Many fungi cause diseases that can infect and kill channel catfish eggs, fry, fingerlings and adults. Most fungal infections are caused by water molds of the family Saprolegniaceae, so fungal diseases in catfish are commonly called saprolegniasis. Within the Saprolegniaceae family, Saprolegnia sp., Achyla sp. and Branchiomyces sp. are the genera that cause most disease in channel catfish. Although some fungal species within this family are primary pathogens (e.g., Saprolegnia parasitica), most (e.g., S. declina and S. saprolytica) cause disease only when there is preexisting illness, mechanical injury, or environmental stress.

Winter saprolegniasis (winter fungus or winter mortality)

The most common and economically important fungal disease of cultured channel catfish is winter saprolegniasis. The species of Saprolegnia responsible for this disease has not yet been identified. Other terms used to describe this disease are winter fungus, winter mortality and winter kill syndrome. The term winter kill is also used to describe massive fish mortalities that occur in an ice- and snow-covered pond, usually because of the depletion of dissolved oxygen under the ice. This should not be confused with the fungal disease.

Winter saprolegniasis usually occurs between October and March when water temperatures are below 15 °C (59 °F). Mortality usually increases as temperatures rise in early spring. The disease has been reported as early as September and as late as April. The disease causes chronic losses and usually affects harvestable size fish (>1 pound); very high mortalities can occur. Fish greater than 2 pounds in crowded production ponds appear to be especially vulnerable to winter saprolegniasis. For this reason, it is a disease of great economic importance. Winter saprolegniasis is characterized by:

- brownish patches of cottony fungal growth on the skin, including the gills, (Figs. 1 and 2);
- dry, depigmented skin (Fig. 3); and
- endophthalmia (sunken eyes) (Figs. 3 and 4).

At first lesions are small, circular, depigmented areas, sometimes with hemorrhagic margins. In advanced cases lesions can become ulcerative, penetrating through the skin and into the
muscle tissue, and the fish can be almost completely covered with thick fungal growth. Death is thought to be related to the inability of fish to regulate the salt balance in the blood. Protozoan parasites are frequently found on the gills and skin of fish suffering from winter saprolegniasis.

The cause and pathogenesis of this disease are largely unknown; however, sudden decreases in temperature and a significant number of pathogenic *Saprolegnia* sp. zoospores in the water (≥ 5 spores/ml in laboratory experiments) have been identified as risk factors for the disease. The primary risk factor is thought to be the inability of the fish to adapt to rapidly fluctuating water temperatures during the winter months. In experimental trials, rapid decreases in water temperature (72 °F down to 54 °F, or 22 °C down to 12 °C, in 24 hours) have been shown to impair the fish’s immune system, cause a loss of mucus from the skin, and temporarily suppress mucus production by goblet cells in the dermal layers of the skin. Mucus provides a physical barrier that prevents fungal spores from contacting and infecting the skin of the fish. Mucus also contains antimicrobial components (including immunoglobulin or antibodies, lysozyme, complement, C-reactive protein and proteolytic enzymes) that can destroy invading zoospores. Without mucus, skin is unprotected and fungal spores begin developing masses of fungal hyphae that extend into the muscle tissue. If fungal spores are not present in sufficient numbers to establish infection, fish can adapt to a change in temperature and regain normal function of goblet cells and mucus production within 6 days, and can regain their immune cell function within 5 weeks, based on research results. However, once the infection is established, fish do not appear to regain normal immune function, which makes the infection more severe.

Any condition that causes a loss of mucus or compromises the skin or immune system will likely pre-
dispose fish to fungal infections. Physical injuries caused by seizing, handling or crowding, or lesions caused by infectious pathogens create sites where fungal infection can occur.

**Treatment and prevention**

Because of the expense and undocumented efficacy of chemical treatment, control of winter saprolegniasis presently focuses on prevention and development of production strategies that limit the economic loss from the disease. Optimizing water quality and reducing stress, especially in the late summer and fall, can decrease the effect of this disease. Diseases such as columnaris (SRAC publication 479) that occur in late summer or early fall may predispose fish to winter saprolegniasis, so it is important to diagnose and treat those conditions promptly.

