus (haptor) of the parasites. Sections of skin from heavily infected fish show epidermal hyperplasia with zones of degeneration and necrosis.

   Extensive kidney damage, including degeneration and necrosis of the epithelium lining of the renal tubules, was observed in experimentally infected brook trout fry. These fish were heavily infected and had become moribund. The renal pathologies were thought to have been associated with epidermal disruption, caused by the "grazing" of parasites, leading to osmotic imbalance with both inflow of surrounding water and loss of tissue fluids.

E. Disease Diagnostic Procedures

1. Presumptive Diagnosis
   a. Isolation and Detection of Pathogen
      With heavy infections of small fish (e.g. salmonid fry), it is possible to see *Monogenea* on the body surface of a dead or anaesthetized fish in a dish of water using the low power of a dissecting microscope or a 10X magnifying glass. A preliminary identification (to decide which group of *Monogenea* is present) can be made using a temporary preparation. Put a skin scrape or gill filament squash on a microscope slide and add a small drop of water and cover with a coverslip. The worms can then be examined under a compound microscope.

      In order to identify worms to genus (see Figure 1), it is necessary to collect and preserve specimens and make a more permanent preparation for examination under the high-dry

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objective (40X) of a compound microscope. Pritchard and Kruse (1982) have described the following techniques: small *Monogenea* are easily collected by placing infected material (whole small fish or tissue from larger fish e.g. gills, fins, barbels, etc.) in a container of 1:4,000 formalin for 15 to 45 minutes; shake the container vigorously for 2 to 3 minutes and remove fish remains; pour the resultant "soup" into a tall vessel and allow worms to settle for about 30 minutes. Decant supernatant fluid and examine sediment by placing small amounts in a Petri dish in order to scan under a dissecting microscope. Remove any specimens with a pipette and either transfer them to a clean slide for immediate examination (in a drop of water under a cover slip) or into fixative (AFA-alcohol-formalin-acetic acid or 5% formalin) pending the making of more permanent preparations. The worms in the temporary wet preparation should be adequate for identification to genus with reference to the diagrams provided in this manual (see Figure 1).

b. Clinical Signs
As described above

c. Histopathological Examination
Is not necessary for diagnostic purposes.

2. Confirmatory Diagnosis
To identify *Monogenea* to the species level, it is necessary to have a compound microscope, preferably fitted with a 100X oil-immersion lens and a measuring device. It would usually be preferable to send material requiring specific identification to an appropriate diagnostic center or specialist, who would have access to the relevant literature.

F. Procedures for Detecting Subclinical Infections
Subclinical infections are probably universal in salmonid hatchery and culture facilities and may also occur where non-salmonids are reared. A sample of fish can either be killed and examined as described above, or worms can be collected from living fish as described by Cone and Cusack (1988): a sample of fish is placed in a plastic net box containing 1:4000 formalin solution (the size of the net box and volume of formalin solution would vary with the size of the sample; for 60 yearlings four liters of solution is appropriate) and the fish allowed to thrash about for two minutes. After removal of the fish, worms can be concentrated by sedimentation and processed as described above.

G. Procedures for Determining Prior Exposure to *Gyrodactylus* spp.
None available for Monogenean diseases.

H. Procedures for Transportation and Storage of Samples to Ensure Maximum Viability and Survival of the Etiological Agent
Although, under ideal conditions, small *Monogenea* can be more easily studied while alive, this is usually not practical for diagnostic work. Living worms can survive on whole, small fishes or gill
material for about 6 hours if kept in a small quantity of chilled water in a cooled container. However, fixation is generally preferable.

Small fishes can be fixed whole in a 5% formalin solution for 48 hours, and then transferred for shipping and/or storage to 70% ethanol. Similarly, tissues from larger fishes (fins, barbels, gills, etc.) can be fixed and shipped separately.

Figure 1. Generalized *Gyrodactylus* sp.
II. Ancyrocephalids - Including *Ligictaluridus* spp. and *Cleidodiscus* spp.

A. Name of Disease and Etiological Agent

"Monogenean Disease" is an acceptable, general term. *Ligictaluridus* spp. (= *Cleidodiscus*, in part, of previous texts) and *Cleidodiscus* spp. are both in the family *Ancyrocephalidae* (*Platyhelminthes* : *Monogenea*).

B. Known Geographical Range and Host Species of the Disease

1. **Geographical Range**
   *Ligictaluridus* spp. are enzootic to North America, occurring only on ictalurid fishes. Where catfish have been introduced into other countries (e.g. Poland and the C. I. S.), these parasites were introduced at the same time. *Cleidodiscus* spp. are found on fishes in North America and in Eurasia.

2. **Host Species**
   Channel catfish *Ictalurus punctatus* are parasitized by *Ligictaluridus* spp., and bluegill *Lepomis macrochirus* by *Cleidodiscus* sp.

   Parasitized wild fish have been found in many areas throughout the natural ranges of both hosts with no records of disease. However, under culture conditions, problems are known to occur.

C. Epizootiology

1. **Conditions Under Which Disease Occurs**
   Ancyrocephalids, in contrast to *Gyrodactylus* spp., are oviparous (i.e. they do not give birth to live young). Shelled eggs are laid and development of the embryos is external to the body of the worm. The period of development is temperature-dependent, e.g. at 20°C, and generation time is 14 days. As temperatures increase (to an optimum level), generation time becomes less. On hatching, the oncomiracidium, a minute ciliated larva emerges and actively swims about searching for a suitable host. Build-up of intensity is thus potentially not as "explosive" as that of *Gyrodactylus* spp. Both *Ligictaluridus* spp. and *Cleidodiscus* spp. parasitize the gills of their hosts. Overcrowding, especially of fry and/or fingerlings, often leads to increased intensity of infection, which may or may not produce signs of disease.

