Proliferative gill disease (PGD) has become common in farm-raised channel catfish. It can kill a few dozen fish over several days, or up to 100 percent of the fish in less than 3 days. Recurrence in the same pond is rare. From 1991 to 1995 PGD was the fourth most commonly diagnosed disease of catfish in the southeastern U.S., occurring in one out of every ten disease cases. This disease causes catfish to suffocate because of the severe damage to the gills. Swelling and a red and white mottling of the gills gives them a raw hamburger appearance, and many refer to PGD as hamburger gill disease (Fig. 1).

Clinical signs and diagnosis

Proliferative gill disease occurs most often in the spring, but it can occur in the fall at water temperatures between 59 and 72°F (15 to 22°C). It sometimes occurs in winter; PGD mortalities have been reported at 43°F (6°C). Though the disease seldom occurs in the summer, deaths have been reported at 92°F (33°C). Even before the disease occurs, signs of PGD can be seen from March through May in gills viewed under the microscope. As with other diseases, a common early sign of a PGD outbreak is a reduction of feeding activity by the fish. As the disease progresses, the catfish congregate in the water flow behind an aerator or at incoming water. Fish may also swim listlessly at the water’s surface and then lie in shallow water along the edge of the pond before they die. They may die even when dissolved oxygen concentrations are at levels high enough for healthy fish, because the affected gills cannot remove sufficient oxygen from the water.

The skin of catfish affected with PGD appears healthy, and while PGD occasionally is found in internal organs (liver, kidney, spleen and brain), it primarily affects the gills. The gills swell and become mottled red and white in appearance, similar to raw hamburger meat. In advanced stages, the gill filaments do not lie flat and filaments on one gill arch are not distinct from filaments on other arches. The gills often look mashed and may bleed when touched or when the fish are simply lifted from the water.

Microscopic examination at 40X magnification reveals extreme swelling of the gills caused by an abnormally large number of cells at the outer edge of the gill filaments. These swollen areas often appear white. The swelling is often very abrupt and can look like the thumb of a mitten pro-
truding from the side of a gill filament. Some parts of the gill filaments look red because blood cells are pooled in ruptured or dilated capillaries. The gill filaments may become shorter and wider with rounded or squared tips. The cartilage supporting the gill filament appears as a dark gray band along the side of each filament and may have notches, breaks and gaps (Fig. 2). These characteristics are also much more obvious when examined under 40X magnification than at higher magnifications, and are the best features for making an early, presumptive diagnosis. The lesions in the cartilage can be occupied by the parasite that causes PGD. Breaks and gaps in the filaments’ supporting cartilage cause the gills to lose their well-defined structure and collapse onto each other, giving the mashed appearance. Parasite cysts are only occasionally seen in wet mounts under the microscope and appear as small, indistinct, round units. PGD diagnosis is confirmed by histology procedures where the parasite can be seen as a blue stained “cluster of grapes” (Fig. 3) in very thinly cut sections of gill tissue.

**Cause and disease course**

Most scientists believe that a sporozoan, probably the myxosporean parasite *Aurantiactinomyxon* sp., is the causative agent of PGD. Evidence suggests that an oligochaete worm (*Dero digitata*) that lives in the mud and grows up to 1/2 inch in length is the invertebrate host. The PGD organism is thought to develop in the worm, which releases infective spores capable of penetrating and infecting the gills of channel catfish. Most parasites inflict less damage to their natural hosts, and mature spores are usually found in the host tissue. Massive gill

![Figure 2. The supporting cartilage (dark band in center of picture) of the gill filament in catfish with PGD may contain gaps. Note the two clear circular areas in the cartilage and the cell proliferation along the edges of the gill filament. (Photo by Drew Mitchell.)](image)

![Figure 3. PGD diagnosis is confirmed by histology procedures where the parasites can often be seen as large blue stained areas inside hollow spaces near the supporting cartilage (pink band to the right of the parasite on the right). (Photo by Dr. Lenn Harrison.)](image)
destruction, resulting in high mortality, and the lack of mature spores indicate that the catfish may be an unnatural host for Aurantiactinomyxon sp. Most of the gill damage is thought to be caused by an inflammatory response of the fish to the parasite.

Following is a possible life cycle of PGD:

- Mature parasites (possibly Aurantiactinomyxon sp.) or an infective stage are released from the fish host.
- The invertebrate host (probably Dero digitata) becomes infected.
- The parasite develops in the invertebrate host.
- An infective stage of the parasite is released from the worm.
- This infective stage penetrates and infects the catfish gill tissue.

There has been an ongoing controversy among researchers and diagnostic workers about the occurrence of this disease in new ponds built or reworked within the last 3 years as compared with older ponds. Although PGD occurs in older ponds, it seems to appear more often in new ponds, perhaps because they support larger populations of Dero worms.

**Treatment and prevention**

Though no treatments or preventive methods for PGD have been scientifically validated, there are some treatments that appear effective. Since fish infected with PGD suffer gill damage and are less able to obtain oxygen from water, aeration should be used when dissolved oxygen concentrations are low or marginally low. Another option is to quickly harvest and process PGD infected fish. However, many fish may die during harvest and have to be discarded; those making it to the processing plant alive can be quickly processed and pose no danger to the human consumer.
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