Maintaining sufficient oxygen concentrations (4 to 5 ppm) may also be important in avoiding winter saprolegniasis. In a field trial at a commercial catfish operation, repeated stress caused by low dissolved oxygen during the summer and early fall was correlated with increased occurrence of winter saprolegniasis. Unfortunately, optimal water quality is difficult to maintain in large, heavily stocked and fed ponds. One aspect of water quality that is relatively easy to manage is maintaining adequate chloride concentrations to prevent nitrite toxicity (see SRAC publication 462).

Reducing the standing crop of harvestable fish also will decrease the potential loss from winter saprolegniasis. Over-wintering densities should not exceed 4,000 to 5,000 pounds per acre. Where there is a history of the disease, producers should use conservative stocking densities and reduce the standing crop of market-size fish by harvesting before winter. Promptly harvesting and selling fish at the very first sign of winter saprolegniasis can help to avoid large losses. In the early phase of the disease fish are infected on the skin surface with little or no invasion of the muscle and can, therefore, be marketed safely.

Another factor that can influence the development of winter saprolegniasis is pond depth. In theory, deeper ponds have more capacity to resist changes in temperature. Therefore, maintaining ponds at their maximum depth can reduce temperature fluctuation and help fish acclimate to changes.

One prevention strategy being investigated is using prophylactic chemical treatments to reduce the abundance of pathogenic zoospores. Laboratory trials have shown that formalin (25 ppm), copper sulfate (rate dependent on total alkalinity of the water), and diquat (0.125 ppm) can prevent the development of *Saprolegnia* infections in aquaria by inhibiting fungal zoospores. These studies were conducted using well water where the chemicals have higher potency and persist longer. Higher chemical rates will likely be required in production ponds. Once a successful pond treatment is identified, a method will be needed to determine which ponds should be prophylactically treated. In particular, a technique is needed to differentiate between the pathogenic spores of the *Saprolegnia* species responsible for winter saprolegniasis and non-pathogenic spores such as those of *S. declina* and *S. saprolytica*.

Future research for controlling winter saprolegniasis may include the use of fungicides (such as hydrogen peroxide or bronopol) or improved diets. Hydrogen peroxide ($H_2O_2$) is FDA-approved as an anti-fungal treatment on fish eggs. It has shown promise in experimental treatments of fungus on fish in ponds, but is not currently being used for winter saprolegniasis. Some researchers have suggested that fish diets may need to be changed to provide the kind of lipids that allow the fish’s immune cells (especially the T cells) to function properly when exposed to sudden cold temperatures.

**Branchiomycosis**

Branchiomycosis affects a wide variety of cultured fish throughout the world but is a relatively new disease in channel catfish culture. Branchiomycosis has been reported only in fry and small fingerlings stocked in nursery ponds at warm temperatures (above 68 °F or 20 °C). Infections are usually self-limiting and occur in fish up to 2 months old. Infections are located primarily in the blood vessels (intravascular) of the gill and are confined to the gill arches and the base of the primary lamellae. Fungal hyphae can be observed only with a microscope. Swelling of the gill tissues and blockages formed by the fungal hyphae in the vessels of the gills decrease respiratory efficiency. Infected fish are also subject to secondary bacterial and viral infections, which greatly increase mortality.

There is no known treatment for branchiomycosis. Although copper sulfate and formalin are suggested for treating other fish species, no trials have been conducted to evaluate their efficacy with catfish. Because the gills are the primary site of infection, supportive therapy involves maintaining adequately aerated water and increasing the chloride concentration of the water. An antibiotic may also be necessary if secondary bacterial infection develops. Unfortunately, the disease affects very young fish that may not yet accept prepared diets, making drug delivery difficult. Antibiotics usually must be administered in a finely crumbled, medicated diet. Although the disease is not known to recur annually in cultured channel catfish, ponds should be drained and thoroughly dried before stocking fry, especially if the ponds have a history of branchiomycosis.
Suggested readings


This and other SRAC publications can be found on-line at www.msstate.edu/dept/srac.

SRAC fact sheets are reviewed annually by the Publications, Videos and Computer Software Steering Committee. Fact sheets are revised as new knowledge becomes available. Fact sheets that have not been revised are considered to reflect the current state of knowledge.

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