Environmental and other non-infectious diseases

Gregoria Erazo-Pagador

Non-infectious diseases are caused by adverse environmental conditions, nutritional disorders, or genetic defects. While they can result in sudden mass mortality or death, they are not contagious.

Environmental diseases are the most important in aquaculture. These include low dissolved oxygen, high ammonia, high nitrite, or natural or man-made toxins in the aquatic environment.

This chapter presents some of the environmental and noninfectious diseases of aquatic animals.

FISH DISEASES ASSOCIATED WITH PHYSICO-CHEMICAL PROPERTIES OF WATER

Gas Bubble Disease

Gas bubble disease is due to supersaturation of dissolved gases (nitrogen or oxygen); supersaturation may be due to leaks in pump or valve systems in hatcheries; dense algal bloom that presumably caused oxygen depletion at night and supersaturation during the day.

SIGNS:

Affected fish show bubbles in the abdominal cavity, eyes, skin, gills, fins, mouth, swimbladder and within the digestive tract (Fig. 6-1) and exophthalmia. The clinical signs of gas bubble disease should not be confused with Swimbladder stress syndrome because the bubbles in the latter can be seen only in the swimbladder.

EFFECTS ON HOST:

Death due to embolism in blood and emphysema in tissues; edema and degeneration of the gill lamellae; bulging of the cornea; abrupt mass mortalities.

PREVENTION AND CONTROL

- Monitor dissolved oxygen (DO)
- Avoid algal blooms
- Maintain efficient operation of waterlines and pumps
- Sufficient water exchange
Swimbladder Stress Syndrome

Swimbladder stress syndrome (SBSS) is associated with malfunction of the swimbladder and is also associated with a combination of handling, high ambient temperature, high ambient illumination, dense algal bloom that presumably cause oxygen depletion at night and supersaturation during the day.

**Signs:**
Affected fish larvae show large bubble of gas in the region antero-dorsal to and outside the swimbladder (Fig. 6-2).

**Effects on Host:**
Hyperinflation of swimbladder; high positive buoyancy and mass mortalities.

**Prevention and Control:**
- Filter rearing water
- Regulate algal bloom in hatcheries
- Provide strong aeration to maintain the larvae beneath the water surface in hatchery tanks

Asphyxiation/Hypoxia

Very low levels of dissolved oxygen (DO) cause asphyxiation/hypoxia.

**Signs:**
Affected fish gather at the water inlets and outlets. Also fish with wide gaping mouth swim at water surface and show rapid opercular movement.

**Prevention and Control:**
Monitor DO levels and provide aeration.

Salinity

Extremely high salinities are associated with progressive emaciation, scale loss, and opaque eye lenses in affected fish.

Alkalosis

Alkalosis comes about when water becomes too basic. The pH increases to a level higher than the fish can tolerate.

**Signs:**
Affected fish show corroded skin and gills and milky turbidity of the skin.

**Prevention and Control:**
Monitor pH level, and maintain the pH in a range optimal for the species being cultured.

Acidosis

Acidosis is caused by a drop in the pH to a level too low for the species.

**Signs:**
Affected fish show rapid swimming movements and gasping. Increased mucus secretion and death occur very quickly. Affected shrimp show poor growth, low molting frequency and yellow to orange to brown discoloration of the gill.

Figure 6-2. Seabass larva with Swimbladder stress syndrome compared with normal larva (top)
and appendage surfaces. The pond soil turns reddish in color.

**EFFECTS ON HOST:**

Normal metabolism is hindered resulting in retarded growth and eventual death.

**PREVENTION AND CONTROL:**

- Monitor pH
- Apply lime and flush pond bottom before stocking

---

**Sunburn Disease**

Sunburn disease is due to excessive levels of ultraviolet irradiation from sunlight when fish are stocked in shallow uncovered raceways under intense sunlight.

**SIGNS:**

Development of gray focal circular ulcerative lesions on top of the head, pectoral, dorsal and upper tail fins.

**EFFECTS ON HOST:**

Ulcerative lesions may serve as portals of entry for other pathogens and may result in secondary infection.

**PREVENTION AND CONTROL:**

Place sunshades over ponds, and fish should be kept away from ultraviolet installations.

---

**SHRIMP DISEASES ASSOCIATED WITH PHYSICO-CHEMICAL PROPERTIES OF WATER**

**Muscle Necrosis**

Muscle necrosis in shrimp is caused by temperature and salinity shock, low oxygen levels, overcrowding, rough handling and severe gill fouling.

**SIGNS:**

Affected shrimp show opaque white areas on the abdomen (Fig. 6-3); blackening on the edges of the uropod followed by erosion and liquid-filled boils at the tip of uropods in advanced stages; “wood grain” appearance of abdominal muscle in postlarvae (Fig. 6-4).

**EFFECTS ON HOST:**

There is a gradual death of cells of affected parts leading to erosion especially in the tail. This may then serve as portal of entry for secondary systemic bacterial infection.

**PREVENTION AND CONTROL:**

Reduce stocking density and improve water quality by daily water change (5-10%)
Bent/Cramped Tails or Body Cramp

Bent/cramped tails or body cramp in shrimps is associated with handling of shrimp in warm, humid air much warmer than culture water, and mineral imbalance.

