CASE NUMBER: 03F-49-P3-28
DATE: Aug 2, 2003

MORPHOLOGIC DIAGNOSES:
Slide 32, P3-28-1:
There are no overt lesions within the liver, heart, kidney, peripheral nerves, spleen or peripheral vasculature.

Slide 33, P3-28-2:
1). Kidney: Nephritis, interstitial, marked, multifocal to coalescing, lymphohistiocytic, necrotising, subacute with scattered intrahistiocytic coccobacilli
2). Heart, spongy layer: Myocarditis, mild to moderate, multifocal, random, lymphohistiocytic, subacute with intrahistiocytic coccobacilli
3). Liver: Hepatitis, mild, multifocal, necrotising, acute with intrahistiocytic coccobacilli
4). Spleen: Splenitis, moderate, diffuse, necrotising, subacute with scattered lymphoid hyperplasia
5). Peripheral vasculature: Presumptive leukocytosis, moderate, multifocal, mononuclear and neutrophilic

There are no overt lesions within the peripheral nerves.

Slide 34, P3-28-3:
1). Spleen: Splenitis, moderate, focally extensive, botryoid, granulomatous, chronic
2). Heart, compact and spongy layers: Myocarditis, mild to moderate, multifocal, random, granulomatous chronic with reactive endocardia
3). Kidney, anterior: Nephritis, interstitial, mild, multifocal, granulomatous, chronic with lymphomyeloid hyperplasia

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

Slide 35, P3-28-4:
There are no overt lesions within the liver, pyloric caecae, pancreas, adipose tissue, heart, kidney, peripheral nerves, spleen or peripheral vasculature.

Slide 36, P3-28-5:
There are no overt lesions within the liver, pancreas, adipose tissue, heart, kidney, peripheral nerves, spleen or peripheral vasculature.

COMMENTS:
In 3 of 5 examined tissues, there were no discernible lesions; whereas, in slides 33 and 34, the multisystemic inflammatory infiltrate would have contributed significantly to impaired homeostasis. The granulomatous infiltrate is strongly suggestive of bacterial kidney disease (*Renibacterium salmoninarum*) and if fresh tissue is available, follow up culture, immunoflorescence or possibly polymerase chain reaction may be pursued for confirmation. The variation in section involvement suggests a low to intermediate

prevalence of infection within the stock. Nevertheless, based on the extent of involvement in the affected tissues, these fish were likely shedding bacteria antemortem and additional stock have likely been exposed and should be monitored for possible development of clinical signs. There were no other significant lesions within the examined tissues which may adversely impact the stock. The leukocytosis and lymphohistiocytic hyperplasia are attributed to persistent antigenic stimulation.

FINAL REPORT