   Ancyrocephalids are most abundant on wild freshwater fishes in mid to late summer; however, there is much variation and generalizations are not possible. Fish of all ages are found to be parasitized, but young fish appear to be the most affected.

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D. Disease Signs

1. Behavioral Changes Associated with the Disease
   Ancyrocephalids live on the gills of their hosts and respiratory distress is not uncommon. Gasping may occur in heavy infections on young fishes, especially if water temperatures are high. Under these conditions fish are often lethargic and unable to feed.

2. External Gross Signs
   In heavy infections, there is an obvious production of mucus on the gills of young fish with slight flaring of the operculum.

3. Internal Gross Signs
   None reported.

4. Histopathological Changes Associated with the Disease
   Mechanical damage to the epithelium of the gill lamellae is caused by the attachment disc (haptor), which is armed with hamuli and marginal hooks, and by the tissue-grazing method of feeding used by these parasites. A mild tissue reaction, involving hyperplasia, at the site of attachment has been reported. In *Cleidodiscus* infections on adult bluegills, lesions are not uncommon and appear as off-white to yellow cysts on the gill filaments. These cysts are composed of hyperplastic, squamous epithelium covering well developed granulation tissue. Necrosis may occur in areas where vascularization is diminished.

E. Disease Diagnostic Procedures

   See “Disease Diagnostic Procedures” for *Gyrodactylus* spp.

F. Procedures for Detecting Subclinical Infections

   See “Procedures for Detecting Subclinical Infections” for *Gyrodactylus* spp.

G. Procedures for Determining Prior Exposure to Etiological Agent

   None available for Monogenean diseases.

H. Procedures for Transportation and Storage of Samples to Ensure Maximum Viability and Survival of the Etiological Agent

   See “Procedures for Transportation and Storage of Samples to Ensure Maximum Viability and Survival of the Etiological Agent” for *Gyrodactylus* spp.
Figure 2. Generalized *Ligictaluridus* sp.
III. Dactylogyrids - Including *Dactylogyrus* spp.

A. Name of Disease and Etiological Agent

"Monogenean Disease" is an acceptable general term. *Dactylogyrus* spp. (*Platyhelminthes* : *Monogenea*).

B. Known Geographical Range and Host Species of the Disease

1. Geographical Range

   *Dactylogyrus* spp. occurs on a wide variety of freshwater fishes throughout North America.

2. Host Species

   Cyprinids, including goldfish *Carassius auratus*, common carp *Cyprinus carpio*, grass carp *Ctenopharyngodon idella*, and golden shiner *Notemigonus crysoleucas*, are the major hosts of *Dactylogyrus* spp. Parasitized fish probably occur throughout the natural ranges of the above hosts.

C. Epizootiology

   Fry and fingerlings of carp are particularly susceptible to *Dactylogyrus* infection and high mortalities occur in culture situations in eastern Europe and the C. I. S.

1. Conditions Under Which Disease Occurs

   *Dactylogyrus* spp., like the ancyrocephalids, produce shelled eggs and have life cycles in which generation time is temperature-dependent. *Dactylogyrus* spp. on carp kept in colder climates generally have a longer generation time (e.g. 5 to 6 months at 1 to 2° C) than those in warmer areas (e.g. only a few days at 22 to 24° C).

   Overcrowding, especially of young fish, may produce stressful conditions in which infected fish succumb and die. Mortalities of 80-100% have been recorded in some carp fry populations. *Dactylogyrus* spp. typically occur on the gills of their hosts and, in water with a low concentration of oxygen, respiratory impairment may lead to death of fry and fingerlings. In Israel, fish over 35mm long were found to tolerate relatively high intensities of infection and some larger carp are resistant to infection when exposed to *Dactylogyrus* larvae.

D. Disease Signs

1. Behavioral Changes Associated with the Disease

   These are similar to those noted for the ancyrocephalids above.

2. External Gross Signs

   As above for heavy infections.

3. Internal Gross Signs

   None reported.
4. Histopathological Changes Associated with the Disease

*Dactylogyrus* spp. are tissue browsers and cause mechanical damage by feeding with their attachment apparatus (haptor). Marked gill hyperplasia is seen in heavy infections and results in serious malfunction of the respiratory surface and deformation of the gill lamellae. Heavily infected fish may exhibit blood changes, e.g. low hematocrits and depletion of hemoglobin. Host growth rate is not normally affected unless culture conditions are unsatisfactory. If the young fish are able to feed, they grow rapidly and overcome the deleterious effects of the parasite and if culture conditions are not good, growth rate may be retarded.

E. Disease Diagnostic Procedures

See “Disease Diagnostic Procedures” for *Gyrodactylus* spp.

F. Procedures for Detecting Subclinical Infections

See “Procedures for Detecting Subclinical Infections” for Gyrodactylus spp.

G. Procedures for Determining Prior Exposure to Etiological Agent

None available for Monogenean diseases.

H. Procedures for Transportation and Storage of Samples to Ensure Maximum Viability and Survival of the Etiological Agent

See “Procedures for Transportation and Storage of Samples to Ensure Maximum Viability and Survival of the Etiological Agent” for *Gyrodactylus* spp.
Figure 3. Generalized *Dactylogyrus* sp. (a) entire animal, ventral view, (b) dorsal hamulus, (c) dorsal transverse bar, (d) larval hook.
References