SIGNS:
Affected shrimps have partial or complete rigid flexure of the tail (Fig. 6-5).

EFFECTS ON HOST:
Partially cramped shrimps swim with a humped abdomen; fully cramped shrimps lie on their sides at the pond/tank bottom. Healthy shrimps may cannibalize weak ones.

PREVENTION AND CONTROL:
Avoid possible causes.

Incomplete Molting

Incomplete molting is closely associated with low temperature of culture water.

SIGNS:
Presence of old exoskeleton attached to newly molted larvae, especially in appendages (Fig. 6-6).

EFFECTS ON HOST:
Abnormal swimming movement which could lead to easy predation and mortality.

PREVENTION AND CONTROL:
• Maintain optimum temperature in the rearing water
• Use water heater

Asphyxiation/Hypoxia

Reduced dissolved oxygen due to high organic load or algal bloom and subsequent die-off and high temperature

SIGNS:
Affected shrimp showed surface swimming and sudden mass mortality.

EFFECTS ON HOST:
Prolonged respiratory distress leads to death and sublethal levels may cause impairment of metabolism resulting in growth retardation.

PREVENTION AND CONTROL:
• Decrease stocking density
• Monitor water parameters frequently
• Provide aeration facilities and water pump for ready water change
Acidosis is caused by low water and soil pH.

**SIGNs:**
Affected shrimp show poor growth, low molting frequency and yellow to orange to brown discoloration of the gill and appendage surfaces. The pond soil turns reddish in color (Fig. 6-7).

**Effects on Host:**
Normal metabolism is hindered resulting in retarded growth and eventual death.

**Prevention and Control:**
- Monitor the pH
- Apply lime and flush the ponds before stocking

Black gill disease is due to chemical contaminants, heavy siltation and ammonia or nitrite in rearing water; high organic load due to residual feed, debris, and fecal matter on pond bottom (i.e. black soil).

**SIGNs:**
The gills of affected shrimps show reddish, brownish to black discoloration and, in advanced cases, gill filaments become totally black (Fig. 6-8).

**Effects on Host:**
Histological observations show that blackening of the gills may be due to the deposition of melanin at sites of tissue necrosis and heavy hemocyte activity.

**Prevention and Control:**
- Avoid heavy metal discharges of nearby factories from getting into the rearing facilities
- Remove black soil by scraping after each harvest and by draining pond water from the bottom during the culture period

Red disease in shrimps is associated with high application of lime (2-6 tons/ha) in the pond that gives it a high initial pH; prolonged exposure to low salinity (6-15 ppt).

**SIGNs:**
Affected shrimps have red short streaks on gills or abdominal segments, yellowish to reddish discoloration of the body (Fig. 6-9) and increased fluid in the cephalothorax, emitting foul odor.

**Effects on Host:**
Yellow to red discoloration in affected shrimps; histopathology of the hepatopancreas shows hemocytic infiltration in the spaces between the tubules; more advanced lesions are in the form of fibriotic and melanized encapsulation of necrotic tissues, either in the tubule itself or the sinuses around it.
Chronic Soft-Shell Syndrome/Soft-Shelling

Chronic soft-shell syndrome is associated with exposure of normal hard-shelled shrimps to pesticides and piscicides. Aquatin at 0.0154 - 1.54 ppm, Gusathion A at 1.5 - 150 ppb, rotenone at 10-50 ppm, and saponin at 100 ppm for 4 days can induce soft-shelling in initially hard-shelled stocks.

**Signs:**

Shell is thin and persistently soft for several weeks, shell surface is often dark rough and wrinkled, and affected shrimps are weak. The disease must not be confused with the condition of newly-molted shrimps, which have clean smooth, and soft shells that harden within 1-2 days.

**Effects on Host:**

Affected shrimps are soft-shelled, grow slowly, and eventually die; histopathology of shrimps exposed to Gusathion A shows slight hyperplasia of the gill epithelium, delamination of the cells lining the tubules of the hepatopancreas, and general necrosis and degeneration of these tissues.

**Prevention and Control:**

- During pond preparation, flush ponds thoroughly particularly when pesticide contamination is suspected
- Maintain pond water and soil of good quality

---

**DISEASES ASSOCIATED WITH PHYSICAL FACTORS**

Diseases associated with physical factors are mainly due to handling, transport, high stocking density and predation. Secondary bacterial, viral, or parasitic infection may easily set in once injuries are introduced.

---

**DIAGNOSIS OF ENVIRONMENTAL AND OTHER NON-INFECTIONOUS DISEASES**

Diagnosis of noninfectious diseases can be done through gross examination of fish for external/internal disease signs; histopathological/histochemical analysis; hematological analysis to assess the cellular composition of the blood in response to environmental stress; analysis of physico-chemical characteristics of rearing water; and evaluation of culture operations and management practices.
REFERENCES/SUGGESTED READING


Ferguson HW. 1989. Systemic Pathology of Fish. Iowa State University Press, Iowa, 263 p


