CASE NUMBER: 03F-96-A3.2-22
DATE: Dec 19, 2003

HISTOPATHOLOGY:
Slide 40, A3.2-22-1:
1). Gills, respiratory epithelia: Degeneration and necrosis, mild to moderate, multifocal, acute with multiple intralesional phytoplankton and algal like elements
2). Liver, hepatocytes: Necrosis, mild to moderate, multifocal, acute with scattered hemorrhage
3). Spleen: Congestion, moderate, diffuse, acute

There are no significant lesions within the peripheral vasculature, heart, kidney or peripheral nerves.

Slide 41, A3.2-22-2:
1). Gill: As in slide 1.
2). Spleen: As in slide 1.

There are no significant lesions within the peripheral vasculature, heart, kidney, liver or peripheral nerves.

Slide 42, A3.2-22-3:
There are no significant lesions within the gills, spleen, peripheral vasculature, heart, kidney, liver or peripheral nerves.

Slide 43, A3.2-22-4:
1). Gill: As in slide 1, but more generalized branchial involvement

There are no significant lesions within the peripheral vasculature, heart, kidney, liver, spleen, or peripheral nerves.

Slide 44, A3.2-22-5:
1). Gills: As in slide 43, but with scattered acute hemorrhage.

There are no significant lesions within the peripheral vasculature, heart, kidney, liver, spleen, or peripheral nerves.

Slide 45, A3.2-22-6:
1). Gills: As in slide 43.

There are no significant lesions within the peripheral vasculature, heart, anterior kidney, posterior kidney, liver, spleen, or peripheral nerves.

Slide 46, A3.2-22-7:
1). Gills: As in slide 40.
2). Liver: Degeneration and necrosis, mild to moderate, multifocal, acute

There are no significant lesions within the peripheral vasculature, heart, kidney, spleen, or peripheral nerves.

Slide 47, A3.2-22-8:
1). Gills: As in slide 44 with scattered superficial accumulation of extracellular coccobacilli

There are no significant lesions within the peripheral vasculature, heart, liver, anterior kidney, spleen, or peripheral nerves.

COMMENTS:
The acute respiratory epithelial necrosis, hemorrhage and intralesional phytoplankton are consistent with an algal bloom. Due to acute hepatocellular degeneration and necrosis, the possibility of concurrent hypoxia (hypoxemia secondary to impaired branchial function, reduced environmental oxygen levels or increased metabolic demands) cannot be discounted. These processes would likely have contributed at least moderately to antemortem clinical signs and presumably the loss of these fish. The role of possible neurotoxins elaborated by the alga in the loss of these fish is unknown. Appropriate environmental monitoring should be instituted. There were no other significant lesions within the examined sections.

FINAL REPORT