Saprolegniasis – Cotton Wool Disease

I. Causative Agent and Disease

The disease saprolegniasis is caused by water molds (oomycetes) mostly in the genus *Saprolegnia*. Genetic sequencing places oomycetes in the class Oomycota, phylum Heterokontophyta, related to photosynthetic brown algae, diatoms and apicomplexan protozoa. They are filamentous protists based on production of oospores, diploid chromosomes, cell walls of beta glucans and cellulose, two types of zoospore flagella and tubular cristae in the mitochondria. Saprolegniasis describes any cotton-like growth adherent to skin or gills that include several genera of molds. Water molds occur in fresh and brackish water less than 2.8 ppt salinity. Most are saprophytes occurring naturally in the environment and are opportunistic pathogens, usually requiring prior injury of external tissues from mechanical abrasion or other primary pathogens. Some species of *Saprolegnia* (*parasitica*) are primary pathogens producing a systemic disease.

II. Host Species

All freshwater fish species, incubating eggs and other lower aquatic vertebrates/invertebrates worldwide are susceptible to saprolegniasis.

III. Clinical Signs

The mold produces white/brown cotton-like foci on the surface of the skin and/or gills. Early foci are pale with peripheral areas of erythema and central zones of lifted scales that frequently ulcerate, exposing underlying musculature. Systemic infections produce mycelial masses in the gut and viscera causing peritonitis, 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 mold 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 mold hyphae. Environmental stress plays 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 mold 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 of white, cottony tufts of hyphae on the skin, gills and other surfaces of infected fish or eggs. Wet mounts of mycelium from lesions show large, branching, non-septate hyphae. Terminal ends of older hyphae form club-shaped sporangia containing biflagellated zoospores. The mold can be isolated on cornmeal or potato agar.

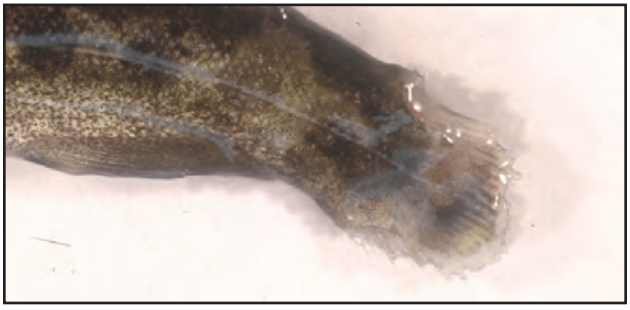
VI. Prognosis for Host

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-

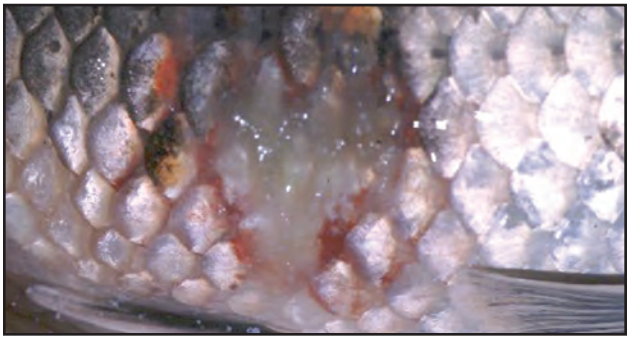
fully with 1hr formalin drips. There is no treatment for systemic molds that are rapidly fatal.

VI. Human Health Significance

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



Tail rot on a juvenile salmonid infected by *Saprolegnia* mold.



Typical lesion with peripheral hemorrhaging due to *Saprolegnia* mold on the skin of a slender bitterling.

Wet mount of *Saprolegnia* mold: large hyphae with no cross-walls and a sporangium (arrow) containing zoospores, X 1000.