

NOTE

Apparent chronic bacterial myeloencephalitis in hatchery-reared juvenile coho salmon *Oncorhynchus kisutch* in Alaska

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ABSTRACT: Chronic losses in juvenile coho salmon at 2 Alaskan hatcheries were associated with neurologic disorders and with chronic myeloencephalitis of apparent bacterial etiology. The syndrome occurred as a sequela to bacterial coldwater disease, which also occurs in Alaska. This case report appears to represent an extension of the geographic range of occurrence of the chronic neurologic form of coldwater disease, earlier cases of which have been reported in Washington and Oregon. Clinical signs are similar to whirling disease and nutritional blacktail, both of which must be considered in the differential diagnosis.

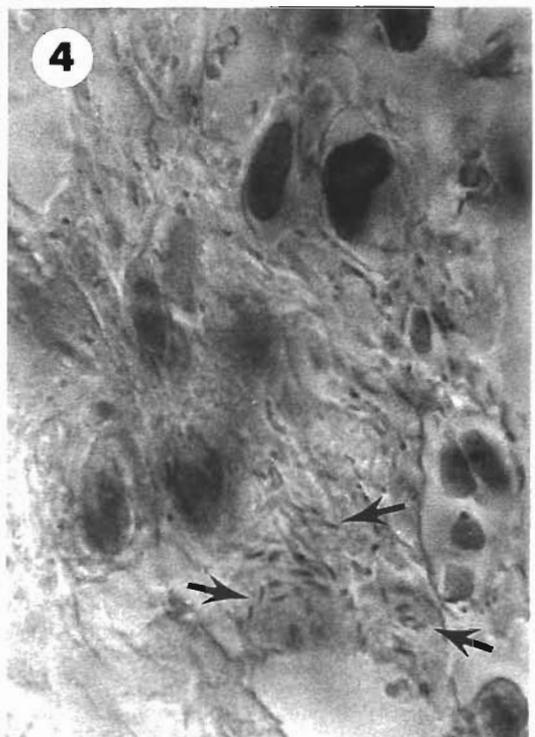
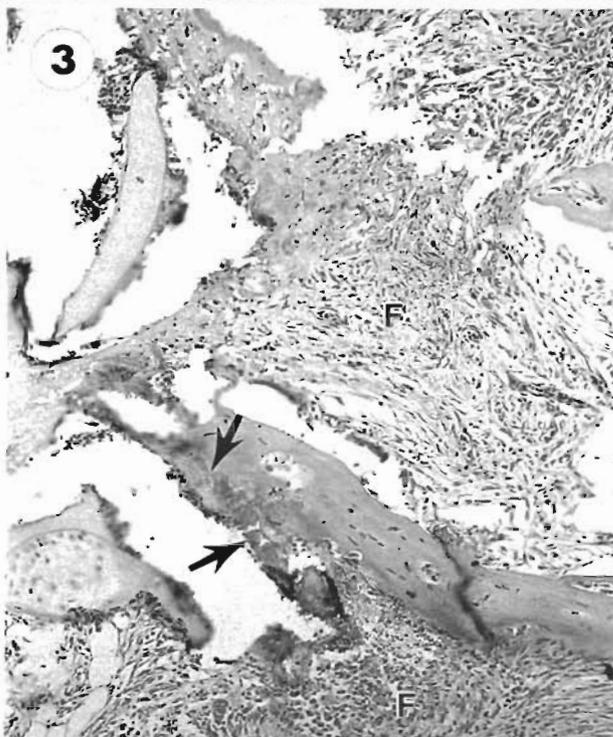
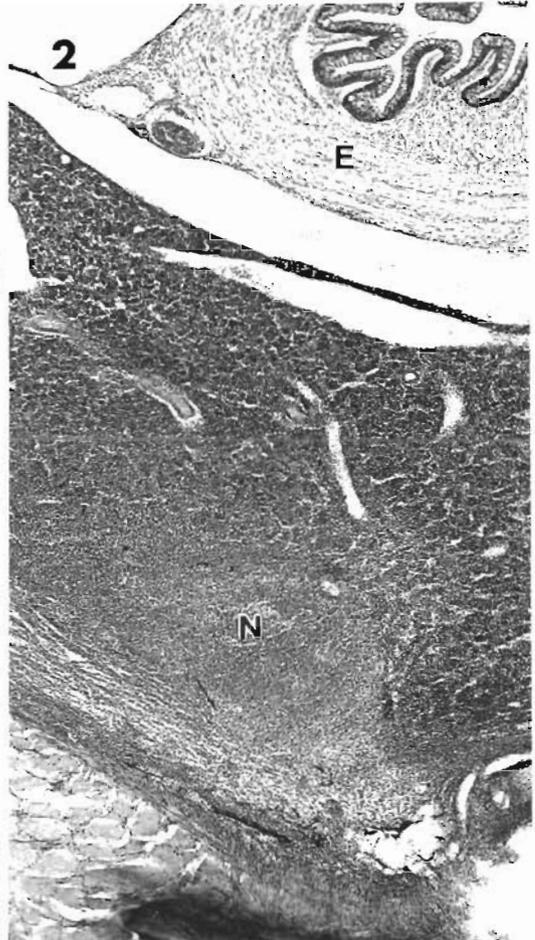
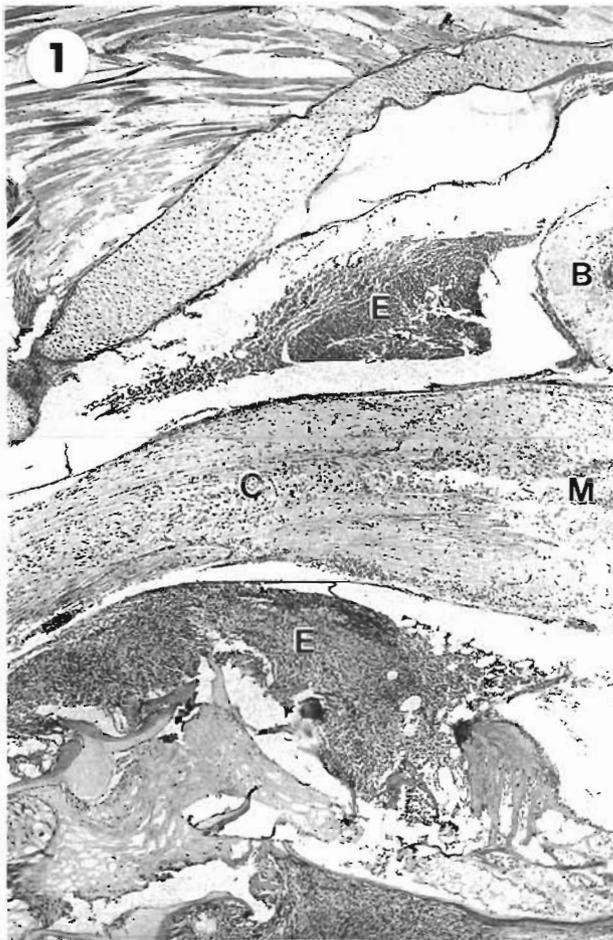
During July 1987 and August 1988 respectively, fingerling (4 to 6 g) coho salmon *Oncorhynchus kisutch* at 2 southeast Alaskan hatcheries sustained chronic mortality with neurological signs including: twirling behavior around the longitudinal axis; post-cephalic protrusion of the cranium; spinal deformities; and mild loss of melanocyte control in the posterior body (black-tail). Affected fish were anorexic, with mortality generally occurring in 3 d after the onset of clinical signs. However, sudden death of apparently healthy fish when handled was also a clinical feature. Mortality continued for weeks at levels of 0.1 to 0.2% d⁻¹ until affected fish dropped out of the population. During these episodes, ambient freshwater temperatures were 11 to 16°C, dissolved oxygen levels were at saturation, and fish densities were low (no greater than 5 kg m⁻³). The affected fish were being held in aluminium raceways and had been fed 3 commercially available diets of known nutritional quality.

