Organ and Tissue Anomalies

I. Causative Agent and Disease

Developmental anomalies that are not caused by infectious disease or pollution are occasionally observed in salmonids and other fish species. These anomalies (coagulated yolk, blue sac disease and pigment aberrations discussed in other specific sections) may result from genetic or congenital defects and/or suboptimal environmental or water quality conditions during early life stages that cause abnormal development of various organs and tissues.

II. Host Species

All wild and cultured fish species are susceptible to developmental anomalies worldwide. These disorders are more easily observed in fish species that are cultured on a large scale such as salmon and trout.

III. Clinical Signs

Some of the more common deformities observed in Alaskan salmonid aquaculture occur in fry and fingerlings including malformations such as: siamese twinning; microphthalmia (small eye) or anophthalmia (missing eye); spinal curvatures; vertebral compression and fusion (sunfish and humped back); pughead (hypoplasia of upper jaw); and shortened opercula (exposed gills). Three other deformities require further explanation: **Double mouth** results from displacement of the lower end of the hyoid arch downwards and backwards through the gap in the mouth floor. Trapped air from an obstructed pneumatic duct and/or mechanical injury damages muscles in the jaw so that the retractor muscles, left unopposed, pull the lower end of the arch into the deformed position; **Mandibular ankylosis** is an incomplete ossification of Meckel's cartilage with displacement of angular bone from possible phosphorous imbalance resulting in a permanently fixed wide open mouth with flared opercula. Gill respiration requires swimming continuously (ram ventilation); **Fibrous osteodystrophy** in Arctic char and rainbow trout cultured at one hatchery in Alaska results from incomplete morphogenesis of dermal bones in the sensory cranial canals and lateral line. This causes tissue separation and replacement with fibrous connective tissue. The condition is associated with recirculation of well water and suspected imbalance of calcium and phosphorous due to the chemistry of the water supply. Affected fish show lower Ca:P serum concentrations, but further study is ongoing to confirm the cause.

IV. Transmission

These anomalies have no infectious causes and cannot be transmitted. However, excluding those defects that are strictly congenital, some of the other conditions are likely caused by suboptimal environmental and/or water quality variables during early development that might be corrected in a hatchery facility to prevent deformity occurrence.

V. Diagnosis

Diagnosis is based on observation of the specific deformity and any other associated characteristics that may require histological examination.

VI. Prognosis for Host

Some of these anomalies may be fatal and prevent fish from reaching maturity and/or predispose them to early death by predation.

VII. Human Health Significance

There are no human health concerns associated with these developmental anomalies in fish from unpolluted waters.

Left: Eye deformity (sunken, darkened eyeball) in Kenai River sockeye salmon; **Inset:** Eye dissected demonstrates concave center; **Center:** Siamese twins and curled spine in juvenile sockeye salmon; **Right:** Sablefish with spinal deformity (kyphosis and scoliosis).

Left: Double mouth deformity in juvenile coho salmon- displaced hyoid arch (arrow) below mouth (photo: SSRAA staff); **Center:** Mandibular ankylosis (fixed gaping mouth) in adult sockeye salmon; **Right:** Fibrous osteodystrophy in Arctic char; note the fibrous separation of the sensory canals in the cranium and lateral line (arrows).