Although other complicating features in the 1988 occurrence (including gill costiasis and gas supersaturation) undoubtedly contributed to the reported mortality levels, the outstanding clinical finding common to both occurrences was a neurologic lesion. This lesion was found in 12 of the 18 moribund fish examined using standard histologic procedures and hematoxylin and eosin staining. Microscopic features of this lesion

included fibrous inflammation of the vertebral canal at the junction of the spinal cord and the medulla oblongata. The inflammation caused an upward compression of the anterior position of the spinal cord (Fig. 1). The inflammation occasionally included the anterior kidney (Fig. 2). Erosion and necrosis of vertebral bone and cartilage (Fig. 3) and exudative edema within the posterior cranial cavity (Fig. 1), causing the gross post-cephalic protrusion, completed the histologic picture. The clinical signs were not typical of whirling disease caused by *Myxobolus cerebralis*, in that tail chasing behavior was not evident and caudal melanocyte hyperpigmentation was not as intense. Whirling disease was further contraindicated by the absence of trophozoites or spores of the causative agent in histologic sections, or in malachite green-stained smears of homogenized heads from 21 additional moribund fish. The condition also differed from nutritional blacktail because fish with the latter disease failed to show twirling behaviour and had normal caudal fin pigmentation.

Additional histologic sections, stained by the Giemsa method, from 1 of 4 fish with neurologic lesions in the 1988 episode revealed a single focus of medium-length bacterial rods within an area of active inflammation surrounding the vertebral column (Fig. 4). Previous culture attempts with kidney tissues on tryptic soy agar from both cases had failed to yield evidence for the presence of any significant bacterial agents. Unfortunately, bacteriology was performed prior to the discovery of the brainstem lesions. Consequently, the opportunity for isolating the agent from the cranial lesions was lost.

Chronic cumulative mortalities of 2 to 10% in juvenile coho salmon, rainbow trout *Salmo gairdneri*, and steelhead *S. gairdneri* showing spinning behavior, posterior paralysis, spinal deformities, and neurologic



Figs. 1 to 4. *Oncorhynchus kisutch*. Histologic sections from hatchery-reared juvenile coho salmon with chronic bacterial myeloencephalitis. Fig. 1. Inflammatory exudate (E) in the cranial cavity and within the vertebral canal where it is causing upward compression (C) of the spinal cord. M: medulla oblongata; B: cerebellum. H&E; $\times 144$. Fig. 2. Focal nephritis (N) of the head kidney. E: esophagus. H&E; $\times 144$. Fig. 3. Fibrous inflammation (F) of vertebrae near the brainstem with necrosis and erosion of bone (arrows). H&E; $\times 360$. Fig. 4. Medium length bacterial rods (arrows) within inflammatory exudate surrounding the vertebral column. May-Grunwald Giemsa; $\times 4333$

lesions similar to those described herein have been observed in Oregon and Washington States, USA, often following outbreaks of coldwater disease (Yasutake 1965, Conrad & DeCew 1967, Wood 1979, W. Brunson unpubl. [1987 AFS Western Fish Health Workshop, Bozeman, MT], Kent et al. 1989). Foci of myxobacteria-like cells in cranial lesions have been histologically demonstrated in coho salmon, and myxobacteria have been isolated from cranial lesions in coho salmon, rainbow trout, and steelhead (Kent et al. 1989). The isolated myxobacteria have been serologically identified as *Cytophaga psychrophila*, causative agent of coldwater disease (Kent et al. 1989). Overall, the results and epizootiological observations indicate that an occasional sequela in survivors of clinical or subclinical coldwater disease is the later invasion of the spinal cord by the causative agent of coldwater disease. The described neurologic disorders and chronic mortality then ensue. Fish from our 1987 case developed neurologic disorders and chronic mortality 2 to 3 mo following an outbreak of coldwater disease. The outbreak was controlled after 2 treatments with oxytetracycline followed by one with nitrofurizolidone. In the 1988 case, the condition was preceded by occasional losses due to cold water disease complicated by gill costiasis and gas bubble disease. In both cases, after onset of the neurologic stage of the disease the fish did not respond to dietary antibiotic therapy.

This case report extends the geographic range of chronic cranial and vertebral lesions apparently associated with coldwater disease. Further investigation of our embedded material might be worthwhile using serological staining techniques on histological sections to establish conclusively whether the bacterial agent in

these brainstem lesions is indeed *Cytophaga psychrophila*. However, in view of the low number of bacterial foci present in the lesions, the better course would be to sample the brainstem area bacteriologically in any future accessions showing neurological signs. From the clinician's perspective, the correct diagnosis of the disease syndrome condition presents a challenge. The clinical signs are markedly similar in some respects to whirling disease (Hoffman 1976) and, to a lesser degree to nutritionally-caused 'black-tail' (Wolf et al. 1981). Both of these diseases must therefore also be considered in the differential diagnosis.

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